BARÇA
INNOVATION HUB

TENDON INJURIES IN FOOTBALL PLAYERS: FC BARCELONA 2021 TENDON GUIDE

The diagnosis and management of lower limb tendinopathy

Jill Cook, Gil Rodas, Alan McCall, Ricard Pruna, Rochelle Kennedy and Lluís Til

Overview

Introduction

— Jill Cook, Gil Rodas, Alan McCall, Ricard Pruna, Rochelle Kennedy and Lluis Til

THE FC BARCELONA TENDON GUIDE: A MESSAGE FROM THE EDITORS

Tendinopathy is a common problem affecting football players and athletes in general. FC Barcelona staff have a vast experience in managing tendinopathy with its many athletes from different sports. FC Barcelona is passionate about enhancing the knowledge and understanding about how tendinopathy manifests in athletes and how to manage it effectively. It is for this reason that FC Barcelona have engaged some of the world's experts in tendinopathy, football and sports, in combination with the club's clinical and research experts, to write this guide on tendinopathy for the benefit of the football and sporting community.

There are many controversies in tendinopathy and a paucity of research in some areas. If research evidence is lacking, the authors clinical experience has been drawn on. Tendons can vary, as can clinical opinion, and some differences in clinical management are apparent between chapters. The best evidence and practical insights are provided for readers to digest and apply in the most appropriate way in their own practice.

Despite the controversies and differences in opinions among experts we have attempted to be consistent in wording throughout the guide. Tendinopathy is the clinical term for persistent tendon pain and loss of function and is related to mechanical load. Tendinosis is a pathological term and is used to refer to tendon abnormalities on imaging. We have used the term peritendinopathy to discuss any issues with connective tissue that surrounds the tendon.

We hope you enjoy this journey with us.

Biographies

JILL COOK,
Ph.D., BAppSci(Phty)

Jill Cook is a Professor in musculoskeletal health in the La Trobe Sport and Exercise Medicine Research Centre at La Trobe University in Melbourne Australia. Trained as a physiotherapist, Jill's research areas include sports medicine and tendon injury. She has investigated tendon pathology, treatment options and risk factors for tendon injury and she has published more than 300 scientific papers. Jill currently supplements her research by conducting a specialist tendon clinical practice and by lecturing both in Australia and overseas.

GIL RODAS,
MD, Ph.D.

Dr. Gil Rodas is an Exercise and Sports Medicine specialist who has worked for more than 15 years as a team physician for FC Barcelona in both football and basketball. He has developed an unparalleled clinical expertise on sports injuries during a career spanning 25 years. Currently he is the head of Medical Area for the Barça innovation Hub. Dr Rodas has been instrumental in building a global network of opinion leaders in the field of skeletal muscle and tendon injuries through establishing the MuscleTech group and coordinating several conferences on the topic. Dr Rodas has also established a rare research program between FC Barcelona and all it's sports teams and local academic scientists in Barcelona, where clinicians and the scientist pursue solutions/new therapies and assess their effectiveness for sports & musculoskeletal injuries. Altogether through these initiatives, more than 30 projects have been implemented from genomics, metabolomics to Return to Play. Dr Rodas has 81 scientific publications and 1265 Citations in Scopus (h-index: 20). There are 73 publications in Web of Science with 1281 citations (1242 not counting own citations) and with h-index: 21. Most of these publications, especially in more recent years, correspond to high impact journals (Q1 and Q2). Additionally, as head of Exercise and Sport medicine Unit in Clinic Hospital he is trying to transfer the knowledge from the elite athlete to impact the health of the general population. Finally, as a Barcelona University is promoting the formation of a new Exercise and Sport Medicine.

ALAN MCCALL,
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Dr. Alan McCall is Head of Research & Development for Arsenal Football Club and scientific consultant for FC Barcelona and the Barca Innovation Hub. He is also joint-Head of Research & Innovation for Football Australia. Alan's background is as a fitness coach and sport scientist with over ten years experience on the field with professional club teams competing in Ligue 1, English Premier League, A-League, Scottish League and UEFA Champions & Europa League competitions. He was Head of Sport Science and fitness coach for the Australian Socceroos at the 2014 FIFA World Cup and the U20 Young Socceroos at the FIFA 2013 World Cup.

Alan is a member of the FIFA Scientific Advisory Board, UEFA's Football Research Group and an Associate Editor for the Science and Medicine in Football Journal as well as the British Journal of Sports Medicine. With >50 scientific publications, Alan's main research interests include performance, recovery, injury prevention and innovation strategy in football. He holds a PhD in 'Injury Prevention in Elite Footballers' from Université de Lille 2 and a Msc in Strength & Conditioning from Edith Cowan University, Australia.

RICARD PRUNA,
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Ricard Pruna is Medical Doctor for Sharja FC and FIFA Medical Centre, Dubai, UAE. Ricard specialises in Sport & Exercises Medicine with a Masters in both 'Traumatology and Sports' and 'Biology and Sports' and additionally holds a PhD in 'Genetics and Injury in Football'. Ricard has a rich and vast experience in top-level football and was the first team doctor of FC Barcelona during 26 years as well as having overseen the Medical Services at FC Barcelona in addition to his first team football duties. Ricard is currently working as medical consultant for the Institut Català de Traumatologia I Medicina de l'Esport (ICATME), Barcelona.

Ricard's clinical interests lie in football medicine, muscle injuries, genetics, return to play, anatomy and injury diagnosis. He has many scientific publications in the football medicine areas and has received various awards for his scientific work, including, the Award for Medical Excellence from the Medical College University of Barcelona, a National and UEFA Award for research in sports medicine.

ROCHELLE KENNEDY,
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Rochelle graduated from La Trobe University in Melbourne with a Bachelor of Health Science, Masters of Physiotherapy Practice. Rochelle has since worked in private practice in Melbourne, treating a wide variety of musculoskeletal injuries. She is currently undertaking her Masters of Exercise Science (Strength and Conditioning) at Edith Cowan University. She has a special interest in managing tendon injuries and has begun research assisting at the La Trobe Sport and Exercise Medicine Research Centre.

LLUÍS TIL,
MD.

Lluís Til has developed his career as an Orthopaedic Surgeon in different hospitals in Catalonia (Vic, Pallars and Terrassa) from 1989 to 2017. Since 1998 he has also worked for the High Performance Centre in Sant Cugat (Barcelona) and since 2003 at FC Barcelona. In both positions, he has worked to enhance the health and performance of elite athletes. Lluís specialises in various topics, such as sports injuries, ultrasound diagnostics and regenerative therapies. He also works teaching junior sports physicians and young physiotherapists. As a Manager, from 2006-2016 he has been in charge of the medical team at Car de San Cugat as well as coordinating FC Barcelona doctors.

Between 2017 and 2019 he has been the Clinical Director at Sport Lisboa e Benfica, the biggest club in Portugal. During the years 2019 and 2020 he was the Chief Medical Officer at AS Monaco, the club from the Principality of Monaco that competes in the French league. Starting from September 2020, Lluís returned to FC Barcelona as the Medical Director and Team Doctor of the football men's first team.

Experts

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General Section Part 1

— Martin Hägglund, Markus Waldén

1.1 THE TENDON INJURIES LANDSCAPE IN FOOTBALL

FREQUENCY AND NATURE OF TENDON INJURIES

Tendon injuries are a frequent cause of training modification in football. Although many players can continue to train and play with symptomatic tendinopathies, some are unable to continue playing. Data from FC Barcelona show that approximately 30% of tendinopathies in professional athletes led to time loss from play (1). This means that measuring tendon injuries with a time-loss definition (missed training and match play) likely represents only the tip of the iceberg (2), and many players with tendon pain are not recorded as being injured.

In the UEFA Elite Club Injury Study (ECIS) of European top-level male teams, tendon injuries represent 7% of all time-loss injuries (3), with a similar proportion reported in English professional players (4). The overall incidence of tendon injury is 0.6 /1000 hours of football training and match play, corresponding to four time-loss tendon injuries per team each season on average (3).

Most tendon injuries result in short duration of absence from training and matches, as a short absence from the high loads of training and playing can decrease tendon pain substantially. In the UEFA ECIS cohort, 29% of tendon injuries were slight or minimal (0-3 days absence), 22% minor (4-7 days), 31% moderate (1-4 weeks) and 18% severe (>4 weeks) (3). However, the player who continues to train and play may experience negative effects on their function and performance, evident in slower change of directions, slower sprint speed and reduced jump height.

The location of tendon injuries is dependent on playing position. Outfield players who run and sprint repeatedly have almost exclusively lower limb tendon injuries (>90%), while goalkeepers who catch and throw the ball as well as dive, incur approximately 40% of tendon injuries to the upper extremities (Figure 1). Tendon injuries appear less common in female elite players, representing 3% of all injuries in the Swedish women's top division compared with 11% in the men's top division (5) (Table 1). Specifically, Achilles and patellar tendon injuries were twice as common in male than in female elite players in a cohort of Northern European elite teams (6). Similar findings are reported from FC Barcelona with a two-fold higher incidence of tendinopathy in male than female players (1).

Figure 1. Location of tendon injuries in elite football from the UEFA-ECIS cohort.
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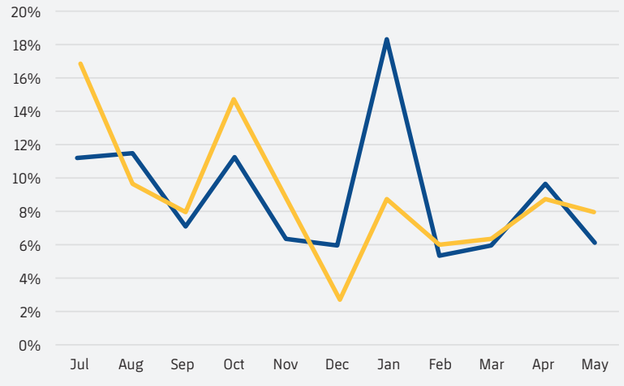
		NUMBER OF INJURIES	ATHLETE EXPOSURE (HOURS)	INCIDENCE (95% CI)
GENDER	**Female**	60	485	12.4 (9.4- 15.9)
	Male	783	3354	23.3 (21.7- 25.0)
CATEGORY	**Youth**	360	2495	14.4 (13.0- 16.0)
	Professional	483	1344	35.9 (32.8- 39.3)
SURFACE	**Outdoor**	385	2277	16.9 (15.3- 18.7)
	Indoor	458	1562	29.3 (26.7- 32.1)
TIME-LOSS	**No**	546	2887	18.9 (17.4- 20.6)
	Yes	297	2771	10.7 (9.5- 12.0)
SEVERITY	**No time loss**	546	2887	18.9 (17.4 -20.6)
	1-3 days	76	1062	7.2 (5.6- 9.0)
	4-7 days	82	1221	6.7 (5.3 -8.3)
	8-28 days	98	1333	7.4 (6.0- 9.0)
	>28 days	41	674	6.1 (4.4- 8.3)

^ Table 1. Factors affecting incidence of tendinopathy (3).

GRADUAL ONSET TENDINOPATHIES

Four out of five tendon injuries occur with a gradual onset of pain. The Achilles and patellar tendons are the two most commonly affected sites, representing 1-3% of all injuries in professional male players (Table 2)(7, 8). Other tendinopathies, e.g. the peroneus, tibialis posterior and quadriceps tendons, infrequently cause time-loss from play, representing less than 0.3% of injuries. Both Achilles and patellar tendinopathy show seasonal variations, with a high proportion of injuries occurring in the pre-season (7, 8). A higher prevalence is also seen after a mid-season break (Figure 2).



< Figure 2. Season distribution (%) of Achilles and patellar tendinopathy in elite football (7, 8).
Achilles tendinopathy
Patellar tendinopathy

	% OF ALL INJURIES	INJURIES/ 1000 H	INJURIES PER TEAM	MEDIAN ABSENCE	RE-INJURY
Achilles tendinopathy	1.9	0.13	1 per season	9 days	23%
Patellar tendinopathy	1.1	0.08	1 every 2nd season	8 days	22%
Achilles tendon rupture	0.1	0.01	1 every 15th season	5.5 months	7%
Patellar tendon rupture	<0.1	0.005	1 every 31st season	4 months*	8%

*Patellar tendon ruptures included mainly injuries diagnosed as partial tears

^ Table 2. Incidence of Achilles and patellar tendon injuries in elite football (3).

ACUTE TENDON RUPTURES

Whilst one-fifth of tendon injuries are reported to have an acute symptom onset, complete acute tendon ruptures are quite uncommon, comprising only 4% of all tendon injuries in professional football (3). A team with 25 players can thus expect one Achilles tendon rupture every 15th season (Table 2). The absence from football following Achilles tendon repair in the UEFA ECIS was 5.5 months on average (8), whilst return to training in 4 months has been described in a single case study of a professional player (9). A recent systematic review on professional players showed an average of 7 and 9 months to return to training and match play, respectively, after Achilles tendon rupture and surgical repair (10).

Patellar tendon ruptures are even more uncommon, with only one recorded injury per team every 31 seasons on average. The absence following a patellar tendon rupture in the UEFA ECIS was around 4 months (7), but the sample was small and most injuries were classified as partial tears. These could equally be classified as severe patellar tendinopathy as there is currently no consensus regarding diagnosis and classification of partial tendon tears, and this is a limitation in the reporting from most sport injury surveillance data including the UEFA ECIS. Total patellar tendon ruptures always require surgery, and this results in longer rehabilitation with an average of 10 months to return to play (11).

Barça Way

FC Barcelona has five elite team sports (football, basketball, roller hockey, futsal at handball including men's, women's and junior levels). In 2019, we published an overview of tendinopathies across 8 seasons in these sports (1) (Table 3).

- The incidence rate is twice as high in male compared to female athletes, with the most frequently injured tendons being the patellar and Achilles.
- Men had higher incidence of adductor tendinopathies while women had more proximal rectus femoris tendinopathies.
- In Academy (junior) teams, the rate was one third of what it is among professional teams.
- Despite a high prevalence of tendinopathies in team sport athletes, our data also suggested that most athletes were able to continue playing despite tendinopathy. Approximately two thirds of tendon injuries did not result in any missed training or matches.

	Level	NUMBER OF INJURIES	ATHLETE EXPOSURE (HOURS)	INCIDENCE (95% CI)
Basketball	Professional	140	203	69.9 (58.0- 81.4)
	Youth	107	422	25.4 (20.8- 30.6)
Football	Professional	136	411	33.1 (27.8- 39.1)
	Youth	113	1052	10.7 (8.9- 12.9)
Woman's football	Professional	39	257	15.2 (10.8- 20.7)
	Youth	21	228	9.2 (5.7- 14.1)
Futsal	Professional	55	152	36.2 (27.3- 47.1)
	Youth	21	177	11.9 (7.3- 18.1)
Handball	Professional	66	248	26.6 (20.6- 33.9)
	Youth	71	478	14.9 (11.6- 18.7)
Roller Hockey	Professional	47	73	64.4 (47.3- 85.6)
	Youth	27	138	19.6 (12.9- 28.5)

^ **Table 3.** Incidence of tendinopathy based on sport participation at FC Barca.

Summary:

- Tendon injuries are common in football players, however, the majority of players with a tendinopathy are able to continue to train and play. The true prevalence of tendon injuries is underreported as they often do not result in time loss from football.
- The most commonly affected tendons are the Achilles and patellar, representing 1-3% of all injuries in professional football.
- Tendon symptoms can negatively affect performance, such as ability to sprint, jump or change direction at speed.
- Tendon injury location varies with playing position due to different demands, and also playing level. Junior players are less commonly affected.
- Most tendon injuries in football are of gradual onset (tendinopathy) and usually occur after a change in training load, such as return to load in the pre-season or after the mid-season break.
- Tendon ruptures are quite uncommon, but cause substantial time loss from training and competition.

Clinical Implications:

- Athletes with tendinopathies can continue to train and play, however, it can impact aspects of the athlete's performance. In-season management of tendinopathy should consider this.
- Load monitoring or other strategies to minimise fluctuations in tendon load are important.
- The risk of developing tendinopathy is higher in the pre-season period.

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— Jill Cook and Craig Purdam

1.2 WHAT IS THE PATHOPHYSIOLOGY AND PATHOAETIOLOGY OF TENDON INJURIES?

Normal tendons are a composite matrix, comprising cells (tenocytes), collagen (primarily type 1) and mostly small proteoglycans as well as a broad array of complementary elements. Tendon load such as compression at bony entheses alter the collagen and proteoglycan content, and an increase in type 2 collagen and larger proteoglycans that attract and hold water is seen in these regions (1). Tendons are surrounded by a peritendon, differentiated tissue that supports the vascular, lymphatic and sparse nerve supply (Figure 1)(2).

Tendons are arranged hierarchically; collagen fibres and associated proteoglycans are bundled into fascicles surrounded by connective tissue (endotendon or inter-fascicular matrix (IFM)) that supports some vascular, neural and lymphatic structures (Figure 2). Tendons rely on slide and rotation between the fascicles to facilitate energy storage, which is a key function of athletic tendons (Figure 3) (3). The intra-tendinous connective tissue is continuous with the peritendon structures.

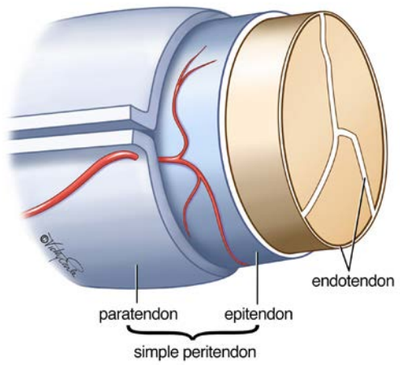

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Figure 1. The complexity of tendon connective tissue

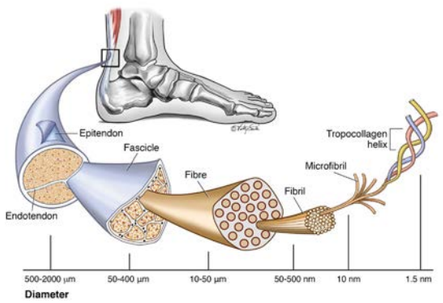

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Figure 2. Tendon collagen hierarchy

Peritendinous structures vary in complexity depending on the loads placed on them. Tendons where there is a lot of movement between the tendon and the surrounding tissues have a complex tenosynovial peritendon structure to facilitate this movement, examples are the foot and ankle tendons (tibialis posterior, peroneal tendons). Tendons where the movement is substantial but not excessive have a tenovagium, which is a series of membranes that allow gliding between the tendon and surrounding structures (Achilles) (4). Other lower limb tendons that have little movement have simple peritendon structures (patellar tendon, hamstring tendon).

Normal tendons maintain their matrix and hierarchical structure through a balance of tissue breakdown and synthesis (5) and can adapt to changes in load (6, 7). Tendons retain homeostasis at both a cellular and tissue level within an ideal window of load, with both significant under- or overload leading to changes in the matrix. Repeated energy storage loading during sport leads to mechanical adaptation of the tendon to these higher loads at both the inter-(8) and intra-fascicular level (Figure 4) (9). Relative rest, perhaps greater than 2- 3 weeks, leads to a reduction in mechanical capacity of the tendon (10), leaving potential for overload.

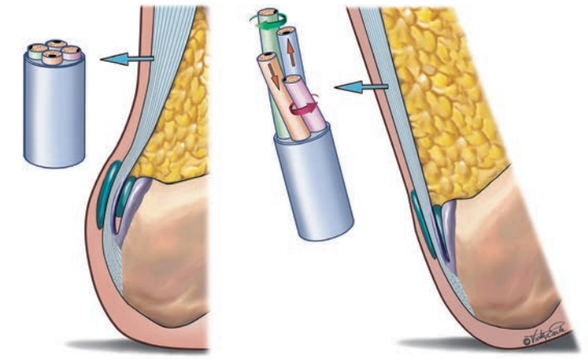

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Figure 3. How interfasicular sliding contributes to energy storage in a tendon

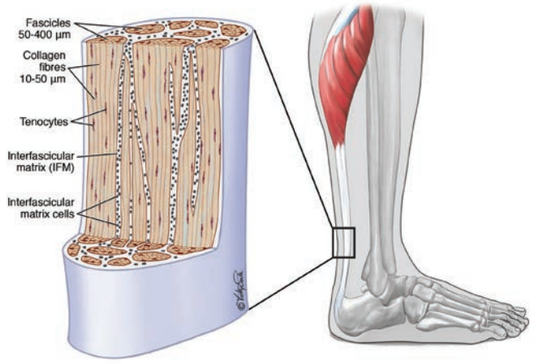

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Figure 4. Interfasicular matrix

TENDON PATHOLOGY

There are several models of the pathoaetiology of tendon pathology. Some models implicate inflammation as the primary factor (11, 12), some propose failed matrix healing after injury (13, 14), and others suggest the cell is the primary driver (15). A continuum model based on a cell driven response describes four primary states of tendon pathology (reactive, dysrepair, degenerative and reactive on degenerative) (Figure 5) (16). Whatever the aetiology, the pathology appears to be the same in all tendons (17, 18). A caveat is that many histopathological specimens are from people with long term tendon pathology and tendinopathy and the pathology is mostly degenerative.

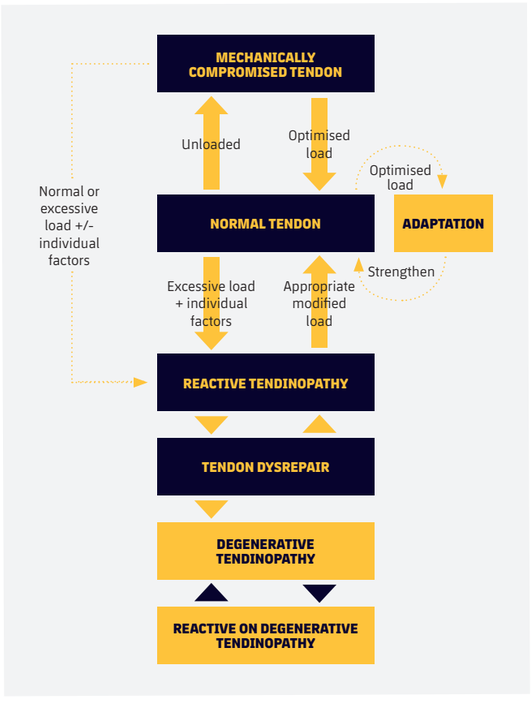
Reactive tendon pathology has upregulated tenocytes and increased proteoglycans (15, 19) (Table 1). This early stage of tendon pathology is characterised by an increased tendon size and an increase in some anabolic/ catabolic signalling, however the mechanical tissue properties of the tendon remain unchanged (20). Models of acute overload following fatigue loading of tendons have demonstrated changes in the tendon structure and matrix, with cellular changes and a 'loosening' of the tightly packed matrix (21, 22) with no evidence of frank collagen tearing. These changes mirror our understanding of early tendon pathology, and support the notion that no major disruption to the tendon matrix is necessary to initiate a tendinopathy (20). These (likely) non-inflammatory changes may be within the inter-fascicular matrix rather than within the fascicle bundles, as a result of excessive sliding in the inter-fascicular matrix in energy storage and release loads (23). Once irritated, the tenocytes appear to remain sensitised for a considerable time (24). The tenocyte sensitisation in intact younger tendon appears to be strongly communicated through cytokine signalling between the cells (25, 26), mimicking some signs of inflammation but with lower cytokine levels than a traditional inflammatory response. Imaging demonstrates a fusiform increase in tendon thickness with a homogeneous, normal arrangement of the matrix with diffuse interstitial swelling. Some capacity appears to exist for normalisation of these matrix changes at this stage (27).

Dysrepair describes similar cell and inter-fascicular matrix changes with greater within fascicle change resulting in preliminary breakdown of the hierarchical structure and probable loss of function of the inter-fascicular matrix and progression towards fusion. This less reversible pathology is more heterogenous on imaging.

Degenerative pathology involves further destruction of the hierarchical organisation of the tendon, with change to both the tendon cells and the matrix. Regions of degenerative pathology have little organised type I collagen (more type II and III) and are unlikely to be able to transmit tensile loads. The inter-fascicular matrix is no longer evident. Degenerative pathology may be heterogenous and scattered throughout the tendon (Achilles) or a central core

	NORMAL TENDON	REACTIVE TENDON	DEGENERATIVE TENDON
Cells	Tenocytes	Potential change in cells in inter-fascicular matrix	Chondrocytic cells
Proteoglycans	Mainly small proteoglycans	Change in proteoglycans in the inter-fascicular matrix	Larger proteoglycans
Collagen	Mainly Type I	Collagen unaltered	Increase in Type II and III collagen
Connective tissue	Normal inter-fascicular matrix	Inter-fascicular matrix intact but altered	No inter-fascicular matrix
Vessels	Vessels found in the connective tissue throughout the tendon	Vessels found in the connective tissue throughout the tendon	No connective tissue. Vessels are increased and random throughout
Nerves	Nerves peripheral and close to bone and muscle junction	Nerves peripheral and close to bone and muscle junction	Nerves peripheral and close to bone and muscle junction

^ **Table 1.** Changes in tendon components with pathology.



(patellar tendon). Critically, there is usually considerable volume of normal tendon surrounding the degenerative pathology (28). Imaging reveals increased tendon thickness with hypoechogenic areas and variably increased vascularity. This vascularity is likely opportunistic (29), and does not provide improvement in tissue perfusion or facilitate repair (30).

Reactive on degenerative describes reactive tendon pathology in the normal part of a degenerative tendon. A degenerative tendon cannot take load because of its lack of matrix structure, when load is placed on a degenerative tendon it is the normal part of the tendon that takes the load. If the load exceeds the tendon capacity, then reactive changes can occur in the region with normal structure. The US or MRI imaging shows a typical area of tendon degeneration, but it is difficult on imaging to detect the reactive changes in the normal part of the tendon.

Figure 5. Continuum model of tendon pathology
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FAT PAD

The Achilles (Kager's fat pad) and the patellar tendon (Hoffa's fat pad) are intimately linked to an adjacent fat pad (Figure 6). The fat pad is a source of cytokines and is the path for tendon vessels entering the tendon (31). The fat pad can become hyperechogenic on imaging. Exactly what changes occur in the fat pad in tendon pathology is unknown (32).

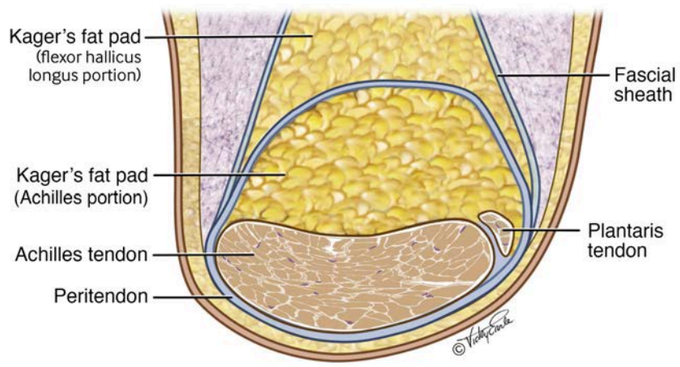

Figure 6. Peritendon structures in the Achilles.
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PERITENDON PATHOLOGY

Pathology can occur in the peritendon structures, where the overload is excess movement between the tendon and surrounding structures. Tendons with peritendon structures such as tenosynoviums or tenovagiums develop peritendon pathology, tendons with simple peritendon structures do not. Whether inflammation is a key part of the pathology is unclear (33). Peritendon pathology can be diagnosed on imaging and can co-exist with intra-tendinous pathology.

THE TENDON ENTHESIS (BONE TENDON JUNCTION)

The mid-Achilles is the only tendon to suffer a true tendon pathology, in the remaining lower limb (and most upper limb) tendons pathology occurs at the attachment of the tendon to bone. This is a complex attachment (Figure 7), that is designed to attenuate forces across the transition from tendon to bone (34). The fibrocartilage differs from mid-substance tendon (more type II collagen and larger proteoglycans) and is present in both the tendon and on the associated bony surface, to deal with the high compression loads proximal to the attachment. There is also bursa in the region to reduce friction between structures.

When the insertion becomes pathological, usually in response to excess combined compression and tensile loads (35), there are changes throughout the enthesis. First, there is a response in the tendon with an increase and change in collagen and proteoglycans that causes the tendon to enlarge. The bursa can also become larger (stromal thickening) (36), and inflammation may be present in the bursa. However, a histopathological study of patients diagnosed with greater trochanteric bursitis found no evidence of acute or chronic bursal inflammation, therefore, the role of inflammation in 'bursitis' may be questioned (37). As pathology progresses, some changes in the bone (such as the development of osteophytes) may be evident. These changes actually increase the compressive forces at the enthesis, which results in a vicious cycle of overload, further tissue changes and even greater overload.

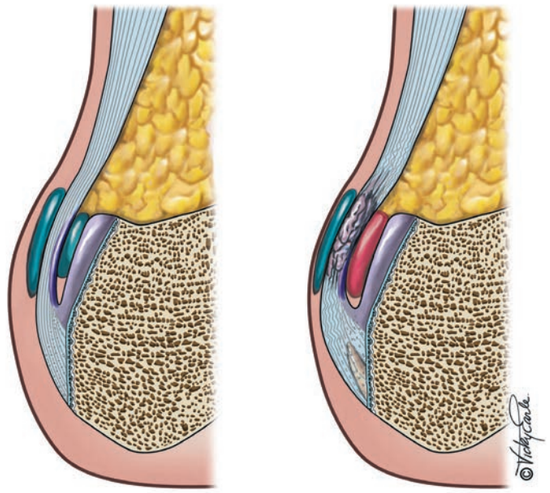

Figure 7. Normal vs pathological tendon enthesis. Note changes in both the bursa and the tendon of the pathological enthesis.

WHAT ROLE DOES MECHANICAL LOAD PLAY IN TENDON PATHOLOGY?

The changes seen in tendon pathology are linked to load, although absolute understanding of exactly what load (refer to tendon load section), and how much is excess is unknown and likely individual (dependent upon factors such as genetics, morphology, sex). What is known is that accumulation of load over time is linked to tendon pathology, the more load a tendon experiences the higher the chance of pathology (38) (Table 2). Thus, an association exists between older age and tendon pathology, as an older person has accumulated more tendon load throughout their life, thereby increasing their risk of pathology. Similarly, a tendon in a young person exposed repeatedly to high tendon load (gymnastics) can become pathological in adolescence.

	ACHILLES TENDINOPATHY %		ACHILLES RUPTURE %	
Population	**<45 years**	**Lifetime**	**>45 years**	**Lifetime**
Football Players	23%	28%	7%	12%
All Sports	18.2%	23.9%	5.4%	8.3%
Controls	2.9%	5.9%	1.2%	2.1%

^ **Table 2.** Cumulative Incidence of Achilles Tendinopathy or Rupture in Former Athletes and Controls (38).

TENDON HEALING AND REPAIR

The loss of the hierarchical tendon structure in the dysrepair and degenerative stages of tendon pathology is likely irreversible, and it is unlikely any intervention can normalise tendon structure in adults. It seems the tendon responds to pathology by increasing the amount of normal tendon in parallel to the region rather than repairing the area of pathology (Table 3)(39). Importantly, the cross-sectional area of normally aligned fibrillar structure within the matrix is generally greater in pathological tendons than normal tendon (39) (Table 3). This maintains capacity of the tendon to meet the demands of activity.

ACHILLES TENDON	NORMAL TENDON	PATHOLOGICAL TENDON
AP diameter (mm)	6.5 ± 0.5	8.4 ± 1.5
Mean cross-sectional area of poor structure (mm^2)	1.4 ± 1.4	4.7 ± 8.3
Mean cross-sectional area of good structure (mm^2)	**80.8 ± 15.8**	**94.8 ± 26.5**

PATELLAR TENDON	NORMAL TENDON	PATHOLOGICAL TENDON
AP diameter (mm)	6.0 ± 0.6	7.8 ± 2.6
Mean cross-sectional area of poor structure (mm^2)	4.5 ± 3.4	17.1 ± 22.3
Mean cross-sectional area of good structure (mm^2)	**125.9 ± 11.7**	**139.9 ± 23.1**

^ **Table 3.** Pathological tendons have sufficient good tendon structure (28).

Reactive tendon pathology that may occur in the inter-fascicular matrix appears to be a reversible pathology, as the tendon fascicles and the intra-tendon connective tissue remain intact. It is possible that this stage is simply the tendon adapting to load and developing better mechanical properties, however what constitutes tendon adaptation (39), and when it becomes a reactive tendon pathology is not known.

PATHOLOGY AND PAIN

There is a disconnect between pathology and pain in most, if not all non-traumatic, musculoskeletal conditions (40) including tendinopathy. This has several important clinical implications; it explains why isolated tendinopathy is a clinical diagnosis and imaging is not required (41) and may be misleading (see section 2.3), it enables understanding of why pathological tendons may be asymptomatic, and helps to explain why imaging is unchanged following successful improvement in pain and return of function (42).

To go a step further, pain is an output from the brain following multiple inputs that include nociception, environmental cues, memory and emotion. The nociceptive input from tendon in the production of pain is a key driver in the tendon pain experience, the clinical presentation of tendon pain shows that pain is intimately linked with load (increased load = increase in pain) and remains well localised regardless of the length of time of symptoms. The transmission of nociceptive information from the tendon is via a primary afferent neuron to a secondary neuron in the dorsal horn of the spinal cord. This occurs via the spinal thalamic tract and this secondary neurone is likely a nociceptive specific neuron rather than wide dynamic range neuron, which results in a lack of pain spread.

Exceptions to this exist where nociception comes from other structures such as bursa (gluteus medius tendinopathy can refer down the leg), and peritendon structures where pain spread occurs. Different physiology underpin these conditions and accurate differential diagnosis is critical for optimal management.

Activation of polymodal ion channels (activated by different stimuli) on a sensory neuron located outside the tendon may drive nociception (43). These ion channels must be mechanosensitive (load responsive), as well as potentially activated by changes in the biochemical environment (pH for example) however the exact receptor is not known (44). Tendon warm up phenomenon may be a result of ion channel saturation, and ion channel features may also explain why tendinopathy causes increased pain 24 hours after high tendon load.

After tendon nociception is triggered, there is modulation at a spinal cord level through the interneurons and glia that surround the synapse, including the potential for facilitation (amplifying nociceptive signal) or inhibition (reducing nociceptive signal). Importantly the clinical features of tendinopathy may be explained by homosynaptic sensitisation, meaning that the pathway of activation is facilitated over time without pain spread (44-48). This may be different in tendons of the lower limb compared to the upper limb (49). The secondary neuron arrives at the brain via the

thalamus where the brain decides whether to produce pain. Many regions are involved in this activation pattern including the motor and sensory cortex, anterior cingulate cortex (complex cognitive function including emotion), insula (empathy, motor control) periaqueductal grey matter, dorsolateral prefrontal cortex (planning), visual and auditory areas. When these neurons and brain regions are activated a neurotag that represents tendon pain is formed for that person.

Little is known about the source of nociception in tendons, several structures such as increased vascular or sensory neural supply fail to explain the clinical presentation. Neural supply is unchanged with pathology (43) and substantial tendon pathology may be asymptomatic (2). Glutamate, an important substance in ion channel activation has been shown to be high in tendinopathy (50, 51). however resolution of tendon pain with rehabilitation did not change glutamate levels (52).

There is evidence for increased corticospinal excitability and inhibition in relation to motor changes in patellar (53) and elbow tendinopathy (54), that may affect the control of movement. However, athletes with patellar tendinopathy are exceptional jumpers (known as the jumpers knee paradox) (55) and is a clinical example of how complex this condition is.

There is no evidence of secondary hyperalgesia (sensitivity) in association with the lower limb tendinopathies. There is evidence of peripheral sensitivity (sensitive over the tendon and likely related to homosynaptic sensitisation) (49), indicating that tendons are sore to palpate. There is no evidence of deficits to left / right judgement in tendinopathy that has been shown in other persistent pain conditions (56).

There is some debate in tendinopathy about the relative contribution of nociception and central changes. There are alterations to how the body perceives stimuli even following an ankle sprain (57), this is protective (increased sensitisation – on alert) and with proper management this sensitivity abates. This indicates that the central sensitisation should not be the focus of management and education is a critical intervention in all clinical presentations of tendinopathy.

THE TENDON IN ADOLESCENCE

Most tendons in adolescence have an apophysis, where the bone tendon junction can mature as the skeleton grows. Tendon injury during puberty can affect the apophysis, causing pain (such as in Sever's disease at the Achilles insertion) (Figure 8) or frank injury (ischial tuberosity avulsion of the hamstring tendon). Common sites where the apophysis is disrupted include tendon attachments around the pelvis, especially in young men, and around the elbow in throwing athletes.

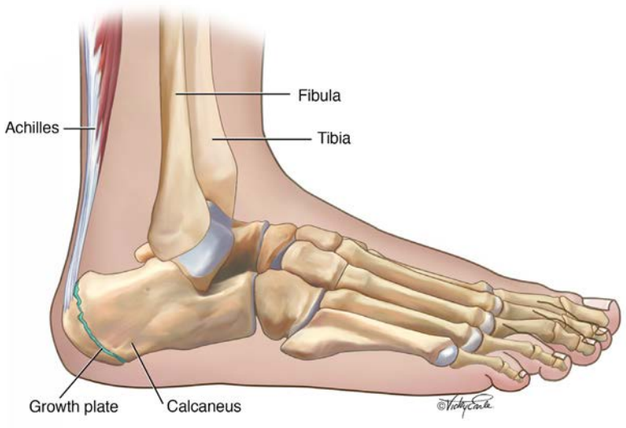

Figure 8. Achilles tendon apophysis
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The patellar tendon may be the exception to this, most patellar tendon pathology begins in adolescence (58). The patellar tendon does not have an apophysis but matures through a cartilage plate at both the proximal (59) and distal (60) end. These appear to be vulnerable to overload when the bone tendon attachment is maturing (59, 60) leading to tendon pathology that is indistinguishable from pathology seen with load through life (61). This is clinically important because tendon pathology that develops during puberty will remain through life.

THE TENDON IN AGEING AND DISEASE

Significant cellular, structural and mechanical changes within the tendon occur as part of the ageing process (62). Increasing age is associated with decreased potential for cell activity and density (62). Small increases in tendon cross-sectional area and enzymatic cross links may occur, but these changes are often accompanied by small decreases in collagen content, fibril diameter and proteoglycan content (62). The only consistent and major compositional change associated with age is an accumulation of non-enzymatic advanced glycation endproduct (AGE) cross-links, which may occur erratically throughout the tendon (62). Persuasive evidence indicates that cell turnover within the core of the tendon after maturity is very slow or completely absent (62). Tendon fibril diameter, collagen content, and whole tendon size seem to be largely unchanged with ageing (62). Ageing appears to be associated with reductions in both modulus and strength, which may result in increased risk of injury if this is not offset with the positive adaptations associated with exercise (62). Some age-related changes may be mediated by an increase in the prevalence of lifestyle-related conditions such as diabetes and high cholesterol.

Tendons are also affected by systemic disease. A direct link is clear with enthesitis in seronegative auto-immune conditions (such as psoriatic arthritis), but less direct links have also been found to conditions such as diabetes (63) and elevated cholesterol (64). Both diabetes and hypercholesterolaemia may affect the structure of the tendon. In diabetics, collagen cross links may be altered due to the presence of increased advanced glycation endproducts. Elevated cholesterol may result in the deposition of low-density lipoprotein (LDL) cholesterol in the collagen matrix, as LDL cholesterol has a high affinity for decorin, which is the primary proteoglycan in normal tendon.

Summary:

- Normal tendons are a composite matrix composed of tenocytes, collagen, and mainly small proteoglycans, along with other complementary elements.
- Tendons are arranged hierarchically, with fascicles surrounded by connective tissue, which supports the vascular, lymphatic and neural structures of the tendon.
- Tendons maintain their matrix and hierarchical structure through a balance between tissue breakdown and synthesis in response to load. Both significant under- or overload of the tendon may disrupt this balance and lead to pathological changes in the tendon matrix.
- The continuum model of tendon pathology describes four primary stages of tendinopathy; reactive, dysrepair, degenerative, and reactive-on-degenerative.
- The loss of hierarchical structure in stages of dysrepair or degeneration are likely irreversible. However, tendons adapt to pathology by adding normal tendon tissue in parallel to pathological regions. This maintains the capacity of the tendon to meet the demands of activity.

Clinical Implications:

- Load is very likely implicated in the pathogenesis of tendon pathology, with both significant under- or overload potentially leading to tendon matrix changes.
- As tendon pathology progresses through the continuum, the tendon adapts to irreversible changes by adding more normal tendon tissue in parallel to the pathological region. Consequently, the tendon is able to maintain capacity to meet the demands of activity. Therefore, it is still possible for athletes with pathological tendon changes to attain a high functional capacity with appropriate rehabilitation.
- Tendon pain is poorly understood, and tendon pathology does not equate with pain.
- A tendon's response to load changes throughout life, adolescent tendons respond structurally to load, whereas older tendons respond with mechanical adaptations.

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1.3 HOW DO TENDONS RESPOND TO LOAD?

Tendon load is essential to maintain tendon structure, mechanical properties and capacity, but excess load is linked to pathology. Different types of tendon load impact on the tendon structure and tendon pain. Tendons experience three different types of loads; tensile load, compressive load and friction load. Importantly, it is combinations of these loads that can be the most provocative for tendons (1).

TENSILE LOAD

In low load situations tendons transfer muscle action to bone, however, this is not the primary action of a tendon in an athlete. The highest tensile load is where the tendon is used like a spring to improve speed of movement and reduce the metabolic cost of sporting activity, that is, to store energy in the tendon to immediately release it to produce movement (Table 1).

This activity must be a fast action as tendon is viscoelastic (2), therefore slower movements do not result in energy storage. It is faster and more efficient to use the energy stored in the Achilles tendon to propel the athlete rather than to use a concentric and eccentric contraction of the calf muscle complex (Figure 1) (3, 4). Slower musculotendinous movements, for example those involving weights, apply slower tensile load on tendons, and do not reach the high magnitudes of tendon load associated with energy storage and release (Figure 2).

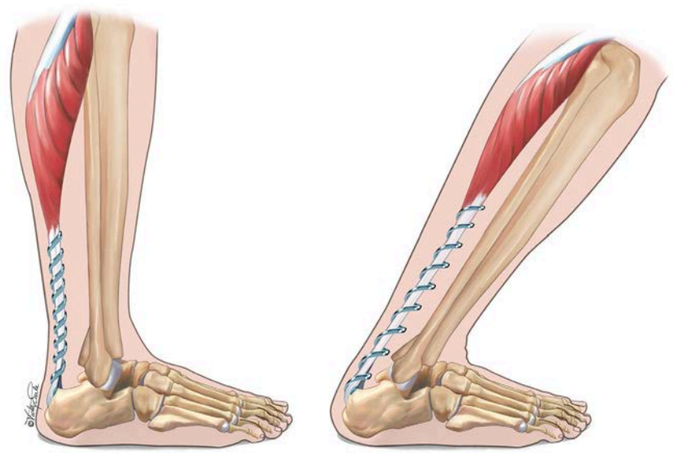


Figure 1. Tendon energy storage. Note the spring is stretched with dorsifelxion.

If a tendon is painful then reducing tensile load on the tendon can be beneficial and generally requires a reduction in the amount of high speed loading. Consideration should be given to force per step, type of load and rate of load and to consider cumulative load. For example the Achilles tendon in 10 kilometers of running sustains a total of 3,750 steps (per limb) and a bodyweight of 80kgs would equate 1.5million kg of force for each Achilles tendon. This insight can help athletes comprehend why an additional 1 kilometer of running can cause an increase in symptoms. Additionally, loads encountered external to a players structured training can offer an insight into the symptom variation that is often missed when only considering training loads. For example, amassing an additional 5000 steps per day is equivilent to approximately 10,000BWs of force through the Achilles tendon. Similar load consideration for other tendons allow tensile loads to be modified through amended training loads.

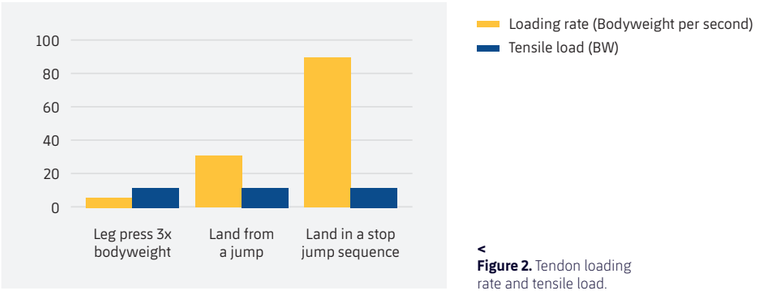


Figure 2. Tendon loading rate and tensile load.

TENDON	HIGH TENSILE LOAD ACTIVITIES
Achilles	Sprinting, faster running, change of direction
Patellar tendon	Deceleration, change of direction, jumping
Hamstring tendon	Running uphill, fast lunging
Adductor tendon	Change of direction

Table 1. High tensile load activities.

COMPRESSIVE LOAD

Compression occurs when a tendon abuts against another structure, most commonly bone. This occurs primarily at the tendon bone junction, usually just proximal to the insertion. Tendons can be compressed away from their insertion, tibialis posterior is compressed around the medial ankle malleolus, substantially proximal to its insertion. Compressive load occurs in most lower limb tendons, but is not evident in the patellar tendon and is debatable in the adductor tendons. Other sources of compression include retinacular structures and intrusions such as pathology into a tendon's space, and external compression such as tight ankle taping or socks can cause a negative tendon or peritendon response.

A plantaris tendon close to the Achilles tendon can cause compression of the Achilles that can result in a tendon or peritendon response (Figure 3). Whether compression is highest in plantar or dorsiflexion has been debated. There are some Achilles tendons that have an invaginated plantaris that seem to result in pathology in the plantaris itself, a different response to the plantaris that is close to the Achilles that cause a response in the Achilles tendon.

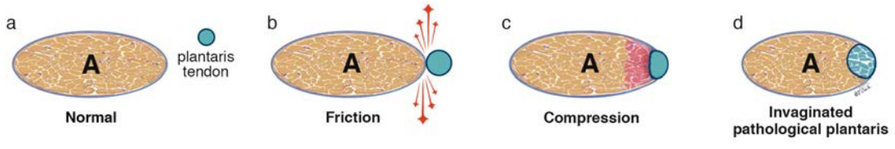
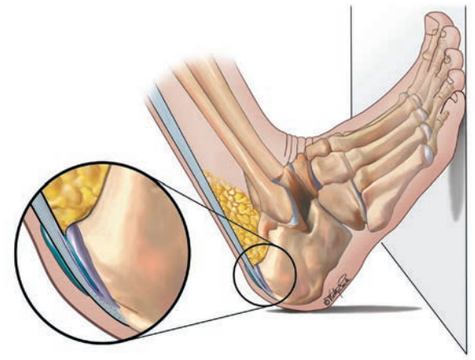


Figure 3. Plantaris variants.

Compression is greatest towards the end of muscle tendon length. The Achilles tendon is compressed against the superior aspect of the calcaneus in ankle dorsiflexion (for example in football when an athlete pushes off from a dorsiflexed position) (Figure 4) and the hamstring tendon is compressed against the ischial tuberosity in hip flexion (such as running when leaning forward). Reducing compressive loads can improve tendon pain (5).



Stretching can increase the compressive load on a tendon. Therefore, stretching as a treatment for tendinopathy is not recommended. Similarly, ensuring that a tendon is managed in inner range in early rehabilitation will reduce compressive loads. For example, a substantial heel raise can reduce pain in many foot and ankle tendons (Table 2).

Figure 4. Compression in dorsiflexion. Note compression of the Achilles tendon against the calcaneus.

TENDON	REDUCES COMPRESSION	INCREASES COMPRESSION
Achilles	**Heel raise** • Use shoes with a higher heel • Wedge sole of football boot	**Have a flat or negative heel position** • Walking in bare feet **Calf raises off a step** **Stretching**
Hamstring	**Modify sitting** • Reducing hip flexion angle in weight bearing • Reduce sit time and hard chairs • Relieve pressure on ischial tuberosity in sitting	**Weight bearing mid to end range hip flexion** • Squats, deadlifts in deep hip flexion **Stretching**
Adductor	**Reduce exercise into end range abduction**	**Stretching**
Patellar		**Stretching**

Table 2. Simple strategies to change compressive forces for different tendinopathies.

FRICTION LOAD

Friction load occurs between the tendon and the surrounding structures. The tendon develops a peritendon to manage this load; tendons where substantial movement occurs have a sophisticated peritendon that includes a synovial membrane, for example the foot and ankle tendons. Tendons with less movement such as the patellar, hamstring and adductor tendons have a vestigial peritendon and therefore have no clinical presentation of peritendinopathy. The Achilles tendon falls in between, with a series of gliding membranes, instead of a synovial structure. Excess movement between the tendon and surrounding structures can cause a response in the peritendon. Clinical examples of this include the football player who complains of Achilles region pain in the absence of high tensile loads, such as during cycling, where the Achilles is exposed to repeated plantar and dorsiflexion friction loads but not high tensile loads.

COMBINATION LOAD

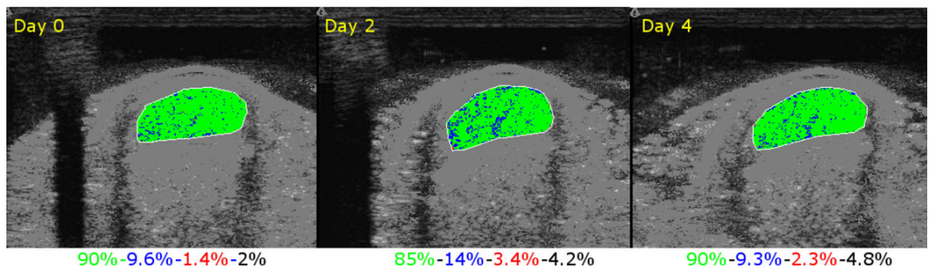
Combination loads are common in a sporting environment. The combination of spring like tensile loads with compression has been shown to be more provocative than each load in isolation (1). For example pushing off in end range dorsiflexion will place high compressive and energy storage loads on the tendon. Similarly, friction loads with an external compression will increase the response of the peritendon, for example a tight sock or tape during a training session can cause peritendon irritaion.

WHEN AND HOW DOES A TENDON RESPOND TO LOAD?

Tendon response to load may not always be clinically apparent, tendons can progress through the pathology continuum to the degenerative stage while remaining asymptomatic (6). Imaging has been used to assess the tendon response to load, with both short and long term, positive and negative changes being observed. It is unclear if short term changes are maladaptive or simply part of the normal phyiological and/or adaptive response of the tendon.

TRANSIENT RESPONSES

When subjected to high magnitude, short-term loading, both the Achilles in humans (7), and the superficial digital flexor tendon in horses demonstrate changes on imaging (Figure 5) (8). These changes were distributed throughout the tendon and were most apparent two days following exposure to load, with changes appearing to have resolved by day four (8). It could be argued that these changes occur in the inter-fasicular matrix, as energy storage and release loads primarily occur in this tissue (9), and that changes in water or proteoglycans content may resolve if no further load is placed upon the tendon.



^ **Figure 5.** Percentage of aligned fibrils at baseline, 2 days post game and 4 days post game. Note the decrease in green at day 2, and return to baseline by day 4.

ADAPTIVE RESPONSES

Persistent loads that require a tendon to improve its capacity may result in mechanical and/or structural changes (10). Achilles tendons that were exposed to an Australian football pre-season showed improvements in structure over 5 months, however several tendons also demonstrated a negative response and developed pathology (11). Improvements in mechanical properties results in greater tendon stiffness that enables the storage of more mechanical energy, which results in a greater overall capacity of the tendon.

Structural changes (an increase in tendon cross-sectional area) are more likely in younger athletes, older people respond with improvements in mechanical properties (tendon stiffness). Studies using carbon-14 pulse dating found that the core of the tendon is formed during the first 17-years of life (12), with extremely limited turnover thereafter, indicating that is the adult tendon matrix remains relatively inert in the mature human tendon (13).

The exception to this may be pathological tendons, as a study using the same method found that tendon pathology was preceeded by several years of abnormally high collagen exchange (14). Whether this abnormally high collagen turnover is a symptom of disease, or a risk factor for the eventual development of pathology is not clear (14).

IS PATHOLOGICAL TENDON LOAD BEARING TISSUE?

The absence of aligned type I collagen in the pathological region of a tendon limits its capacity to transmit load. Therefore, the remaining normal tendon tissue becomes the primary transmitter of load, leaving this load bearing region of the tendon more sensitive to excessive load, and more susceptible to a reactive type response (reactive on degenerative pathology) (Figure 6) (15).

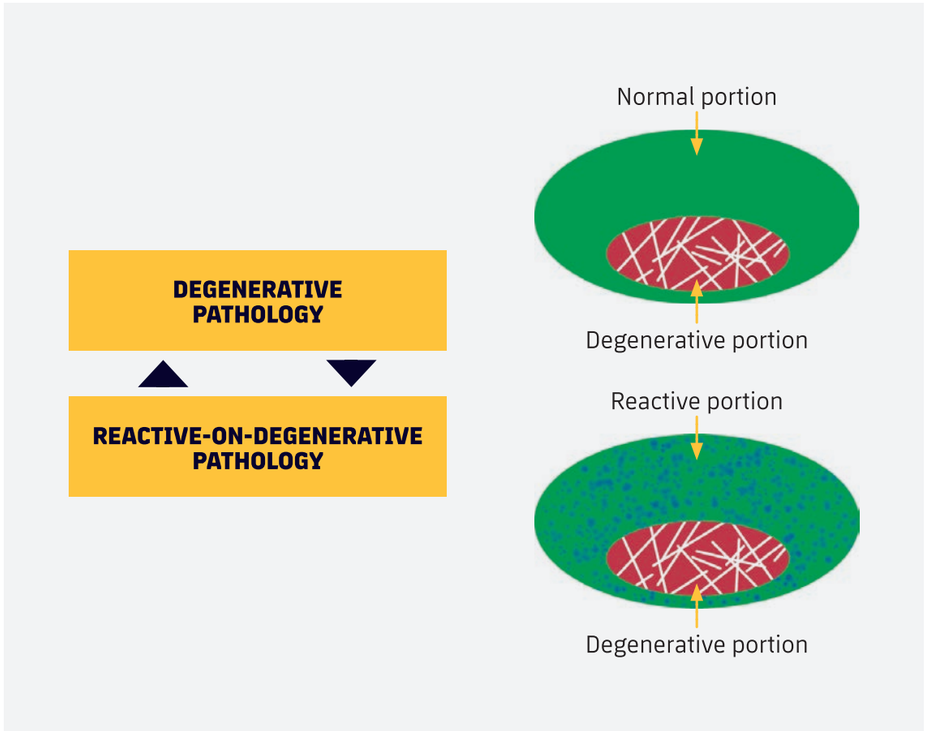


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Figure 6. Reactive on degenerative tendon pathology

CHANGES IN LOAD

Tendons are sensitve to change in load, especially rapid changes to load. Tendon pain may develop when an athlete changes their load suddenly, because the capacity of the tendon (stiffness), muscle (strength and endurance), kinetic chain (strength, coordination) and brain (motor drive) is exceeded (16). Load management therefore becomes a primary prevention strategy; managing the frequency, intensity and volume of loading to ensure there are no fast changes to load can help prevent tendon pain (17). Load modification is also the first step in rehabilitation and managing the athlete in season as reducing load will improve tendon pain.

Tendon overload frequently occurs when an athlete returns to full training after a period of unloading such as time off for an injury or the off-season (representing a big change or relative change in loads) (18). A gradual increase in tendon load over several weeks or months is critical. This requires excellent load monitoring by the performance and medical staff, as well as coach commitment to ensuring a reasoned program. Ideally athletes should retain some loading during the off-season (including strength work and high tendon load) to minimise the change in load on return, especially those with a history of tendinopathy.

Summary:

- Load is essential in maintaining the health of tendons but has also been implicated in the development in pathology. It is important to understand both the types and magnitudes of tendon loads.
- The three main types of tendon load are tensile load, compressive load and friction load. A combination of these loads is often the most provocative and are common in sport.
- The highest tensile load is using the tendon like a spring to store and release energy that requires speed of movement.
- Compressive load occurs when the tendon abuts against another structure, most commonly bone.
- Friction load occurs between the tendon and the surrounding structures and is increased when a tendon repeatedly moves through a large amplitude of movement.
- Rapid changes in load are problematic for the tendon. Tendon pathology and pain may develop when loads change suddenly and exceed the capacity of the tendon.

Clinical Implications:

- Understanding tendon load helps the clinician appreciate if excess load is driving the clinical presentation. It allows the clinician to recognise and remove the provocative loads, and to establish a start and end point of loading.
- Significant over- or under-loading of the tendon may lead to the development of pathology and/or pain.
- Energy storage and release load is high tendon load and requires modification during rehabilitation.
- Reducing compressive load can reduce tendon pain. Exercises that use the muscle-tendon unit in inner range will reduce compression.
- Stretching should be avoided as it increases compression.
- Excessive friction loads can result in irritation of the peritendon structures, which requires different management strategies compared to tendinopathy.
- Changes in load are most commonly implicated in the development of tendon pain. Minimising rapid changes in load through strategies such as effective load monitoring, graduated return to training or play and maintaining some tendon loading during the off-season may reduce the prevalence of tendinopathy.
- In elite athletes, subtle change in the environment, such as a change in shoes or training surface can provoke tendinopathy.

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1.4 RISK FACTORS FOR TENDON INJURY IN FOOTBALL

This chapter outlines intrinsic (player-related) and extrinsic (environment-related) risk factors for lower limb tendon injury in football players, focusing on the two most commonly affected tendons; the Achilles and patellar tendons (Table 1). Most tendon injuries (ruptures or tendinopathy) can be divided into injuries with acute or gradual onset, where the latter ones dominate the injury panorama in football. There is, however, a paucity of high-quality prospective studies evaluating risk factors for Achilles and patellar tendinopathy (1, 2), and still fewer studies focus on football players specifically. The literature on risk factors for acute tendon ruptures are even scarcer, but ruptures generally occur in tendons with pathology (3-5), and the risk factors are likely identical. Most studies only investigate men and the presented risk factors refer to men unless sex is specified.

INTRINSIC FACTORS

PREVIOUS INJURY

Previous injury is the most consistent risk factor for tendon injury in football. Overall recurrence rates for tendinopathy in elite and professional players range from 20-30%, with even higher recurrence rates found in amateur players (44%), likely confounded by inadequate rehabilitation (7). High early recurrence rates are seen for both patellar (20%) (8), and Achilles tendinopathy (27%) (9). For Achilles tendinopathy, a higher recurrence frequency was found after short (0-10 days) compared with longer (>10 days) recovery periods (31% vs 13%), indicating that early return to play may predispose to recurrence of tendinopathy and require further periods away from training and matches (9).

STRENGTH AND BIOMECHANICAL FACTORS

Prospective evidence is scarce, but it is possible that reduced strength (e.g. due to another injury or period of unloading) is a risk factor for development of lower limb tendinopathy (2, 6). While many studies of those with tendon pain show decreased strength, pain can also cause unloading and loss of strength (10), and it is thus difficult to establish a cause and effect relationship. A recent systematic review identified only one prospective study showing an association between increased isokinetic plantar flexor strength at low velocity and a decreased risk for Achilles tendinopathy (2). There is conflicting evidence regarding an association between other biomechanical factors of the foot (e.g. foot pronation, hindfoot inversion/eversion, foot arch) or ankle (dorsiflexion range of motion) and Achilles tendinopathy (2), however this may still be relevant to consider. For patellar tendinopathy there is limited evidence for an association between ankle dorsiflexion range of motion, and anterior and posterior thigh muscle flexibility and injury risk, while neither knee extensor nor flexor strength demonstrated an association (1).

ANTHROPOMETRICS, AGE AND SEX

There is limited evidence that being over-weight, particularly abdominal fat, is associated with increased risk of Achilles tendinopathy (2), but there seems to be no association between weight and patellar tendinopathy (1). In the relatively homogenous population of elite football players, anthropometrical variables tend to have little or no association with development of tendinopathy (8, 9). Players who develop Achilles tendinopathy are typically older than uninjured players (9), whereas the influence of age is less clear in patellar tendinopathy (8).

Tendon injuries are more prevalent in male players, with an almost 5-fold higher incidence reported in the men's top league in Sweden than in the women's league, 0.85 vs 0.18 tendon injuries per 1000 hours (11). Similarly, Achilles and patellar tendon injuries were twice as common in male than in female elite players in a cohort of Northern European elite teams (12).

BIOMEDICAL FACTORS AND GENETICS

Increased body mass index and adverse lipid profile are associated with tendinopathy in the general population (13). In the homogeneous elite football population, however, no association between body mass index and tendon pathology has been found (8, 9). There is a known association of tendon injury, particularly enthesopathy, with rheumatological conditions (psoriasis, rheumatoid arthritis etc.) (3), consequently, increased vigilance may be required for individuals known to have inflammatory disorders or if these conditions exist in first order relatives.

Genetic markers and their contribution to the risk profile of tendon injury has gained increasing attention in the last decade. A recent systematic review identified several genes associated with Achilles tendon injury risk, where particularly tenascin-C and COL5A1 gene polymorphisms may be of importance in genetic predisposition (13). An association between single-nucleotide polymorphisms (SNPs) and tendinopathy risk has also been shown in some studies in elite athletes (14). No genetic predisposition was found in athletes with patellar tendinopathy (15).

There is some evidence that ethnicity may also have an impact upon risk of tendon rupture (16). Research in a military population found that African-Americans had a higher predisposition to major lower extremity tendon rupture in comparison to their Caucasian counterparts (16).

TENDON ABNORMALITIES

Ultrasound-detected abnormalities in professional players at the start of the season are associated with increased risk of developing tendon pain in-season (17). In that study, players with Achilles or patellar tendon abnormalities had 17% and 45% risk of developing symptomatic tendinopathy, respectively, compared with 3% in players with no abnormalities (17). Similarly, a 7-fold increased rate of Achilles tendinopathy and 4-fold increased rate of patellar tendinopathy is seen with tendon abnormalities in physically active people in general (18). Degenerative pathology is always seen in those who sustain a tendon rupture suggesting such pathology is a risk factor for rupture (3-5).

OTHER FACTORS

Leg dominance was not identified as a risk factor for patellar tendinopathy in professional football players, with 40% affecting the dominant leg, 48% the non-dominant leg, and in 3% of cases both legs were affected (8).

EXTRINSIC FACTORS

CLIMATE AND SURFACE CONDITIONS

Climate and surface conditions may interact with tendon injury risk. In the UEFA professional football cohort it was observed that teams from northern Europe (Marine west coast climate) had a 78% increased incidence of Achilles tendinopathy compared with teams from southern Europe (Mediterranean climate) (19). Climate-related differences in ground hardness or grass type may play a role, where harder pitches result in higher ground reaction forces and tendon load. In contrast, no difference was observed in the rate of patellar tendinopathy between geographical regions.

No difference in prevalence of time-loss from patellar or Achilles tendinopathy has been observed between elite football teams playing on artificial turf compared with playing on natural grass (8, 20). The playing surface per se may not relate to tendon injury risk, but a transition between surfaces may be important. However, in the only prospective study in football to evaluate this, no association between frequency of change between natural and artificial grass and overuse injury risk was observed for teams playing in the Swedish and Norwegian top leagues (20).

TIME OF SEASON

A higher incidence of both Achilles and patellar tendinopathy has been observed during pre-season versus the competitive season in professional football (8, 9). There could be several explanations for this, such as pitch and weather conditions during pre-season, a greater tendency to allow players with tendon pain to rest during this non-competitive period, and importantly, a higher intensity and volume of training during this period.

TENDON LOAD AND GENERAL WORKLOAD

The importance of workload in team ball sports, and particularly spikes in load, and association with risk of soft-tissue injuries has been highlighted in several studies (21, 22). In the UEFA elite football cohort, a relationship between the acute:chronic workload (measured with session-rate of percieved exertion, i.e. exposure minutes multiplied by rate of perceived exertion) and non-contact injury was seen, even though the predictive ability was low (23). For tendon disorders, an association between higher total exposure to football (training and match hours) and patellar tendinopathy was reported in elite players (8). Various risk factors in relation to workload and changes in workload may mediate the association between other potential risk factors and injury (24). Other risk factors (e.g. genetics) can also moderate the relationship between workload and injury (22).

MEDICATION

Whilst evidence from football is lacking, in the general population the use of fluoroquinolones antibiotics increase the risk of Achilles tendon rupture 2.5-fold, Achilles tendinopathy 4-fold, and any tendon manifestation 2-fold (25). Direct injection of steroids and administration of systemic corticosteroids are also associated with an increased risk of Achilles and patellar tendon rupture (3). Combined use of fluoroquinolones and corticosteroids increased the risk of Achilles tendon rupture 5-fold and any tendon disorder 15-fold, strongly suggesting that these two drugs should not be combined (25).

RISK FACTOR	ACHILLES TENDON	PATELLAR TENDON
Intrinsic (player-related)		
Any history of tendinopathy	√	√
Strength and biomechanical factors	√	√
Anthropometrics	-	-
Age	√	-
Leg dominance	-	-
Sex	√	√
Biomedical factors and genetics	√	√
Tendon abnormalities	√	√
Extrinsic (environment- related)		
Climate and surface conditions	√	-
Playing surface	-	-
Time of season	√	√
Tendon load and general workload	√	√
Tendon load and general workload	√	√

√ indicates evidence for an association between risk factor and tendon injury

- indicates the evidence is lacking

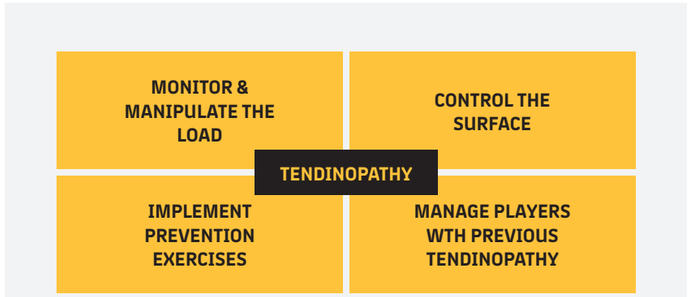
^ Table 1. Potential risk factors for development of tendon injury in football players.

Barça Way

Identifying risk factors for tendinopathy is a key component of the injury risk mitigation strategy at FC Barcelona. Our approach is based on a 4-point injury risk minimisation strategy. (Figure 1)

- **Monitor & manipulate:** Use external training load monitoring (GPS) to measure activity levels and internal measures such as session RPE and athlete monitoring about pain, discomfort etc to measure response to load. Our target is to monitor the amount and intensity of match load and training load (external load) and how they experience this load internally (i.e. internal load) and from this assess how the athletes are coping.
- **Control the surface:** We aim for our teams training and matches to be performed as much as possible on the same surface (natural or artificial grass), and reduce the shifting between surface to the minimum.
- **Be attentive to previous tendon injury:** Players with previous tendon injuries will have their program modified to protect them from new episodes of tendon pain. (2, 6)
- **Implement preventive exercises:** Injury prevention programs are essential for every player, general prevention program is instituted for the most common injuries in football.

We recently published a study in our club (with players from the professional men's football, basketball , handball, futsal and roll hockey teams) with the aim to investigate the association between risk of tendinopathy and genetic markers in elite team sports (26). Thanks to this preliminary study we are analyzing approximately 100 polymorphism of our players that allow us to get an idea of the susceptibility of incurring a tendinopathy over the season. Future research should aim to validate our results in other cohorts and elucidate whether these or other variants might be involved in the risk of a highly feared tendinopathy in elite athletes, and in tendon rupture (27).



^ **Figure 1.** Barça Way of minimising exposure to tendinopathy risk factors.

Summary:

- Intrinsic risk factors for the development of tendon pathology include previous injury, strength and biomechanical factors, age, anthropometrics, sex, biomedical factors, genetics and tendon abnormalities.
- Extrinsic risk factors include climate and surface conditions, time of season, tendon load and general workload, and some medications.
- Recurrence rates of tendon pain in football players are high, which may indicate inadequate rehabilitation.
- Reduced strength has been correlated with the development of tendon pathology, however, strength can be impacted by pain and subsequent unloading.

Clinical Implications:

- Intrinsic risk factors may be difficult or impossible to modify, however, individual risk factors may be taken into account when designing rehabilitation or training programs. For example, a player with a history of tendon injury may require increased load monitoring and a more gradual progression of training loads during rehabilitation.
- Recognise extrinsic risk factors for tendon pathology and pain, as this may allow risk reduction. For example, encouraging players to maintain some tendon loading throughout the off-season may reduce incidence of tendon pathology in-season, or controlling for playing surface as much as practicable may assist in mitigating risk.

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General section Part 2

— Lorenzo Masci, Henning Langberg, Seth O'Neill and Ebonie Rio

2.1. PRINCIPLES OF ASSESSING TENDINOPATHY

Initial presentation of tendon pain in an athlete requires a comprehensive and methodical approach to confirm the diagnosis of tendinopathy. The clinician must assess potential musculoskeletal, psychological and systemic factors contributing to tendon pain. This initial assessment requires a multi-disciplinary approach involving the athlete, medical and health practitioners and coaching staff.

HISTORY

A thorough and detailed history from the athlete is a critical component of the initial assessment. Tendon pain has specific qualities that differentiate it from other pathologies (Table 1). Tendon pain is localised and does not spread with load. It usually has a gradual onset and presents after an increase in high tendon load activities. Sudden onset pain during athletic activity may be consistent with partial (rare) or complete tendon tear. Tendinopathy often has a hallmark sign that assists in the diagnosis.

Achilles	Morning pain and stiffness that warms up within 30-minutes
Patellar	Sit pain especially in the car
Hamstring	Sit pain especially on a hard chair
Adductor	Pain turning in bed, getting in/out of car

^ **Table 1.** Hallmark signs of tendinopathy.

Tendinopathy is load-related and dose-dependent, usually worsening after energy storage and release loading e.g. running for Achilles tendon or jumping for patellar tendon (Table 2). Pain usually has a warm-up element (decreasing with activity) but pain can then increase following training. Pain when loading is evident the next day. Importantly, pain from tendinopathy decreases when load is reduced, either immediately or progressively over weeks (1). The pain should increase as the spring like loads on the tendon are increased, for example, sprinting should be more painful than jogging for an Achilles, and jumping should be more painful than jogging for a patellar tendon. The athlete may report functional impairments because of pain, resulting in poorer performance.

Assessment of irritability is also important. Irritability is defined as the tendon pain response after activity. Studies have suggested that pain provocation lasting less than 24 hours may be acceptable during rehabilitation (2). More irritable tendon pain is thought to last greater than 24 hours or is produced with minimal tendon loading.

TENDINOPATHY	TYPICAL FEATURES OF PAIN
ACHILLES	Localised mid-portion or insertional pain during loading Aggravated by running and plyometric activities Localised morning stiffness and pain Worse after after a period of inactivity (sitting or morning pain) Warms up with activity only to be worse the next day
PATELLAR	Localised pain distal to inferior pole of patella during loading Aggravated by jumping, change of direction and prolonged sitting, especially in a car
HAMSTRING	Localised pain at ischial tuberosity Aggravated by sitting, driving and walking/running uphill
ADDUCTOR	Localised adductor pain Aggravated by change of direction activity or cornering (inner leg)

^ Table 2 . Typical features of tendon pain.

An atypical athlete history includes sudden onset pain, pain related to low tendon loads (such as static loads, weights or cycling), pain at rest or at night and pain that spreads. Presence of atypical features requires consideration of other possible tendon (such as peritendon, Achilles pain due to plantaris compression) and non-tendon causes including inflammatory arthropathy or joint pain (posterior ankle, patellofemoral joint, hip joint).

The history should also include questions about known risk factors such as previous injury history especially to the ankle, calf or knee, individual and family history of systemic disease, previous episodes of tendinopathy and management, current management program, use of orthotics, and effect of shoes on pain.

Questions on current management should investigate strategies used to ease pain, both active approaches such as load modification, and passive interventions such as ice and massage. It is also critical to assess the type and number of direct tendon interventions (injections) and their effect. These may help guide differential diagnosis.

LOAD CHANGES

Changes to training loads are an important factor in the development of tendon pain in athletes. These changes may not just be total weekly volume but could also result from changes in frequency (i.e. two sessions on consecutive days) or increased intensity due to drills requiring repeated energy storage and release loading. An increase in training volume has been associated with Achilles tendinopathy in endurance runners (3), less than 2 days rest per week was associated with overuse injury. More frequent loading of greater than 3 sessions per week doubled the prevalence of patellar tendinopathy in elite volleyball players (4). Changes to training surfaces

or shoes could also be provocative (5). It is also important to enquire about sudden reductions in training load due to other musculoskeletal injuries, illness or work and life issues. A sudden reduction in training load may make the tendon less load-tolerant and more susceptible to pain once normal loads are reintroduced.

EXAMINATION

An initial examination aims to confirm the clinical diagnosis of tendinopathy and exclude other possible differential diagnoses. Reproduction of localised tendon pain with tendon loading manoeuvres and, in particular, energy storage and release loading is an important finding (1, 5) and is used to confirm tendinopathy and assess the degree of tendon irritability.

A thorough examination should be undertaken to identify deficits in the kinetic chain including the hip, knee, ankle, and trunk. Pain and deficits in function may impact ability to complete energy storage and release manoeuvres, such as hopping in Achilles tendinopathy and take off in a jump in patellar tendinopathy. There should be an increase in localised pain as tendon load increases, eg single leg hopping should provoke more Achilles pain than double leg jumping (Table 3). In higher functioning athletes an assessment of muscle and kinetic chain endurance and kinetic chain function when fatigued can also be helpful. The athlete with tendinopathy should report localised pain that remains localised with load, diffuse pain suggests another diagnosis.

ACHILLES	PATELLAR	HAMSTRING	ADDUCTOR
Slow double leg heel raises	Double leg squats (decline board)	Double leg slow bending forward	Adductor squeeze with bent knees
Slow single leg heel raises	Single leg squats (decline board)	Single leg slow bending forward	Standing adduction against resistance (elastic/cable)
Continuous double leg jumps	Double leg jumps	Double leg fast bend forward	Copenhagen adduction exercise
Continuous single leg hops	Single leg jumps	Single leg fast bend forward	Change of direction
Forward hops	Stop jump	Fast single leg change of direction with hip flexion	Kicking

Increasing load ↓

^ Table 3 . Proressive loading test to assess tendinopathy.

Examining movements that provoke compression is critical if compression is considered to be an important load. These movements include loading in dorsiflexion for Achilles insertion or plantaris, and deep body on leg flexion in the hamstring. The role of compression in adductor tendons is debated, but weight bearing hip abduction may induce compression. There are no compressive forces on the patellar tendon.

Muscle strength testing of the affected muscle tendon unit should be undertaken to assess the force generating capacity of the muscle in isolation. There is often loss of muscle bulk in the affected muscle tendon unit.

An examination assessing dorsiflexion range of movement and foot posture (supinated foot posture)(6, 7) should be undertaken to detect significant deficits, particularly those that are modifiable, however there is conflicting evidence of the importance of abnormal biomechanics (8). Joint range of movement, muscle length and neural length are usually not affected.

Palpation tenderness may be misleading and the clinical utility of palpation tenderness is conflicting across different tendons (9-12). While normal tendons can be tender to palpate, an absence of palpation tenderness is more useful as it may indicate a non-tendon diagnosis.

In tendons with substantial peritendon pathology, pain is aggravated by repeated movements causing friction in the peritendon. If low load repeated movements are provocative and pain does not change with increased tendon load then consider a peritendon diagnosis.

It is also important to examine other structures that could be contributing to the pain. For example, performing a posterior impingement test to exclude posterior ankle pathology in Achilles region pain or a slump-test and straight leg raise to exclude referred lumbar spine pain in proximal hamstring pain. There can be more than one source of pain, there can be a component of tendon pain as well as pain from nearby structures. Excellent clinical skills are required to distill the key structures causing pain (5). When athletes report their pain is provoked with low tendon loads ie cycling for the Achilles or patellar tendon, differential diagnosis is critical. Similarly, if the athlete's pain is not provoked with the high tendon loads then differential diagnosis is required.

OUTCOME MEASURES

SUBJECTIVE MEASURES

Tendon specific patient-reported outcome measures include the VISA scales (Achilles, patellar, hamstring and gluteal tendons) (13-16). These scales are scored out of 100 points and encompass measures of pain and function, and are validated measures that can assess severity and monitor outcome (17, 18). A score of less than 80 point is often used as to indicate tendinopathy and changes in score after intervention of 10 or more points can designate symptom improvement. For VISA-P, the minimum clinically important difference is a change of 13 points (19). These outcomes measures should be used to assess rehabilitation progression but may prove less sensitive at detecting short term and subtle improvements in function, particularly at the elite level (5). Short term assessment is important to quantify the change in symptoms, or stability in symptoms with rehabilitation. This is best completed with a 24 hour response to activity test (Table 4), where pain on the test is recorded at a similar time each day to assess the effect of activity on the tendon from the preceding day.

TENDON	24 HOUR RESPONSE TEST	24 HOUR RESPONSE
ACHILLES	Single leg hop	Morning pain and stiffness
PATELLAR	Decline squat	Sit pain in car
ADDUCTOR	Squeeze	
HAMSTRING	Arabesque	Sit pain in hard chair

^ **Table 4 .** 24 hour response to activity test.

Other measures/questionnaires that can be used are the modified Tampa scale, and some assessment of quality of life, as these measures are more important in people with longer term pain.

OBJECTIVE MEASURES

Assessment of the capacity of the muscle tendon unit is important to inform rehabilitative targets for sports performance and to guide clinical intervention. Measurements need to be valid, reliable, time-efficient and suitable for both competing athletes and those in rehabilitation. Measures of muscle strength, endurance and power are one indication of the capacity in a competing athlete to maintain training and to guide the return to play decision making process. Recommendations for strength would include calf testing, quadriceps testing using a seated knee extension unit, hamstring testing using Nordics and hip adductor testing using a groin squeeze. Endurance can be assessed with repeated activities such as single leg calf raise to fatigue. Power can be assessed with hopping, sprinting and change of direction exercises.

MEASURING STRENGTH, POWER AND STRENGTH ENDURANCE DURING REHABILITATION

Objective tests can indicate the starting place for rehabilitation but also monitor in-season capacity. Isokinetic dynamometer testing is more informative than clinical measures but often unavailable. Alternative performance measures like force plates and sprint times can be utilised, but these are often too provocative during the early stages of rehabilitation. Rehabilitation targets need to consider normative values rather than using the un-injured limb as data currently suggests this can also be affected (20). Measures that are easier for clinicians to complete include video and joint angle excursion measurements, contact mat measure of jumping, and measures of strength endurance (number of heel raises). Data from an athlete's gym program can also be used to assess strength improvements and between limb differences.

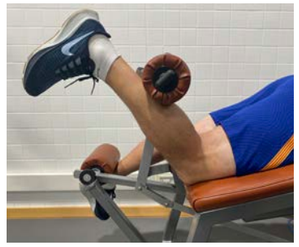
CALF

The calf may benefit from endurance testing using isokinetic dynameter. Simple heel raises until fatigue have not been shown to differ between injured limbs (possibly due to bilateral cortical changes, unloading), and it is unclear whether there is a difference between healthy and injured people. Although this test has shown good reliability and has been recommended as the main impairment measurement for patients with Achillles tendinopathy (18), repeated heel rise is not related to ankle plantarflexion maximum voluntary contraction and may not represent a valid approach for strength testing of this muscle group (21). Calf raise endurance differs to strength so the clinician should consider what they are measuring and what the athlete needs for their sport.

^ **Figure 1.** Single leg standing calf raise.

The single leg calf raise strength/endurance test would not be recommended for use as a sole guide for return to competition. Plantarflexion weakness was associated with Achilles tendinopathy in cross-sectional studies (3, 22), and one study suggested plantarflexion weakness had an 85% sensitivity for predicting Achilles tendinopathy (23). Isokinetic strength testing is recognised as the gold-standard in measuring plantarflexion strength and has shown good reliability (24). However, other measurements of assessing muscle strength and endurance that are less complex and time consuming could be implemented. Clinically, depending on access to equipment a clinician may use a combination of calf raise endurance (single leg) (Figure 1), calf strength in standing (kilograms on the bar on a resistance machine) as well as single leg seated calf raise weight in the gym. These will provide different information that can be integrated into decision making for return to play. The strength/endurance target should be based on sport requirements and not the asymptomatic leg.

Calf, Achilles and kinetic chain power can be measured with hop height and /or endurance (maintenance of height over set repetitions) using a contact mat or other measurement software. This best reflects the capacity of the calf and tendon complex to mimic return to sport loads.


^ Figure 2. Tests of hamstring strength.

HAMSTRING

There is an association between reduced hamstring and gluteus maximus strength and proximal hamstring tendinopathy. In a football population, this is commonly detected using dynamometry assessment. In chronic cases of proximal hamstring tendinopathy, knee flexor and hip extensor muscle weakness is a common clinical finding (25, 26). Weakness of the trunk and gluteus medius have also been reported (26, 27). It is unclear as to whether these deficits are pre-existing, or are a consequence of the tendinopathy.

Clinically, the combination of weights (single leg) in prone hamstring curl, bridging and Nordics may assist with determining hamstring strength (Figure 2). Gluteal strength is difficult to isolate and measure.

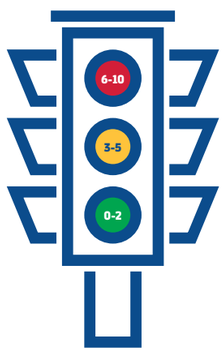
QUADRICEPS

Strength can be measured on leg extension machine, either 1RM or up to 5RM (Figure 3). Similarly leg press can estimate overall kinetic chain capacity, setting a target strength is difficult as machine levers and resistance varies. It is unknown if using the other leg as a comparison is a reasonable target. As with the Achilles, jump height and jump height endurance will place high loads on the muscle tendon unit, reflecting the loads likely to be encountered during play.

^ Figure 3 . Single leg knee extension.

ADDUCTOR TENDONS

The adductors can be tested as a muscle group and strength can be measured reliably with a hand-held dynamometer (28). Adductor squeeze tests can be used as a general measure of isometric adduction strength for assessment and to guide rehabilitation progression. Pain during testing should be considered when evaluating results (Figure 4). Adduction squeeze values can be normalized to bodyweight and compared to normative data in healthy football players (29). In athletes with unilateral pain, measuring each side separately will provide a symmetry index. Isometric testing will be able to provide both peak force and rate of force development (29), however, eccentric testing has shown to have a higher association with pain, and is preferred when possible (30). Both test types can be used for a comparison with abduction strength. In football players, the adductors are normally stronger than the abductors (30, 31). Strength endurance are usually best measured using exercises, for instance through assessment of the maximal number of repetitions performed in a seated adduction machine or with the Copenhagen adduction exercise for stronger athletes (32).



^ Figure 4 . 5 second adductor squeeze test.

IMAGING

There are substantial limitations of tendon imaging in the diagnosis and management of tendinopathy, and initial imaging with ultrasound or MRI should be utilised on a case by case basis. Firstly, abnormality on tendon imaging does not confirm tendon pain, as pathology observed on imaging is present in asymptomatic athletes (33). Importantly, normal tendon imaging suggests a possible non-tendon cause of pain. Secondly, serial imaging is not recommended as symptoms often improve without corresponding changes in pathology on imaging (34). Finally, baseline imaging cannot determine the outcome for the athlete with tendinopathy (28).

OTHER MEASURES

Blood tests to exclude inflammatory or auto-immune disease may be considered in athletes with atypical pain or features suggestive of inflammatory disease.

TENDON RUPTURE

Tendon rupture is a complete disruption of the tendon and occurs most commonly on a background of substantial tendon pathology in those with no symptoms, i.e. 'spontaneous' tendon rupture (35). The most common tendon rupture is the Achilles tendon and can be clinically diagnosed with the Simmonds-Thompson squeeze test or visualised on diagnostic imaging. The patellar and adductor tendons rarely rupture. The hamstring tendon has a greater tendency for bony avulsion in the skeletally immature (36, 37) and extreme load tendon ruptures beyond the mid 20s (38). Tendon rupture can be managed conservatively or surgically, the latter being more common in elite sport. Clinicians should remember that the contralateral side may not be normal in structure (39), and is at low risk of rupture, and loading on that limb should be maintained as much as possible during rehabilitation (40).

DIFFERENTIAL DIAGNOSIS

Differential diagnosis is a critical part of the examination. Many athletes are diagnosed with tendinopathy based on abnormal imaging and palpation soreness when neither of these are solely diagnostic of tendon pain and the source of pain may be another structure (Table 5). Sound clinical examination skills will support a tendinopathy diagnosis.

Tendon pain generally exhibits a warm-up pattern, where pain decreases as the activity continues (41). If the athlete complains of increasing pain with activity or pain aggravated by activities of low tendon load (slow or static tasks), irritation of the peritendon must be considered. If pain is present with little or no loading (night pain, pain resting in the evening) other sources of pain should be considered. Alternative diagnoses should be considered in athletes who complain of increasing tendon pain the longer they train or play, or pain that doesn't start until a period of time into the session.

In insertional tendinopathies the bursa is an integral part of the tendon attachment. Tendinopathy as a result of excess compressive loads on the tendon enthesis affects all the enthesis structures and bursitis cannot be a diagnosis in isolation. That is, trochanteric bursitis and retrocalcaneal bursitis are in fact tendinopathies, not isolated bursal pathology. An exception is the superficial calcaneal bursa that is not part of the muscle tendon unit (42). Irritation of this structure relates to compression or friction from footware and an intervention directed at loading without addressing provocative footware will have little or no effect.

Pain can also come from surrounding structures such as joints, nerves and less commonly implicated tendons. Inflammatory conditions such as psoriatic arthritis and other pain conditions must also be considered, these conditions are uncommon in athletes.

PAIN IN THE REGION OF THE TENDON	COMMON PRESENTATIONS	DIFFERENTIAL DIAGNOSES
ACHILLES	Mid-portion tendinopathy Insertional tendinopathy Peritendinopathy	Plantaris tendinopathy Superficial bursa Neural irritation Posterior ankle joint Medial and lateral tendons
PATELLAR	Proximal patellar tendinopathy Distal patellar tendinopathy	Patellofemoral joint pain Bursitis (infrapatellar, prepatellar)
HAMSTRING	Hamstring tendinopathy	Sciatic nerve irritation Hip joint
ADDUCTOR	Adductor tendinopathy	Symphysis pubis irritation Pubic bone pain Iliopsoas tendinopathy

^ Table 5 . Differential diagnoses.

CLINICAL EXAMPLES OF DIFFERENTIAL DIAGNOSIS

Achilles: Peritendinopathy is a common differential diagnosis for Achilles tendon pain and for foot and ankle tendons (Figure 5). Peritendinopathy is provoked with friction loads when the tendon is required to move through a large range of motion with low loads. For example, if a player lacks calf strength or endurance, as they run to fatigue they can may utilise a larger range of motion at the ankle joint, which may irritate the peritendon. Even though they are running (a spring-like high tensile load for the Achilles) clinicians must critically analyse pain behaviour to correctly diagnose the primary issue. Peritendinopathy requires a different initial management, firstly removing excess movement of the tendon through large ranges of plantar and dorsiflexion and then rehabilitating the underlying dysfunction (such as poor calf endurance). This presentation is common in foot and ankle tendons such as tibialis posterior.

On assessment athletes with peritendinopathy may complain of more difuse pain that those with tendinopathy, they may be more provoked with calf raises in assessment than hops. Audible crepitus is often absent but a stethoscope can be helpful to hear the crepitus as they actively plantar and dorsiflex the ankle.

Patellar: Patellar tendinopathy is mainly found in young jumping men and occasionally elite female jumping athletes, anterior knee pain in most other cases is patellofemoral in nature (Figure 7). Provocative activies will vary, with patellofemoral joint pain provoked with lower load activities such as walking and cycling. Patellofemoral pain can occur over the patellar tendon but is not localised pain and can diffuse more with load.

On asssement those with tendon pain will have a very stiff legged hop compared to those with other anterior knee pain, and will have high levels of pain on the decline squat early in range. Although patellofemoral pain also is aggravated by the decline squat pain levels are lower and occur deeper in range. Muscle wasting in both the quadriceps and the calf can be more evident in patellar tendinopathy than patellofemoral pain.

Hamstring: Diagnosis of the cause of pain in the proximal hamstring region can be difficult, due to the complex anatomy and proximity of numerous pain generating structures in this area (Figure 6). Potential differential diagnosis may include somatic referral from the lumbar spine, sacroiliac and hip joint, as well as radiculopathy and peripheral nerve entrapments (43). In particular, differentiation between sciatic nerve irritation and proximal hamstring tendinopathy is a significant clinical challenge, as aggravating activities for both of these conditions overlap (sitting, combined hip flexion and knee extension activities), in addition to the potential for these two pathologies to coexist (44). Proximal hamstring tendon pain should remain localised and demonstrate a dose-dependent increase in pain in response to higher tendon load (43). Pain referred from other structures such as the sciatic nerve is often more diffuse, and may be aggravated by neurodynamic testing (44).

Adductor: Numerous musculoskeletal and non-musculoskeletal structures can refer pain to the adductor region, creating a diagnostic challenge for the clinician (Figure 8). The Doha consensus statement was created in an effort to simplify the diagnostic process in this region (45). In accordance with this agreement, adductor related groin pain should elicit recognisable pain on palpation of the adductor longus insertion and pubic bone, as well as during adduction resistance testing (45). Other regions which can refer pain to this region include the hip joint and lumbar spine. Hip related pain is often described as being deeper compared with adductor related pain, and is aggravated by positions of hip flexion, such as prolonged sitting (46). A negative flexion, adduction, internal rotation test is useful in ruling out the hip joint as a source of groin pain in athletes (47).

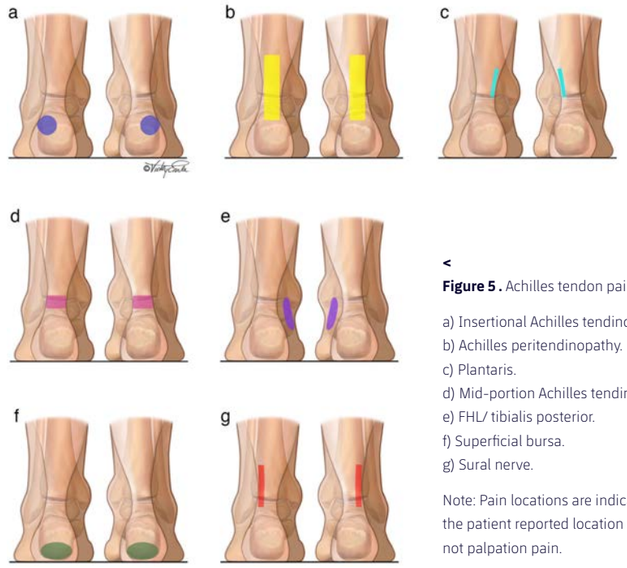


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Figure 5 . Achilles tendon pain map.

a) Insertional Achilles tendinopathy.
b) Achilles peritendinopathy.
c) Plantaris.
d) Mid-portion Achilles tendinopathy.
e) FHL/ tibialis posterior.
f) Superficial bursa.
g) Sural nerve.

Note: Pain locations are indicative of the patient reported location of pain, not palpation pain.

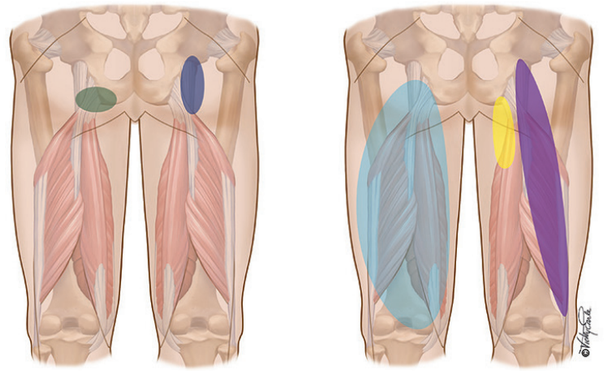

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Figure 6 . Hamstring pain map. Localised hamstring tendon pain (green circle) indicates tendon pain, more diffuse pain indicates other sources".Note: Pain locations are indicative of the patient reported location of pain, not palpation pain .

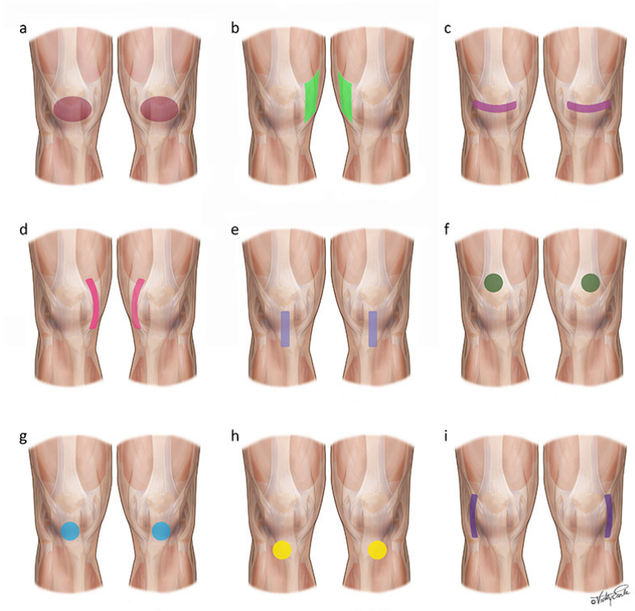

Figure 7 . Patellar pain map. Localised pain indicates tendon, diffuse pain indicates patello-femoral joint or other source of pain.

a) Patellofemoral joint.
b) Patellofemoral joint.
c) Patellofemoral joint.
d) Patellofemoral joint.
e) Patellofemoral joint.
f) Quadriceps tendon.
g) Patelllar tendon.
h) Patellar tendon.
i) Patellofemoral joint.

Note: Pain locations are indicative of the patient reported location of pain, not palpation pain.

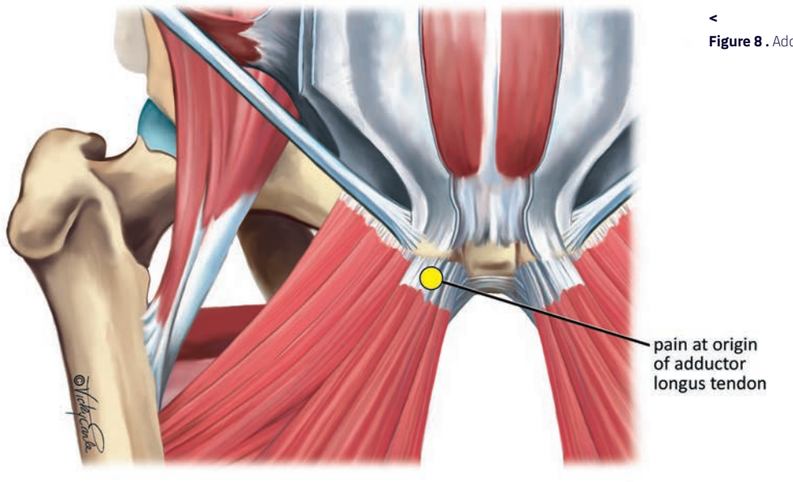

Figure 8 . Adductor pain map.

Summary:

- Tendon pain has hallmark signs that differentiate it from other conditions. Tendon pain remains localised, is latent or of gradual onset, is load-related and dose-dependent, aggravated by tensile and compressive loads, often exhibits a warm-up pattern and decreases when load is reduced. Presence of atypical features may suggest a differential diagnosis.
- Alterations in training loads are likely to be an important factor in the development of tendon pain. This may include changes in training volume, intensity, frequency, or surface.
- Palpation tenderness may be misleading, as normal tendons can also be tender to palpate. Tendons can be painful on palpation in other musculoskeletal conditions such as osteoarthritis. However, the absence of palpation tenderness may indicate an alternative diagnosis.
- Localised tendon pain should increase in a dose-dependent manner with progressive loading of the tendon.
- Clinicians should consider a differential diagnosis if the athlete's pain is aggravated by activities of low tendon load.

Clinical Implications:

- The primary aim of the examination is to confirm the diagnosis of tendinopathy and to exclude other sources of pain.
- Palpation soreness is not diagnostic.
- Isolated muscle testing to assess the strength of the affected tendon should be completed so that deficits are not hidden within the kinetic chain.
- Assessment of strength, endurance and power are essential to understanding the athlete's capacity.
- There are significant limitations associated with the imaging of tendons. Abnormality on imaging does not confirm tendon pain, as pathology on imaging may be present in asymptomatic athletes. Additionally, symptoms often improve without corresponding changes in imaging findings. Furthermore, imaging is not capable of providing prognostic information. Decisions regarding imaging should be made on a case-by-case basis.
- Differential diagnosis is complex and requires good clinical skills.
- Tendon pain often demonstrates a characteristic warm-up pattern, which can make it difficult to determine if the tendon's capacity has been exceeded at the time of loading. It is therefore recommended that 24-hour pain response is carefully monitored, ideally with a standardized loading test at a similar time each day.
- The pain should increase as the spring like loads on the tendon are increased. For example, sprinting should be more painful than jogging for an Achilles and jumping should be more painful than jogging for a patellar tendon.
- In tendons with substantial peritendon pathology, pain is aggravated by repeated movements causing friction in the peritendon. If low load repeated movements are provocative and pain does not change with increased tendon load, then consider a peritendon diagnosis.
- It is important to also be cognisant of recent reductions in training load, as this can lower the tendon's load tolerance and may increase the susceptibility to pain when normal loads are re-introduced.

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2.2 PRINCIPLES OF MANAGING TENDINOPATHY

Initial management of the athlete with tendinopathy needs to consider the tendon involved, the kinetic chain it functions in, the loading history of the tendon, the irritability and severity of pain and the athlete's sport. Based on these factors, the player, clinician and coach must decide if the player can continue to train and play, or if cessation of activity is required in order to rehabilitate the tendon.

Factors that might help make this decision can be performance-based or pain-based. In a football population both factors are important and linked. Tendon pain that impedes an athlete's capacity to sprint and change direction at their full potential affects their performance and may increase their vulnerability to other injuries. The final decision whether to stop competition and training or to manage the athlete in season rests primarily with the athlete and coach. The clinician's role is to highlight deficits in function and capacity and to estimate time to reduce pain to manageable levels and to recover function.

Education of the athlete and coaching team of factors that contibute to tendinopathy development and management is essential, especially a greater understanding of tendon load. This assists in symptom control and helps empower the athlete to better self-manage their injury. A large part of this education should centre around activity modification and pain monitoring, as this offers tools for pain management if the athlete continues to train and play. Essentially, the player needs to ascertain whether the pain is acceptable and reflects the amount of activity/load. If the player decides to stop training and rehabilitate their tendon, this same process is used to continually monitor and progress their rehabilitation.

REHABILITATING AN ATHLETE WITH TENDINOPATHY

The clinician first assesses the current capacity of the athlete and then applies suitable physiological loads to the tendon, the muscle, the kinetic chain and the brain during a progressive loading program. Knowledge of types of tendon load and the importance of rate of loading is required to decide how and when to progress (Figure 2). Exercise prescription may be influenced by symptoms (immediate and one day after exercise), the type and magnitude of load, cumulative load, control of exercise (physiological tremor), aim/target of exercise (force capacity, endurance, higher loading rates), the sport and athlete and their role within that sport.

In the early stages of rehabilitation isolated loading of the musculotendinous unit is crucial to ensure that the affected tendon and muscle is loaded to a sufficiently to stimulate adaptation (greater muscle strength and improved tendon stiffness), and to prevent deficits hiding in the kinetic chain (Figure 1). Therefore, single joint isolated exercises must be completed prior to progression to multi-joint exercises. It is also critical that exercises are performed single leg and each leg is loaded maximally and independently. Improvements in the strength of the affected leg may be also enhanced by strength improvements of the unaffected leg, a phenomenon known as cross education, which is useful in the early stages of rehabilitation (1, 2). For example, when rehabilitating a the patellar tendon injury, an isolated single leg knee extension (figure 1) should be completed maximally on each leg and should be used prior to introducing compound exercises such as leg press, squat or lunge.

^ **Figure 1.** Single leg knee extension.

The target of rehabilitation will gradually shift from strength and endurance to power that will increase the capacity to cope with training loads in preparation for return to play. Once a sufficient strength and endurance base has been established, exercise that slowly increases the rate of loading are introduced. There is currently no definitive readiness test to inform this decision, instead this transition is made largely based upon clinical judgement by ensuring strength and endurance are adequate and then assessing the pain response to exercise with higher loading rates. Once a player can demonstrate tolerance to clinical power-based exercises such as hopping and change of direction the athlete is gradually prepared for a return to training. Measuring plyometric capacity of the limb with contact mats and force plate analysis prior to return to training load may be beneficial.

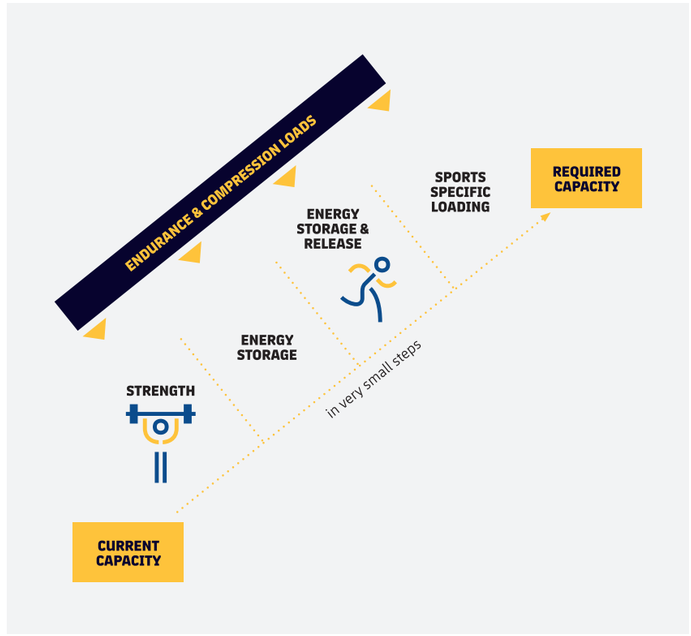

Figure 2. Principals of tendon rehabilitation.
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Return to training rehabilitation must consider the normal loads associated with the training week and match-play. Sports specific drills need to be progressively complex such as varying ball/player interceptions at different speeds on different tangents. Simple progressions could include changing the playing area, playing surface, predictability of play and the complexity of the game (e.g. numbers of players). Time on the ball should be altered to challenge cognitive decision making skills (increased cognitive load). Return to running progression should be based on the athlete's preferred running speed as it indicates the speed that tendon load is optimal for the athlete's current stage of rehabilitation. Running slower or faster than preferred speed appears to increase stress on the musculoskeletal system and may flare tendon pain. Once the athlete is successfully able to run at high speeds, it becomes important to develop more sports-specific capacity by incorporating acceleration/deceleration, change of direction, jumping/landing, and off tangent/cornering drills. These tasks should initially be introduced in a non-fatigued state prior to attempting these tasks when the athlete is more fatigued.

The risk of recurrence is greatest with shorter rehabilitation cycles. This may be due to unforeseen spikes in training load due to the rapid return to play. Data from simple load calculations (distance covered in game/normal training and tendon load) can be used to plan training. If the player normally covers 11.2km per game with an overall tendon load of 1.98million kgs, then rehabilitation needs to ensure cumulative loads achieve this target.

MONITORING REHABILITATION

Symptoms: Symptoms should be monitored in the morning (e.g on first steps in the morning for an Achilles), during a standardised loading test at the same time each day and after training (Table 1). Symptoms should be stable from day to day, with the pain remaining similar or ideally improving compared to the previous day. Worsening pain indicates overload and requires amendment of training to prevent load exceeding tendon capacity. Evening pain, especially in bed, is rare (with the exception of gluteal tendinopathy) and requires careful consideration of differential diagnosis including an inflammatory component or bone stress.

Assessment of pain after activity forms an important part of education for athlete and coach – pain may not always be zero, nor is that a requirement for return to play, pain that is low and stable indicates tolerance to loading. The 24 hour pain response to loading provides feedback on the provocation of the previous day activities, allowing clinician and athlete to amend rehabilitation appropriately. Altering one aspect of rehabilitation at a time during the return to sport phase (such as increasing the number of sprints) is preferable to multiple changes of loading activities. If the pain is worse the day after a single change in training load it becomes clear exactly what exercise was provocative. Importantly, this will also ensure that any flare of pain is both low level and short term. Due to the warm up phenomenon, basing loading decisions on how an athlete feels in a training session is not recommended, as this places the athlete at risk of both overload or underload.

TENDON	LOADING TEST	ADL TEST
Achilles	Single leg hop pain	Morning stiffness (time and/or severity)
Patellar	Single leg decline test	Sit pain
Hamstring	Arabesque	Sit pain
Adductor	Groin squeeze	Pain turning in bed, getting in/out of car

^ Table 1 . Tests for assessing symptoms in each tendon

Psychological readiness to return to play is probably best measured using a battery of tests. Player perceived readiness is an important consideration in the return to play process. The ability to complete all activities without conscious thought, (the absence of fear-avoidance movement or behaviour or catastrophic thought patterns), as well as symptoms remaining stable is an important first step in the process. Once this is established, the athlete must be able to cope with the simulated demands of competition, and is able to successfully perform for the required amount of time. Finally, it is important that the athlete has reached (or is very close to) their normal training workloads, as this is often a good indicator of the ability to withstand competition demands. Consideration must be given to acute/chronic workload ratio, as merely achievieng their normal training loads without the background period of time for adaptation of the musculoskeletal system may expose the athlete to increased risk of re-injury or new injury.

RETURN TO PLAY

Return to play and return to performance are not necessarily the same. An athlete who returns to training or competition may be considered to have 'returned to play', however they may not yet have returned to their usual performance level.

Despite the prevalence of tendon problems in football there are few data on the return to play decision following tendon injuries. The medical and coaching staff face considerable pressure from both managers and the player to return to training/competition as soon as possible. This can result in an underprepared player returning early, which may result in ongoing or recurrent pain, and ongoing functional and performance impairments (3). This may be especially important in older players, for whom re-injury rates have been shown to be as high as 27% following a short rehabilitation period (3). Coach and player education about monitoring symptoms and a graduated return to training loads is essential.

Accurately predicting when an athlete can return to play is ideally a criteria based decision, and requires careful consideration of planned training sessions and team composition leading into competition. There is a lack of valid criteria to definitively guide return to play decision-making.

Factors that influence return to play, aside from meeting rehabilitation goals, include previous injuries to any other structure or previous tendon injury. Although rare in a young athletic population, co-morbidities such as diabetes or underlying rheumatic disorders may also require consideration, as these conditions may interfere with ability to load the tendon to build sufficient capacity to return to play.

A number of wider contextual factors such as the timing within a season, the wishes of the player and external pressure from the coach, media and agent may also impact return to play decision-making. A decision-making process can be adjusted to football (4). This process follows three stages encompassing all relevant biopsychosocial components.

STAGE 1: EVALUATION OF HEALTH STATUS

Patient demographics: Age is an important factor with older players being more likely to be re-injured (5) and possibly requiring more time for rehabilitation, although this may vary depending on the tendon affected.

Symptoms: Continued monitoring of symptoms remains critical, and daily or tri-weekly assesssment of pain is essential especially after a change in training load.

Personal medical history: Previous injury, especially to the affected musculotendinous unit, is an important consideration. Additionally, whether this tendon injury is the first injury or re-occurrence should also be taken into account. Previous research has identified that individuals who sustain tendon ruptures have higher levels of tendon degeneration (6, 7). About 6% of those who have had a previous Achilles tendon rupture will rupture the other side (8). Previous use of systemic or injected corticosteroid may increase risk of rupture although evidence for this is lacking (9).

Functional tests: The functional capacity and degree of irritability of the condition is considered when deciding how much activity the athlete is able to complete. Functional ability via testing of isolated muscle-tendon unit strength through isokinetic dynamometry or similar can be used with specific targets for each muscle group. For the calf more than two times body weight eccentric or isometric load is considered the minimum and more than 2.5 body weight is desirable, with these values being independent of knee position. For the quadriceps the aim is 3x body weight on the leg press (10).

A variety of other load based metrics (total distance covered, high speed running) is also required. The aim is to have the players achieving close to their normal training loads. High speed running tests (10m and 20m sprints) should be completed before return to play. The athlete should be close to their normal uninjured performance before return to play.

Muscle endurance can be tested, but data suggests that this needs to be completed on an isokinetic dynameter as functional exercises (eg heel raises to fatigue) may or may not identify deficits and may induce short-term muscle soreness for players (11).

Whilst balance tests are commonly used in return to play decision making for other disorders such as ligament injuries they are not useful for tendinopathies. However more complex movement tasks like cutting movements and ability to change direction at speed are important before return to play. These tests involve high strain rates and energy storage demands for the loaded tendons and could be highly provocative and thus should be performed as the final test before return to on-field training when all the basics loading tests are manageable.

Psychological state: The psychological state of the player needs consideration during the decision-making process, as withdrawal from training or competition can cause substantial problems with mental health. Apprehension, fear and anxiety following return to play have also been associated with higher risk of re-injury, as well as negative impacts upon performance (4).

STEP 2: EVALUATION OF PARTICIPATION RISK OF NEW OR RE-INJURY AND OPTIONS TO DECREASE THIS RISK

LOAD MANAGEMENT

Football is a high-speed change of direction sport that requires full fitness to participate, and there is limited possibility to passively protect the tendon in any meaningful way by taping and bracing. A comprehensive rehabilitation program that adequately prepares the athlete to tolerate the loads associated with training and match-play is the best way of protecting the athlete from relapse or recurrence. Ongoing modification of training loads may be required in order to manage the amount of load to which an athlete is exposed, in accordance with the current capacity of the tendon.

FOOT AND SHOE MODIFICATIONS

A heel raise may reduce Achilles tendon load (12). This is particularly useful in the case of an insertional Achilles tendinopathy, as a heel raise/wedge lifts the calcaneum, reducing the compression between the superior lateral calcaneum and Achilles tendon whilst also reducing the tendon load (13). It may be necessary to incorporate the heel raise in the heel of the shoe as an in-shoe raise can often cause irritation to the heel at the location of pathology, due to compressive forces between the shoe itself and the heel or may cause irritation to the superficial bursa. The same approach may be helpful in plantaris associated Achilles tendinopathy and in those athletes with limited dorsiflexion as well as those with medial and lateral foot tendon issues.

STEP 3: DECISION MODIFICATION

Other important considerations in the final decision to return to play include the time within the season, (e.g. finals, international competitions), but also the ability to have time-off after this period for rehabilitation. There may also be pressure from the athlete, coach and club encouraging continued play for the purpose of sponsorship deals, and this may represent a conflict of interest between the player, medical professionals and coach. One of the unspoken considerations in profession football is the potential for litigation against the medical team for withdrawal from play (potential loss of earnings) or serious injury resulting from early return.

SUMMARY OF RETURN TO PLAY

Throughout the entire return to play decision-making process, the medical team should continue to evaluate the symptom response to training and competition. Assuming the player's symptoms are stable, and loading is similar to normative values, then a decision of continued training/play could be made. This process of decision making is complex and varied and will not be the same for every player even in the case of a similar injury.

It is critical to assess an athlete's ability to tolerate tendon loading activities replicating those required in their sport. Readiness for return to play also needs to encompass non-medical aspects related to athlete and coach's wishes, their rehabilitation journey – smooth or bumpy as well as the risk for new or re-injury due to the time period absent from full and proper training.

MANAGEMENT STRATEGIES THAT ARE NOT CONSIDERED HELPFUL

COMPLETE REST

Resting completely can reduce tendon pain but also increases unloading and therefore dysfunction. The athlete feels better when resting but on resumption to loading and training the symptoms return immediately and can be more severe than prior to rest. Both isometrics and heavy slow resistance training are not painful and can be immediately used even in athletes with substantial tendon pain. These serve to preserve muscle strength and endurance and tendon mechanical stiffness, both essential for eventual return to sport. Continued loading of the kinetic chain and the other leg is also essential.

ECCENTRIC ONLY PROTOCOLS

Eccentric exercise is an essential part of tendon loading protocols (Figure 3). The management strategies in this section propose that they are not done in isolation and not done based on a recipe approach. Exercise programs must be tailored to the individual and their deficits, a standardised eccentric exercise program cannot provide this.

^ **Figure 3.** Ecccentric calf exercise.

PASSIVE AND ADJUNCT BASED THERAPIES

Tendinopathy management must be active, the use of passive and adjunct therapies can be used in conjunction with an active approach but must not be used in isolation.

MANAGEMENT OF PERITENDINOPATHY

The medial and lateral foot tendons have a tenosynovium because of the large range of motion they move through, hence irritation through friction loads results in a tenosynovitis. The Achilles has a tenovagium (a series of gliding membranes) posterior to the tendon (14) and repetitive plantar and dorsiflexion can irritate this structure. Other lower limb tendons have vestigial peritendon structures and do not present clinically with peritendinopathy.

Interventions to reduce repeated motion through a big range will help settle the irritation and pain. For example removing repetitive movement activities such as exercise bike and consider shoes with a higher heel to limit excursion of the peritendon over the tendon when walking around. Clinical experience suggests isometrics do not help, and anti-inflammatory approaches (topical or oral) may be more successful than in tendinopathy. Clinically the use of heparin-based creams can be beneficial (15). In the case of a mixed presentation that is there is pain from both the tendon and the peritendon, the peritendon must be managed first and settled before exercises can be progressed through range to address the tendon pathology.

MANAGING THE ATHLETE IN SEASON

The major dilemma faced by players, medical staff and coaches when managing an athlete in-season is the decision whether an athlete can continue to train and play (and if so, to what degree), or whether the athlete should cease activity and start rehabilitation.

Rehabilitation for the in-season athlete is substantially more challanging, but follows the same principles as outlined above: initial load modification in order to control symptoms, isolated muscle tendon unit rehabilitation for identified deficits, and progression of training with symptom control to a level equivalent to those usually encountered during training and competition. In practice, the complete resolution of symptoms for these athletes is often difficult, and the role of medical staff may simply be to manage the athlete's symptoms to a degree which is acceptable to the athlete until the end of the season, at which time a more substantial

rehabilitation program can be commenced. There are four key aspects of managing a player in-season.

LOAD MODIFICATION:

First line management in the competing athlete is to modify provocative load so that the player can control pain. Three aspects of load can be modified; the type of tendon load, frequency of loading and volume of load. Lastly, making the kinetic chain and other leg resilient to load will provide benefits for the affected tendon.

Type of load: Tensile or energy storage and release type loads are the most provocative for a tendon, and simply modifying activities that require substantial amounts of tensile load may assist in managing tendon pain. Changes such as reducing or removing change of direction drills, reducing the number or speed of sprint repetitions, or removing other provocative drills may have a signifiant impact upon the athlete's pain and overall performance capacity.

Similarly, it may be possible to achieve symptom relief by reducing tendon compression. In the case of hamstring tendinopathy for example, changing the athlete from a deadlift exercise to a prone hamstring curl or long leg bridge is important. In the case of insertional Achilles tendinopathy, avoiding dorsiflexion during the calf exercises but also in the rest of their program such as taking care that they stay out of dorsi-flexion with a squat by using a backsquat or elevating their heels during squatting.

Frequency of loading: Tendons respond to energy storage and release load by changing their structure, these changes are present 48 hours following loading and having recovered to baseline by 72 hours (16). This suggests that loading on alternate days allows the tendon sufficient time to recover from the previous load. Athletic tendons are generally more resilient to load, and are able to adapt to daily loading, however in the athlete with a tendinopathy, high load change of direction and sprinting may need to be on alterate days, with potential to increase the frequency of loading as tendon capacity improves.

Volume of loading: If tendon pain still interferes with training despite modifications to the types and frequency of tendon loading, then consider decreasing training duration. Two hour sessions are unlikely to be tolerated, reducing these to 1 hour or even less can allow to tendon to recover for the next training session.

Kinetic chain resilence: Landing and change of direction energy should be spread across as many joints, muscles and tendons as possible to reduce excess load on any one structure. As load is taken up by the lower limb from distal to proximal the function and capacity of the foot, ankle and calf complex is critical. Decreases in foot and ankle range of movement have been associated with both Achilles and patellar tendinopathy (17, 18). Knee and hip muscle strength and control are also critical. The capacity of the unaffected leg is also paramount, efforts to maintain or increase this are essential.

AVOID CHANGE:

Any changes in tendon load must be minimised, and it is also important to consider wider contextual factors that may also affect the tendon. These factors may include changes in footwear or playing surface. It is therefore important to carefully monitor not only the loads imposed on the tendon during training, but also other extraneous variables such as competition schedule that may also impact symptoms, in order to ensure that symptoms do not flare.

ADJUNCT TREATMENTS:

Medications and various adjuncts may help the athlete to continue to load. Any adjunct treatments that reduce pain during training without an increase in pain the following day, and do not pose a substantial risk to long-term athlete or tendon health may be considered.

PASSIVE

Most passive interventions give short term pain relief and often do not change load related pain. Repsonses to passive interventions are likely to be highly individualised, and therefore if a particular intervention is helpful for an athlete, and is known to be safe and legal, the its use may be considered despite a lack of research evidence supporting its efficacy.

ACTIVE

Isometrics may change pain and increase strength and can be used before training and playing. The right exercise prescription may be critical (long duration and heavy).

Both isometric and heavy slow resistance training can be continued as neither are provocative for tendon pain. Strengthening the unaffected side can benefit both sides (1, 2) and promoting load absorption through maintaining capacity in the other muscles is a critical part of managing the in-season athlete.

DO ISOMETRIC EXERCISES RELIEVE TENDON PAIN?

Some tendon chapters in this book advocate for isometric exercise and others do not. There are strong clinical and research opinions regarding their effectiveness. This section will review the evidence for isometric exercises in tendinopathy and propose a clinical pathway for their use (Tables 2-4).

Isometrics were first applied to patellar tendinopathy and showed an immediate and substantial improvement in pain as well as a reduction in cortical inhibition (19). Further studies have looked at other tendon responses to isometric exercise, however no other studies have examined cortical responses.

A significant degree of variability in effectiveness of isometric exercise is present within the research literature. There are likely several clinically important reasons for this variability. Firstly, the load and duration of isometric holds varies significantly between studies. The original study used a heavy load of 70% maximal voluntary isometric contraction (MVIC) for a long duration of 45 seconds, with five repetitions completed with 2 minutes between to allow for muscle and cortical recovery (19). Numerous different loading parameters and durations have been trialled following this study, with varying results. The second reason for this variability may be due to the uncertainy in diagnostic criteria used to identify tendinopathy in these studies. It is important to exclude pain from other structures, as heavy isometric loads may be ineffective or even aggravate other structures that may masquerade as tendon pain.

EVIDENCE ON ISOMETRICS IN ACHILLES TENDINOPATHY AND PLANTAR HEEL PAIN

TITLE	AUTHORS	DIAGNOSTIC CRITERIA	LOAD	OUTCOMES
Acute sensory and motor response to 45-S heavy isometric holds for the plantar flexors in patients with Achilles tendinopathy.	O'Neill, S., Radia, J., Bird, K., Rathleff, M. S., Bandholm, T., Jorgensen, M., & Thorborg, K	Localised Achilles tendon pain, aggravated by dose dependent manner, pain on palpation, positive London hospital test/ painful arc sign, positive ultrasound findings.	5x 45 sec isometric plantarflexion holds at 70% MVIC with 2 mins rest.	No meaningful acute benefit for sensory or motor output
The effect of isometric exercise on pain in individuals with plantar fasciopathy; a randomised crossover trial.	Riel, H., Vicenzino, B., Jensen, M. B., Olesen, J. L., Holden, S., and Rathleff, M. S.	Minimum three-month history inferior heel pain, pain on palpation, thickness of the plantar fascia, pain during static stance, squat or heel raise.	Heel raise for forefoot on step. Load equal to the heaviest load that could be sustained for 1 min. 5x 45 sec holds with 2 mins between, once daily.	No significant difference between isometric exercise, isotonic exercise or walking
Isometrics do not provide immediate pain-relief in Achilles tendinopathy	Van Der Vlist, A., van Veldhoven, P., van Oosterom, R., Verhaar, J., de Vos, R.	Pain on Achilles mid-portion and ultrasound findings	Single session of isometrics vs isometrics vs rest	No significant change in VAS scores in any group

^ **Table 2 .** Evidence for isometrics in Achilles tendinopathy and chronic plantar heel pain.

EVIDENCE ON ISOMETRICS IN PATELLAR TENDINOPATHY

TITLE	AUTHORS	DIAGNOSTIC CRITERIA	LOAD	OUTCOMES
Isometric exercise induces analgesia and reduces inhibition in patellar tendinopathy.	Rio, E., Kidgell, D., Purdam, C., Gaida, J., Moseley, G. L., Pearce, A. J., & Cook, J.	Pain localised to inferior pole of the patella with jumping or landing activities or single leg decline squat and confirmed with ultrasound imaging	5x 45 sec holds on leg extension machine with 70% MVIC with 2 mins rest between	Immediate reduction in pain and cortical inhibition for at least 45mins following isometric exercise
Isometric Contractions Are More Analgesic Than Isotonic Contractions for Patellar Tendon Pain: An In-Season Randomized Clinical Trial	Rio, E, Van Ark, M., Docking, S., Moseley, G.L., Kidgell, D., Gaida, J. E., Van Den Akker-Scheek, I., Zweryer, J., Cook, J.	Pain localised to inferior pole of the patella with jumping or landing activities or single leg decline squat and confirmed with ultrasound imaging	5x 45 sec holds on leg extension at 80% MVIC with 1 min rest between	Isometrics elicited greater immediate analgesia throughout the four-week trial
Isometric Exercise to Reduce Pain in Patellar Tendinopathy In- Season: Is It Effective "on the Road"?	Rio, E., Purdam, C., Girdwood, M., & Cook, J	Pain localised to inferior pole of the patella, pain aggravated by energy storage and release loads, minimum 2/10 pain on single leg decline squat	5x 30 sec holds of double leg squat using a rigid belt	Significant reduction in pain following four-week trial
Do isometric and isotonic exercise programs reduce pain in athletes with patellar tendinopathy in- season? A randomised clinical trial.	Van Ark, M., Cook, J. L., Docking, S. I., Zwerver, J., Gaida, J. E., Van Den Akker-Scheek, I., & Rio, E	Focal tendon pain at superior or inferior pole of the patella and history of exercise induced knee pain in the same location	5x 45 sec single leg isometric holds on leg extension machine at 80% MVIC	Clinically important decrease in pain. No significant difference between isometric and isotonic groups.
Immediate and short- term effects of short- and long-duration isometric contractions in patellar tendinopathy	Pearson, S. J., Stadler, S., Menz, H., Morrissey, D., Scott, I., Munteanu, S., & Malliaras, P.	Pain localised to inferior pole of patella aggravated by jumping and abnormalities on ultrasound	85% MVC on leg extension, either six repetitions of 40 sec duration or 24 repetitions of 10 sec duration.	Reduced pain with single leg decline squat and hop testing, no significant differences between groups.
Isometric exercise and pain in patellar teendinopathy: A randomised cross-over trial	Holden, S., Lyng, K., Graven-Nielsen, T., Riel, H., Lars, H	Pain localised to inferior pole of patella	Single session of high load isometrics vs dynamic isotonics with cross-over at 7 days	Significant reduction in SLDS but no difference between groups

^ **Table 3.** Evidence for isometrics in patellar tendinopathy.

EVIDENCE ON ISOMETRICS IN OTHER TENDINOPATHIES

TITLE	AUTHORS	DIAGNOSTIC CRITERIA	LOAD	OUTCOMES
The effectiveness of isometric contractions combined with eccentric contractions and stretching exercises on pain and disability in lateral elbow tendinopathy. A case report.	Stasinopoulos, D.	Pain on palpation, aggravated by wrist and middle finger extension and gripping.	Three sets of ten wrist extension holds for 10 seconds with no external resistance	Combination of isomeric and isotonic exercises can produce significant improvements in pain.
Unsupervised isometric exercise versus wait and see for lateral elbow tendinopathy	Vuvan, V., Vicenzino, B., Mellor, R., Heales, L. J., and Coombes, B. K.	Lateral elbow pain > 6 weeks, pain on palpation, pain with resisted wrist or second or third finger extension, pain with gripping, or forearm stretching and decreased grip strength.	4x 30-45 sec isometric wrist extension holds with 20-35% MVC	Greater improvement when compared to 'wait and see' at 8 weeks
Isometric exercise above but not below an individual's pain threshold influences pain perception in people with lateral epicondylalgia	Coombes, B. K., Wiebusch, M., Heales, L., Stephenson, A., & Vicenzino, B.	At least two of the following; pain on palpation, pain on resisted wrist or middle finger extension, pain with forearm stretching, >30% strength deficit compared to contralateral side.	10x 15 sec isometric wrist extension holds with 15 secs recovery, completed with a load 20% above or below pain-free threshold.	Increased pain if exercise completed above pain free threshold.
Isometric versus isotonic exercise for greater trochanteric pain syndrome: a randomised controlled pilot study.	Clifford, C., Paul, L., Syme. G., and Millar, N. L.	Lateral hip pain >3 months, pain on palpation, pain with one of five pain provocation tests	Side lying hip abduction- six 30 sec holds with one minute between. Standing hip abduction- 3x 10 reps with 60 secs between (standing on affected leg)	Both isometric and isotonic exercise groups improved at 4 weeks. No significant difference between groups.

^ **Table 4.** Evidence for isometrics in other tendinopathies.

CLINICAL APPLICATION

Isometric exercises are a simple and low risk intervention that can be trialled for athletes with tendinopathy. A pragmatic approach would be to trial isometrics and to include them in an athlete's rehabilitation program if they provide a pain-relieving effect. They can be particularly effective for this purpose prior to training or playing especially in the athlete continuing to play wirh tendinopathy. The cortical effects of reducing cortical inhibition suggest they may also be beneficial prior to completing a strength training program, as the motor drive to the muscle will be greater (19).

Clinical experience suggests that shorter duration holds are less effective. However, there is some evidence that the total time under tension may be more important than the duration of each individual repetition (20). This may represent a viable option for athletes unable to tolerate extended duration holds, whereby a larger number of shorter duration holds may be used to elicit a similar tendon response.

It is recommended that the isometrics are trialled as they were originally proposed and used in this fashion if effective. If ineffective, the exercise should be modified or abandoned. The source of the pain should also be reviewed, to evaluate the possibility of a non-tendon source of pain.

Isometrics should be used as part of the initial pain management strategy, and then as a pain modifier when the athlete returns to training or play. They can also be of benefit prior to the athlete commencing their strength program. They should not be used in isolation, nor should they be continued when they are no longer required for pain control. They should be viewed as one component of a comprehensive rehabilitation program.

A CLINICAL REHABILITATION PLAN

A standard rehabilitation program would include four stages (Table 6).

1. Isometrics can be used as a start point for treatment if they prove helpful. It is critical that they are used for correctly diagnosed tendon pain and that these are heavy and of long duration. They can provide pain relief for several hours and can be used before training and games. These are used early in the program (and rarely in isolation) and can be used before heavy slow resistance training to decrease cortical inhibition.

2. Heavy slow resistance is used as soon as feasible. As what constitutes a 'heavy' load is highly variable depending on the individual, these exercises may range from weighted exercises in the gym (as is the case in most athletes) to simple body weight exercises for less active or older individuals (more of a strength endurance program). It is crucial to load the affected muscle tendon unit as well as the muscles in the rest of the kinetic chain (calf, quadriceps, hamstrings, gluteals). The calf complex is essential for all lower limb tendinopathies as it is a major shock absorber in athletic activity, both landing from a jump and in change of direction tasks (Figure 4). Once some strength is gained then more functional strength endurance exercises are performed, and strength is maintained for an extended period, usually until the end of an athlete's career. People with unilateral tendinopathy have asymmetries in strength so where possible the heavy slow resistance should be completed as an isolated, single leg program with some multi-joint exercises. Continued maximal loading of the unaffected limb is essential. This part of the program can take up to 12 weeks to gain sufficient strength and endurance to progress to the next step in rehabilitation. The time is dependent on the length of symptoms prior to clinical presentation, which in turn determines the amount of muscle, tendon, kinetic chain and brain unloading.

3. Adding speed to the program is the first time a provocative load is placed on the tendon. The load must be applied 2-3 times a week to allow the tendon to recover between bouts of loading. Start with slow energy storage loads (Figure 5) and progress the speed before adding the energy release part of the exercise sequence. The athlete needs to continue with their gym program on the non-speed days. This step requires evidence that the tendon is tolerant to these loads, that the pain the following day remains stable and that there is progress in the speed and the number of repetitions the tendon tolerates. Note that pain doesn't need to, and may not, be zero but pain needs to be low and stable. Pain that is the same or better the next day indicates the tendon has tolerated the loads the day before. This information is used to progress the loads the next time the athlete completes a speed day. If they have an increase in pain then reducing the loads on the next speed day is essential, but they should continue with heavy slow loads and isometrics if they found them useful. Changing one thing at a time is helpful as if the athlete has a flare it will be small and the load is easily modified at the next session.

4. Once the tendon is tolerant to energy storage and release loads then sport specific loading is incorporated into the program, again 2-3 times a week, replacing the third stage of the program (Figure 6). The heavy slow resistance training continues on the other days. The number of repetitions, type of exercise and frequency per week must be increased to reflect training loads. This stage may take several weeks as the tendon can take time to adapt to these loads.

5. Return to training and competition must be progressive, in terms of frequency, volume and intensity. Rushing a person back into coach-controlled training often flares the tendinopathy, the clinician should control aspects of training until the tendon becomes resilient to these loads.

^ **Figure 4.** Single leg seated calf raise.

^ **Figure 5.** Early energy storage and release drill.

^ **Figure 6.** Return to training.

STAGE OF REHAB	TYPE OF EXERCISES	EXAMPLES FOR EACH TENDON SINGLE LEG EXERCISE ESSENTIAL IF TOLERATED			
		Achilles	**Patellar**	**Hamstring**	**Adductor**
1	Isometric	Standing calf raise hold; body weight or calf machine (modify range to avoid compression)	Leg extension holds Spanish squat	Double or single leg bridges Prone hamstring curl	Adductor squeeze with bent knees
2	Heavy slow resistance	Standing and seated calf raise	Leg extension, leg press	Bridges, hamstring curls	Adduction machine Standing adduction against heavy resistance elastic/cable) Copenhagen adduction exercise (short progressing to long lever)
	Functional endurance	Standing calf raise to fatigue Stair climbing on toes	Walk lunges Stair climbing on toes, especially 2-3 stairs ascent	Arabesque, RDLs within suitable range	Standing adduction against resistance to fatigue (elastic/ cable)
3	Energy storage and release loads	Skipping, stair running (running up before running down)	Stair running split squats	Spilt squats, split jumps	Standing adduction against resistance-fast and/or combined movements and kicking Slide board
4	Return to training loads	Running, sprinting, change of direction	Deceleration, change of direction, jumping	Bent forward running, tackling	Change of direction, agility drills

^ **Table 6.** A staged rehabilitation program.

Barça Way

FC Barcelona's general approach to managing tendinopathy involves a three pronged approach; 1. control of training loads, 2. pain relief and 3. strengthening and conditioning of the musculoskeletal system.

- **1. Control training load:** This involves careful monitoring of the external load of players using GPS and concomitant monitoring of the internal response using rating of perceived exertion.
- **2. Pain relief:** We use isometric contractions to acutely decrease the pain. The isometric exercises are an initial treatment during the in-season for pain management. We use protocol of 5 sets x ~45 secs (30 to 60s) single leg isometric contraction of each leg on a leg extension machine in patellar tendinopathy. In Achilles tendinopathy, the isometric contractions and resistance bands as the initial treatment are recommended, however the concentric and eccentric contractions can be involved in combination with isometric exercises. Other tools that could be used for pain relief are the physiotherapy, cryotherapy, extracorporeal shock wave therapy.
- **3. Strength & conditioning:** Tendon pain negatively affects the muscle ability to produce the appropriate strength. Muscle fatigue and decrease of strength can cause a deterioration of pain. A lower limb strength program 2 to 3 times per week must be implemented. Blood flow restriction training 2 to 3 times per week after training could have a pain-relieving effect.

Summary:

- Initial management of the athlete with tendinopathy needs to consider the tendon involved, the kinetic chain it functions in, the loading history of the tendon, the irritability and severity of pain and the athlete's sport.
- Loading of the tendon should be graduated and progressed according to the athlete's symptoms. Symptoms should be monitored in the morning on a standardised loading test at the same time each day, during training/loading and after training. Symptoms should be stable or improve over time.
- Isolated loading of the muscle-tendon unit is required prior to progression to multi-joint exercises. It is critical that exercises are performed single leg, and that each leg is loaded maximally and independently.
- Once an initial strength and endurance base has been developed, higher magnitude (faster) loading is introduced. The decision as to when to progress is individualised, and depends upon the strength, symptoms, current exercise tolerance and ability of the athlete to cope with the proposed demands.
- Recurrence rates may be greater with shorter rehabilitation periods because of insufficient time to regain tendon capacity with the rapid transition back to full training and match-play.

Clinical Implications:

- Initial management needs to consider the involved tendon, relevance of the different loading types, irritability and severity of pain and the athlete's sport.
- Tendon pain cannot be considered in isolation, as resultant alterations in an athlete's capacity to sprint and change direction impacts both upon their game and leaves them vulnerable to other injuries. Therefore, consideration of the consequences to the entire kinetic chain is required as part of the decision-making process.
- Education of both athlete and coach of factors that contribute to tendinopathy development and management is essential, especially about tendon load. A fundamental understanding of what constitutes high tendon load enables the modification of training in accordance with the tendon's current capacity and assists the athlete to self-manage their condition.
- Improvements in the strength of the affected leg may be enhanced by strength improvements of the unaffected leg. This phenomenon may be useful in the early stages of rehabilitation.
- Initial return to running progression should be based on the athlete's preferred running speed, as this is the speed at which tendon load is optimal for its current capacity.
- Tendon pain throughout rehabilitation may not always be zero, nor is this a requirement for return to play. Pain that is low and stable indicates tolerance to loading.
- Resting completely can reduce tendon pain but also increases unloading and therefore dysfunction.
- The goals of in-season management of tendinopathy may vary from those during the off-season. Complete resolution of symptoms in these athlete's is often difficult, and the role of medical staff may simply be to manage the athlete's symptoms to a degree which is acceptable to the athlete until the end of season, at which time a more substantial rehabilitation program can be commenced.
- Isometric exercises are a simple and low risk intervention that can be trialled for athletes with tendinopathy. A pragmatic approach would be to trial isometrics and to include them in an athlete's rehabilitation program if they provide a pain-relieving effect. If ineffective, the exercise should be modified or abandoned. They should not be used in isolation, nor should they be continued when they are no longer required for pain control. They should be viewed as one component of a comprehensive rehabilitation program.

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— Justin Lee and Sean Docking

2.3. THE ROLE OF IMAGING IN DIAGNOSING AND MANAGING TENDINOPATHY

In 1895, Wilhelm Conrad Roentgen saw the bones of his wife's hand on X-ray and opened the world's eyes to imaging inside the body. This serendipitous discovery saw technological advances and a medical revolution that enabled visualisation of tissues and organs and improved diagnosis and treatment of disease. But 100 years on from Roentgen's discovery, we are in the midst of another revolution in understanding how imaging provides clinical value and positively impacts patient outcomes for musculoskeletal conditions such as tendinopathy.

Tendinopathy is characterised by pain and loss of function and the diagnosis is made by taking a careful clinical history and completing a physical examination, with expert consensus stating that imaging is not always necessary for diagnosis (1). There are times where imaging can provide useful clinical information, but occasions where imaging may be unnecessary and potentially harmful. Identifying the right patient at the right time to undergo the right imaging tests are key principles in ensuring imaging's appropriate use.

POINT 1: WHICH IMAGING MODALITY?

The choice of imaging modality depends on the clinical presentation, patient/player/club preference, cost, availability, and clinical indication (Table 2).

X-RAY AND COMPUTED TOMOGRAHY

Several stadia around the world have X-ray facilities. However, unlike bone and joint injury, plain radiography has a relatively limited role in the assessment of tendon injury. A plain radiograph may identify entheseal, intra-tendinous or peritendinous calcium deposits that may not be visible on other imaging modalities. The plain radiograph may also detect small tendon avulsions and secondary signs of tendon rupture, such as patella alta following infrapatellar tendon rupture. Computed tomography (CT) similarly has limited role in the assessment of tendon injury as tendons are poorly visualised and the imaging incurs a substantial ionising radiation penalty.

ULTRASOUND

Ultrasound imaging provides excellent visualisation of the internal architecture of the tendon, to the level of individual collagen fascicles. Normal tendon has a uniform echogenicity (brightness) due to its highly organised structure, with parallel fascicles observed on the longitudinal view. Various abnormalities can be observed on ultrasound (Table 1).

Ultrasound is increasingly being performed in stadia medical rooms and, in combination with an experienced operator, can provide a rapid dynamic assessment of the tendon at relatively little cost. However, as ultrasound is user-dependent, substantial training and experience is required for appropriate use. Altering the transducer tilt angle by as little as 5º can create an imaging artefact that is indistinguishable from tendon abnormality (2). Interpreting how the images contribute to diagnosis and management of tendinopathy adds another layer of complexity.

ABNORMALITY	IMAGING APPEARANCE
Intra-tendinous abnormalities	
Tendon thickening	Can appear as uniform thickening over the length of the tendon or confined to a focal segment of the tendon
Hypoechogenicity (decrease in tendon brightness)	Represents a loss in the aligned fibrillar structure of the tendon. Differentiating whether this hypoechoic area represents an area of degeneration or a macroscopic tear is complicated, with no reliable methods for distinguishing between them
Vascularisation	Infiltration of blood vessels and increased intra-tendinous flow can be observed on Doppler ultrasound
Calcification	Seen as hyper-intensive areas, frequently at the tendon insertion, but can be seen within the tendon
Extra-tendinous abnormalities	
Bursa	Bursal thickening and effusion are often seen and bursal hyperaemia on Doppler imaging may be present.
Peritendon changes	Tendon sheath thickening and the presence of fluid within the peritendinous space is seen. Dynamic ultrasound may reveal adhesions between the sheath and tendon

^ Table 1 . Summary of abnormalities observed on ultrasound.

MAGNETIC RESONANCE IMAGING

Ultrasound has the resolution to accurately observe the fibrillar internal tendon structure, but magnetic resonance imaging (MRI) is superior in providing an accurate 3-dimensional visualisation of the tendon and surrounding structures. Standard MRI protocol for tendon imaging typically includes a combination of sequences to optimally visualise anatomy of the region and changes within the tendon. There are typical sets of sequences for tendons, such as the Achilles tendon.

In normal tendons, the water protons are tightly bound to the collagen fibres resulting in a very short T2 values, which results in the tendon appearing dark on all conventional MR sequences (3). Magnetic resonance imaging has typically been used to measure tendon dimensions (4, 5) and to identify intratendinous fibrillar disorganisation. The change from collagen type I to type II-III and changes in the hydration state of the tendon result in an increase in MR signal within the tendon. Neither hypoechogenicity on ultrasound nor high intratendinous signal on MRI can differentiate degeneration from partial-thickness tear (6). Furthermore, intratendinous microcalcifications and mild neovascularization cannot be identified on MRI. A tendon abnormality on imaging may also show bone or soft tissue oedema, which is seen as increased signal intensity on T2 or STIR.

An area of increased MR signal cannot be ascribed to specific pathoanatomical findings (partial thickness tear, neovascularisation, delamination) (Figure 1). Intratendinous increased signal intensity alone may be oedema, granulation tissue, neovascularisation, ganglia cysts or calcification. The diagnosis of partial tear is a particular challenge on imaging.

Peritendinopathy can be seen on MRI as a thickened peritendon. It is mandatory to study the tendon as part of a bone-fat pad-peritendon-tendon complex, as all these structures are connected by neurovascular and connective tissue structures (7). Peritendon abnormalities can accompany tendon abnormality but can also occur without underlying tendon pathology.

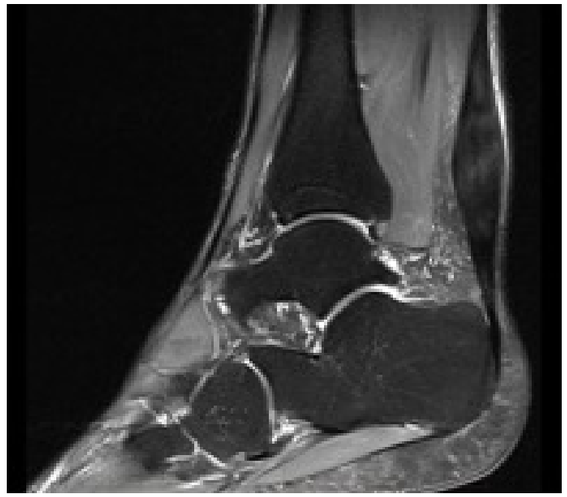

Figure 1. Sagittal fat saturated fluid sensitive MRI demonstrating classic fusiform expansion of the Achilles tendon with ill-defined areas of increased signal within the tendon.
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	ULTRASOUND	MRI	X-RAY/CT
Intratendinous abnormalities (thickening, fibrillar structure)	✓ ✓ ✓	✓	X
Complete rupture	✓ ✓ ✓	✓ ✓ ✓	X
Surrounding structures (bursa, sheath)	✓ ✓ ✓	✓ ✓ ✓	X
Visualisation of anatomical area	✓	✓ ✓ ✓	X

^ Table 2 . Suitability of ultrasound, MRI, x-ray, and CT in visualising various structural tendon abnormalities.

POINT 2: IS IMAGING GOING TO PROVIDE USEFUL INFORMATION TO BENEFIT THE PATIENT?

HOW ACCURATE IS IMAGING IN THE ASSESSMENT OF TENDINOPATHY?

Tendinopathy is a clinical syndrome and imaging alone cannot diagnose tendinopathy. Studies show the accuracy and sensitivity for both ultrasound and MRI are relatively high when using clinical assessment as the gold standard. Direct comparisons of MRI and ultrasound in the diagnosis of tendinopathy shows ultrasound has a higher accuracy assuming the study was performed by an experienced operator (7). This increased accuracy may be due to the increased spatial resolution and real time analysis to pinpoint site of symptoms and perform dynamic manoeuvres. While the data on the accuracy of imaging in diagnosing tendinopathy may be impressive, serious limitations remain. These studies are unable to determine whether imaging is superior to, or provides additional information beyond, a thorough clinical assessment to diagnose and differentiate tendinopathy from other conditions. This is not to say that imaging is unhelpful in diagnosis, but simple measures of diagnostic accuracy do not support or refute the use of imaging.

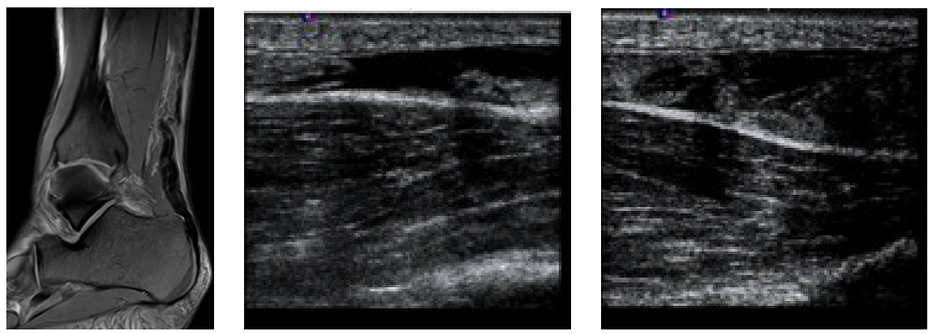
WHAT IF DIAGNOSIS IS EQUIVOCAL FOLLOWING CLINICAL EXAMINATION?

No clinical test is 100% accurate and clinicians may be unable to rule tendinopathy or other differential diagnoses in or out following clinical examination. Diagnostic uncertainty and differential diagnosis does not always require imaging, especially in circumstances where treatment is not altered based on imaging findings (differentiating retrocalcaneal bursa involvement from insertional Achilles tendinopathy) or imaging is inaccurate in differentiating conditions (differentiating patellar tendinopathy from patellofemoral pain).

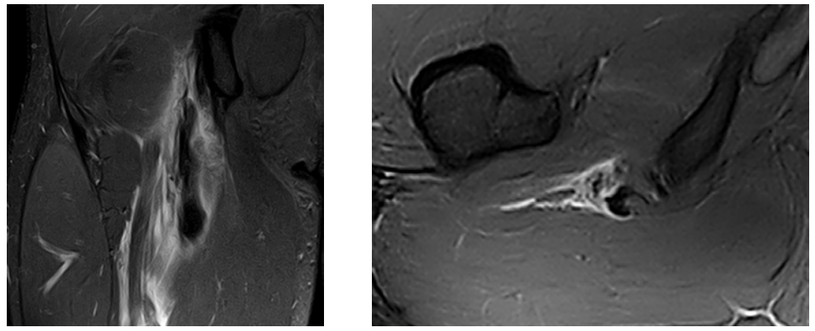
Imaging can be used to visualise the presence or absence of structural abnormalities, for example, the tendon may be structurally normal on imaging allowing the clinician to rule out tendinopathy as a clinical diagnosis. Similarly, a clinician may be unable to determine whether an athlete has typical Achilles tendinopathy pain or if the presentation is complicated by peritendinopathy. This differentiation is important as it may require alternative treatment pathways to ensure return to play (using strategies to reduce friction loads that have irritated the surrounding tendon sheath). Imaging in this case may be useful to identify sheath thickening and fluid within the peritendon.

WHAT IF DIAGNOSIS IS TENDON RUPTURE?

Imaging findings of tendon rupture may determine the need for surgical repair. In Achilles tendon rupture, a tear gap of less than 5 mm assessed on dynamic ultrasound with foot in equinus position (Figure 2) has been shown to correlate with reduced re-rupture rate during conservative management (8). A decision on surgical repair of full-thickness avulsions of the hamstring complex from the ischial tuberosity are often based on imaging findings including the length of tendon retraction on MRI (Figure 3) (9).



^ Figure 2. A. MRI High Achilles rupture in 28 year old athlete (male). B. Dynamic sonogram of the same patient in neutral (B) and equinus (C) position showing apposition of tendon ends dynamically.



^ Figure 3. Coronal (A) and axial (B) MRI of the right hamstring origin in a 20-year old male hockey player, with acute semimembanosus avulsion. Note the intact conjoint semitendinosus-long head bíceps femoris tendon and complete avulsion of the semimembranosus proximal tendon.

POINT 3: IS THERE POTENTIAL FOR IMAGING TO HARM THE PATIENT?

Imaging has the potential to identify clinically unimportant incidental findings that lead to unnecessary treatment. A high proportion of asymptomatic physically active individuals who have no history of lower limb tendinopathy exhibit abnormality on tendon imaging (10). Nearly one in five physically active asymptomatic individuals will exhibit an abnormal Achilles or patellar tendon. Interpreting the relevance of imaging findings is critical as tendon abnormalities can co-exist with other pain conditions. Approximately 32-72% of individuals with patellofemoral pain exhibit patellar tendon abnormalities, confusing an already complicated diagnostic picture (11, 12). It is important to iterate that imaging findings are not necessary to make a diagnosis of tendinopathy and incidental imaging findings may confuse the diagnostic picture for both the clinician and the athlete.

PROGNOSTIC VALUE OF IMAGING IN ATHLETES

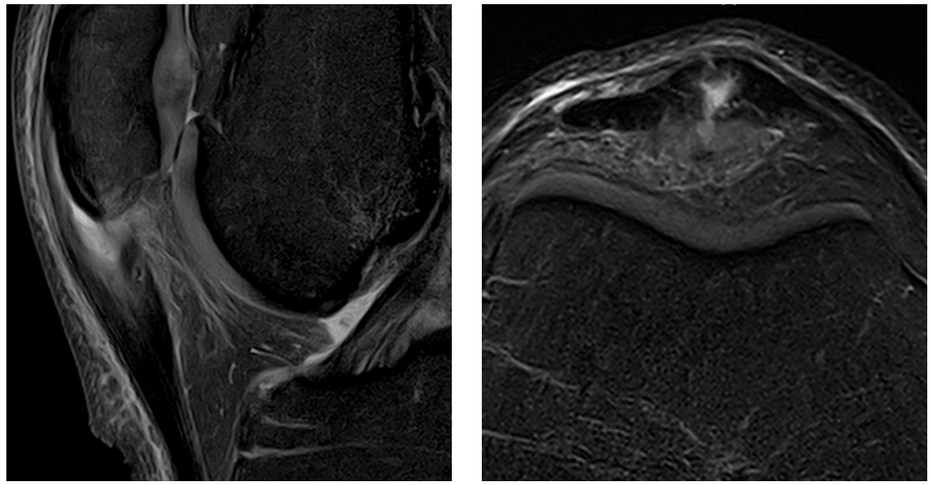
Imaging athletes in elite sport is proposed to identify at-risk athletes so prevention strategies can be implemented to reduce the impact of tendon injuries. Abnormalities observed on imaging are one of the strongest known risk factors for the development of tendinopathy. Those with an abnormal Achilles or patellar tendon are approximately 7 and 4 times respectively more likely to develop symptoms than those with a structurally normal tendon (13, 14). However, only about one fifth of those with an abnormal tendon will develop symptoms (13, 14). You would need to scan 167 athletes with no history of Achilles tendon pain to identify five athletes who will develop symptoms over the course of the season*. Although imaging may be a potential screening tool, it may lead to unnecessary anxiety for the player and the club if incidental asymptomatic findings are detected. Monitoring progress through rehabilitation using imaging is also unrewarding, as symptom improvement can occur without imaging changes.

* The prevalence of abnormalities in asymptomatic athletes is ~15% (167 x 0.15 = ~25 athletes) (14). Only 20% of those athletes develop symptoms (25 x 0.20 = 5 athletes)(13, 15).

WHAT TO REPORT ON IMAGING AND WHAT IT MEANS TO THE CLINICIAN AND PATIENT

Accurate communication of imaging findings to the athlete and clinical team can enhance its utility and ameliorate unnecessary anxiety. A systematic review found that the use of a more medicalised term to describe a condition was associated with higher ratings of anxiety and perceived severity, and a preference for more invasive treatments (15). Furthermore, terminology that suggests a structural abnormality can impact adherence to an exercise-based rehabilitation.

Partial tears are often reported on tendon images, and the reporting of a partial-thickness tear may cause the athlete to think of their tendon as less capable of tolerating load, and that any form of exercise should be avoided for fear of complete rupture. However numerous studies that compared imaging to surgical findings have found that both US and MRI are limited in their ability to differentiate between an area of degeneration and a partial tear (Figure 4) (6, 16, 17). Diagnosing partial tears is difficult using either imaging or clinical assessment.



^ Figure 4. Sagittal (A) and axial (B) MRI through the right knee. Despite the intense high-signal and distortion of the posterior margin of the tendon, imaging cannot reliably diferentiate tear from tendon degeneration. The axial image demostrates a small cavity of fluid inside the high-signal area that we could consider as a tear but it could also be a small haematoma, focal myxoid change, hypervascular granulation tissue or a ganglion cyst.

POINT 4: NEW IMAGING MODALIITIES / TECHNIQUES

Recent developments in transducer technology and computing power have allowed ultrasound tissue characterisation (UTC) and ultrasound elastography (USE) into mainstream imaging of tendon disorders. Developments in MRI field strength and coil technology have also seen in an interest in the use of ultrashort TE (UTE) MRI sequences in tendon assessment.

UTE MRI SEQUENCES

An increase in non-collagen extracellular matrix and disorganisation of collagen structure in tendinopathy affects the T2 and T2* values of the tendon. The shortest time to echo (TE) in routine spin-echo MRI is 8-10 msec, by which time the short T2 signal of tendon has disappeared (18). Ultrashort time to echo (UTE) pulse sequences can achieve echo times as short as 0.05-0.5 msec enabling measurement of very short T2 values within tendon (19, 20).

Gardin et al demonstrated that raised T2 values within the Achilles tendon helped differentiate between chronic tendinopathy and healthy controls but this was not associated with symptoms and function measured by VISA-A (21). Ultrashort time to echo sequences and other novel sequences including dynamic contrast enhanced MRI have been available for several years but have not become routine practice. Pragmatics such as scanning time, cost-benefit assessment and reproducibility may be the reason that tendinopathy is principally a clinical diagnosis.

ULTRASOUND TISSUE CHARACTERISATION

Ultrasound tissue characterisation (UTC) uses an automatic tracking unit to serially capture transverse ultrasound images of the tendon at 0.2mm segments rendering a 3-dimensional image (22, 23). It combines the high spatial resolution of ultrasound with the 3-dimensional visualisation of MRI. This allows quantification of tendon structure based on the alignment of tendon fibres removing subjective interpretation. The ability of UTC to detect subtle tendon changes has enabled research not possible with conventional imaging modalities. Subtle changes in tendon structure in response to exercise (24), the limited association between extent of disorganisation and symptoms (25, 26), and the suggestion that tendon thickening is the pathological tendons mechanism to adapt for areas of disorganisation (27), have all been observed using UTC. However, UTC's ability to quantify tissue structure does not improve the limited association between imaging findings and symptoms. Ultrasound tissue characterisation is limited to large, linear tendons such as the Achilles and patellar tendon.

ELASTOGRAPHY

Elastography evaluates the mechanical properties of tendon. Two types of elastography (compressive and shear-wave) are used in clinical practice. Compressive elastography captures tissue displacement when the scanner applies pressure via the probe. Shear-wave elastography uses an ultrasound pulse to measure the shear-wave velocity and provide an estimation of the mechanical properties of the tendon. Like UTC, it generates quantifiable measures of tendon mechanical properties. However, its clinical utility is not yet established.

CONCLUSION

Ten years after Roentgen's discovery, Karl Benz patented the first motor vehicle and provided transportation, mobility, and independence to the world. Our understanding of how to operate vehicles safely did not occur overnight. In the years following, there have been laws, infrastructure, and technological advances to maximise the benefit and minimise harm from operating cars.

Clinical imaging has the potential to greatly benefit patients by assisting in diagnosis and treatment planning, yet can take a patient (and clinician) on a pathway where there are considerable harms. Understanding the appropriate use imaging in tendinopathy will maximise its value to both players and clinicians.

Barça Way

Tendon imaging (magnetic resonance and ultrasound) can be used to evaluate tendon injuries and assist in their diagnosis. There is still much to learn about the role of imaging in clinical presentation of tendinopathy. Magnetic resonance imaging allows visualisation of the tendinous structure, but it does not represent the entire clinical picture and should not be used as the sole diagnostic criterion in determining whether the clinical presentation is generated by the tendon (6).

The key points to consider when assessing tendon pathology with ultrasound are that:

- It is known that an asymptomatic tendon with structural changes on US has 5 times more risk of developing tendinopathy (13). Despite this, structural changes can NOT be considered by themselves as a predictor of tendinopathy because pain and pathological changes are not directly related.
- Ultrasound structural changes and individual predisposing factors (load increase, inadequate materials, systemic diseases, lower limb mechanics) can help in the prediction of tendinopathies.
- Ultrasound is the best imaging method to assess tendon pathology although MRI can provide a better differential diagnosis.
- Every reported tendon pain in FCB is scanned with ultrasound in order to assess:
  - anatomical variability
  - Hoffa and Kager fat pad status
  - peritendon status
  - neovascularisation
  - suspected intrasubstance partial tear
  - thickness and echogenicity
  - calcification
- Each ultrasound exam is also performed with dynamic manoeuvres and with tension and relaxation.

Summary:

- Tendinopathy is principally a clinical diagnosis, and imaging may not be necessary for the diagnosis.
- Plain radiography and CT has a limited role in assessment of tendon injury.
- Ultrasound is a commonly used imaging modality with excellent visualisation of the internal architecture of the tendon. However, the accuracy of ultrasound is highly user dependent.
- MRI provides accurate three-dimensional visualisation of the tendon and surrounding structures, however, similar to ultrasound, intratendinous high signal on MRI cannot distinguish between degeneration and a partial thickness tear.
- The diagnosis of a partial tear on imaging is challenging as findings are non-specific.
- Radiologists should employ the same terminology as other clinicians using the terms from a consensus in terminology for persistent tendon disorders.
- A normal tendon suggests the tendon is not the source of pain and dysfunction.

Clinical Implications:

- There is the potential for clinically unimportant incidental findings. These findings may confuse the diagnostic picture and lead to unnecessary treatments. Imaging should only be considered in a situation where findings may alter the management of an athlete.
- The use of medicalised terminology may create unnecessary anxiety for athletes and may cause them to perceive their injury as being more severe, often resulting in a preference for more invasive treatments. Clinicians should be cognisant of the language used when discussing imaging findings with patients, as this may impact on rehabilitation.
- Clinical imaging has the potential to be of great benefit when used appropriately, but also may result in considerable harm when used indiscriminately or without careful consideration.
- Imaging has not been shown to be helpful for monitoring the rehabilitation, improvement in pain and function are considered positive outcomes.

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— Robert-Jan de Vos and Hans Tol

2.4 ADJUNCT TREATMENTS FOR TENDINOPATHY

Much clinical and research effort has aimed to find ways to stimulate tendon recovery through agents that are (1) anti-inflammatory and pro-inflammatory, (2) pain reducing, (3) aimed at increasing or decreasing tendon neovascularisation and (4) aimed at restoring tendon tissue structure. The adjunct treatment classes that have been investigated for these potential beneficial effects are extracorporeal shockwave therapy, medication and injection therapy. We will review their scientific background, the clinical evidence supporting their use, and provide evidence-based recommendations.

EXTRACORPOREAL SHOCKWAVE THERAPY (ESWT)

Extracorporeal shockwave therapy (ESWT) applies local high-energy pulses. There are two types of ESWT: radial and focussed ESWT. Radial shock waves result in a diverging pressure field, which reach a maximal pressure at the source and not at a selected depth in the tendon tissue (1). Focused shock waves result in a maximum pressure field that converges at a selected depth in the tendon tissue. Focussed ESWT can be generated by an electrohydraulic, electromagnetic, or piezoelectric method (2). The main difference between these three methods of focussed ESWT is the moment of shock wave formation. While these methods are different, it is very questionable whether it results in different local and clinical effects (3). These devices can generate different pressure distribution profiles, energy density and the total energy at the focal point in the tendon. Furthermore, the number of shock waves applied per session, frequency of shock waves applied, number and interval of sessions and applied principal modality (radial versus focussed shock waves) can vary.

The mechanisms of ESWT are not fully understood, but it aims to cause interstitial and extracellular responses leading to tissue regeneration (4). Fundamental research has postulated a number of theories on the biological effects of ESWT in tendinopathy. These theories can be roughly divided into (1) pain relief, (2) tissue regeneration and (3) destruction of calcification (1). Pain relief can be achieved in multiple ways. Local overstimulation of the tendon nerves could lead to a diminished transmission of nociceptive signals (5). This could be achieved by influencing expression of neuropeptides in the dorsal root ganglion. Possible regenerative effects of ESWT have been shown in multiple in vitro studies through increased expression of pro-inflammatory cytokines (IL-6 and IL-10), growth factors (TGF-β, VEGF, IGF-1) and of type I collagen (6). Destruction of calcification is shown in in-vitro studies, but it is unknown whether it occurs in the clinical setting and whether it is associated with clinical outcome (1).

CLINICAL EVIDENCE

Extracorporeal shockwave therapy as a treatment for lower limb tendinopathies has shown different outcomes in multiple recent systematic reviews, with inconsistent conclusions and recommendations (7-9). This difference and inconsistency may be caused by publication bias, language bias, not adhering to the guidelines for performing systematic reviews, an arbitrary selection of quality assessment tools and differences in inclusion and exclusion criteria (7). Taking into account these factors, there is moderate evidence against ESWT in patellar tendinopathy. There is moderate evidence for ESWT in proximal hamstring tendinopathy and low evidence for ESWT as an effective intervention for Achilles tendinopathy and gluteal tendinopathy (7). It is still unclear which ESWT parameters influence these results and whether there are specific subgroups of patients who have a better treatment response.

RECOMMENDATION

ESWT may be considered for proximal hamstring tendinopathy, gluteal tendinopathy and Achilles tendinopathy, and is not recommended for patellar tendinopathy. Athletes should expect effectiveness after 3-5 sessions. If there is no response after 5 sessions, it is recommended to stop this adjunct therapy as no further improvement is expected. Safe treatment protocols for ESWT settings have been developed (10).

MEDICATION

NON-STEROIDAL ANTI-INFLAMMATORY DRUGS (NSAIDS)

There is a debate about the presence of an inflammatory response in patients with tendinopathy (11). Recent data support an inflammatory component to tendinopathy, which suggests a role for non-steroidal anti-inflammatory drugs (NSAIDs). These NSAIDs can be prescribed in a topical or oral application. Supressing cyclo-oxygenase (COX) activity and reducing synthesis of pro-inflammatory prostaglandins are the main actions of NSAIDs (12). However, a recent randomised in vivo study demonstrated that prescription of ibuprofen has no effect on gene expression of collagen (type I and III) and growth factors in human tendinopathic tendon in vivo (13). Another mechanism of action is the interference of NSAIDs with cell proliferation. These agents decrease cell proliferation in preclinical work, which might be beneficial in the reactive stage of tendinopathy (14). A recent pre-clinical study shows that NSAIDs prevent the restoration of extracellular matrix composition. This suggests that NSAIDs might not be beneficial when structural tendon changes are present (degenerative tendinopathy).

CLINICAL EVIDENCE

Only four randomised controlled trials (RCTs) on NSAIDs in Achilles tendinopathy are available. In one study of 70 patients with up to 6 months symptomatic Achilles tendinopathy, oral NSAIDs were no more effective than placebo (15). In contrast, another RCT showed a significant effect of a topical NSAIDs versus placebo in 227 patients with Achilles tendinopathy (16). However, several quality aspects were not clearly described in this latter study. One large study on the effectiveness of NSAIDs in overuse injuries reported a post-hoc subgroup analysis of 40 people with acute Achilles tendinopathy (less than 48 hours duration) and found favourable results for oral NSAIDs over placebo, but unfairly excluded the 13% of participants who failed to complete the 10-day treatment (17). A more recent blinded RCT included male athletes with reactive Achilles tendinopathy who either received etoricoxib 120 mg oral once daily (n=28) or diclofenac 100 mg oral once daily (n=28) (18). There was no between group difference in pain scores, but patients receiving etoricoxib had fewer side effects (0% versus 14%). Additionally, a meta-analysis demonstrated that topical NSAIDs are efficacious for decreasing acute musculoskeletal pain and not associated with the harms of oral NSAIDs (e.g. gastrointestinal or renal disorders) (19).

RECOMMENDATION

Not recommended.

INJECTION THERAPY

Various types of injection therapies are available for athletes with lower extremity tendinopathy. The main parameters that vary are the injection technique, location of the injection and the content.

CORTICOSTEROID INJECTION

Local corticosteroid injections are widely administered for lower extremity tendinopathies. The mechanisms of action from injection is only partially clarified. Corticosteroids can enter the cell and modify gene expression by either activating anti-inflammatory molecules and repressing pro-inflammatory molecules (20). Furthermore, dexamethasone has shown to temporarily decrease the neuropeptide substance P (21). This may partly explain the effect of corticosteroids on pain reduction. However corticosteroids are associated with complications. Potentially harmful effects from preclinical studies include a further deterioration of the collagen organisation, a decrease in collagen production and an arrest in tenocyte proliferation (20). These processes may predispose the tendon to a poor long-term outcome or tendon rupture.

CLINICAL EVIDENCE

There are a number of randomised trials evaluating effectiveness of a corticosteroid injection in lower extremity tendinopathies. These have mainly been performed in positional tendons and less in tendons facilitating energy storage and release.

For gluteal tendinopathy, the short-term effect of education plus exercise was similar to corticosteroid injection, but education plus exercise was more effective on the long term (22). }. A placebo-controlled trial on effectiveness of a local corticosteroid injection for gluteal tendinopathy showed no significant between-group differences in pain scores for this condition at 3 and 6-months follow-up (23). One small randomised trial on effectiveness of a corticosteroid injection versus exercise therapy was performed in patellar tendinopathy (24). This trial showed a similar improvement in symptoms within both groups, but the positive effect was only maintained in the exercise group at 6 months follow-up. Another small randomised controlled trial on effectiveness of a peritendinous corticosteroid injection in Achilles tendinopathy showed no statistical or clinically relevant between-group differences at 3 months follow-up (25). These results are in line with a large systematic review, showing a promising short-term but poor long-term outcome after corticosteroid injections in patients with tendinopathy (26). Contrary to popular belief, a corticosteroid injection was not associated with more side effects than placebo or control treatments. This may be because it has been investigated in positional tendons more often and these are less prone to complete rupture, or it might be that a single injection is not harmful in the clinical setting. A large case-control study showed an increased risk for Achilles tendon rupture with increasing number of local or systemic corticosteroid injections (odds ratio of 5.3 after 3 or more injections) (27).

RECOMMENDATION

Not recommended.

SCLEROTHERAPY

Sclerosing injections are widely used for treating varicose veins. For the treatment of tendinopathy, sclerotherapy aims to decrease the localised neovascularisation and accompanied neural ingrowth, which is associated with degenerative tendinopathy. Polidocanol (typically 5 mg/ml) is the most frequently applied sclerosing agent for tendinopathies (28, 29). It is injected under guidance of Doppler ultrasonography at the peritendinous location where the vessels enter the tendon from the fat pad. This imaging method also aids in evaluating whether the treatment has been successful (i.e. disappearance of Doppler flow). Polidocanol causes thrombosis of the small blood vessels, which also occurs when injected extravascularly (30). Sclerosis of the adjacent nerves may also occur, either directly (by destruction) or indirectly (by ischaemia), and this is thought to cause an immediate reduction in pain. However, other observations indicate that sclerosing injections resulted in an increased intratendinous vascularity in the first 3 weeks after the injection (31). The decreased Doppler flow which is observed directly after sclerosing injections is thus only temporary and providing more uncertainty regarding the mechanisms of this treatment (32).

CLINICAL EVIDENCE

The first pilot case-control study and small RCT using polidocanol as a treatment for lower extremity tendinopathies were promising (28, 33, 34), with remaining good results at long term (2 years) follow-up (35). However, these promising results could not be replicated in other settings and/or larger prospective case series and RCTs (36-38). In a small randomised clinical trial, a polidocanol injection was compared to arthroscopic shaving in active patients with patellar tendinopathy (39). The surgically treated patients had less pain during sports and were more satisfied compared with the patients who received a polidocanol injection. In another small randomised clinical trial, patients with Achilles tendinopathy were either treated with polidocanol or lidocaine (40). There were no clinically relevant between-group differences in patient-reported outcomes at 3 and 6-month follow-up. These results show that polidocanol is not an effective treatment for lower extremity tendinopathies. This may partly be explained by the difficult injection process that may limit capacity to adequately infiltrate the vessel.

RECOMMENDATION

Not recommended.

HIGH-VOLUME INJECTION

The theory behind a high-volume injection is also based on obliterating the neovascularisation and associated

neural ingrowth (41). The neovascularisation is thought to arise from the fat pad in proximity to the tendon (42). Accordingly, these injections have mainly been investigated in tendons with an adjacent fat pad (e.g. Achilles and patellar tendon). Using a high volume of saline, the fat pad and the tendon hypothetically can be separated, which affects the neovascularisation and accompanied nerve ingrowth.

CLINICAL EVIDENCE

Numerous case series have been performed on the effectiveness of a high-volume injection in patients with Achilles and patellar tendinopathy (43-45). There were large improvements in symptoms within several weeks after this injection in all case series. Importantly, corticosteroids were included in the mixture, which may explain the short-term pain effect in these studies.

This was confirmed in a more recent randomised study, where a high-volume injection with corticosteroids were more effective than a placebo injection after 3 and 6 months (46). A recent small randomised controlled trial in patients with Achilles tendinopathy, a high-volume injection with corticosteroids was compared to a high-volume injection without corticosteroids (47). The mixture with corticosteroids showed a better short-term (6 weeks and 3 months) improvement than the mixture without corticosteroids, but not on the intermediate term (6 months). The long-term effect and safety profile of a high-volume injection (with or without corticosteroids) is currently unknown.

Based on these studies it is questionable whether treatment effect is due to the high volume or as a result of the short-term corticosteroid effect. A retrospective controlled study showed that a high-volume injection of 50 mL was superior to a lower volume injection of 30 mL in patients with Achilles tendinopathy (48). In this study, the injection consisted of saline and an anaesthetic but there were no corticosteroids in the mixture, indicating that the high-volume itself caused the effect. Contrary, in a recent blinded placebo-controlled randomised study, there was no clinically relevant between-group difference between a high (50 mL) and low (2 mL) volume injection (49). The patients were successfully blinded and there was no corticosteroid within the mixture in this trial. This finding calls into question the role of the high volume in this type of injection and suggests that the reported short-term effects are probably induced by the corticosteroids in the mixture. There is moderate evidence for short-term efficacy for high-volume injections with corticosteroids in Achilles tendinopathy. In absence of long term (>1 year) follow-up, this therapy is not recommended in elite athletes.

RECOMMENDATION

Not recommended.

PROLOTHERAPY

Prolotherapy refers to an several injections over time with a relatively small volume of an irritant solution at sites of tendon pain (50). The most frequently used solutions are glucose and dextrose; concentrations, volumes and frequency of injections vary in the literature. The injections are performed at tender points in the subcutaneous tissues adjacent to the affected tendon. Prolotherapy is hypothesised to cause local irritation, subsequent inflammation, tissue healing, and strengthening of the affected tendon. Indeed, a previous preclinical study showed an improved withstand to maximum load of rat patellar tendons after prolotherapy (51). The exact biological effect of prolotherapy remains unknown and preclinical studies are scarce.

CLINICAL EVIDENCE

Numerous case series showed positive effects of 4-5 local dextrose injections in patients with Achilles and patellar tendinopathy (52-54). One small three-armed RCT compared (1) prolotherapy with eccentric exercise therapy to (2) prolotherapy without eccentric exercises and (3) eccentric exercise therapy only in physically active patients with chronic Achilles tendinopathy (55). Improvements at, 3, 6 and 12 months in patient-reported outcomes were larger in the groups treated with prolotherapy with eccentric exercise therapy compared to the eccentric exercise only group. However, the proportions of patients achieving clinically important improvements were similar for all groups. Consequently, there is limited evidence that prolotherapy injections are a safe and effective treatment for Achilles tendinopathy, but it is uncertain whether it results in patient-important changes (56).

RECOMMENDATION

Prolotherapy may be considered for patients with Achilles tendinopathy in case of non-response to adequate loading therapy. Treatment effect should be experienced after a maximum of 5 local injections after which this therapy should be stopped.

PLATELET-RICH PLASMA (PRP) INJECTION

It is hypothesised that injections with platelet-rich plasma (PRP) result in tissue regeneration. With the use of cell-separating systems, platelets can be isolated from the patients' whole blood. Clotting of these platelet leads to degranulation and a subsequent release of many cytokines and growth factors (57). Transforming growth factor-β (TGF-β), vascular-derived endothelial growth factor (VEGF), and insulin-like growth factor (IGF) are frequently mentioned growth factors (58). There is a classification system to define the different forms of PRP, distinguishing different forms based on presence of leukocytes and type of application (solution or gel) (59). Most preclinical studies demonstrate that PRP results in increased cell proliferation and growth factor concentration (mainly the angiogenetic VEGF). An increased histological vascular network has been reported in multiple studies, but negative studies are also present and one study even showed reduced vascularity. Results are conflicting on formation of a histological vascular network, collagen production and organisation and the inflammatory response (57). As tendinopathy is characterised by increased cell activity and neovascularisation, it is at least questionable whether the above-mentioned effects of PRP are necessary for the treatment of tendinopathies.

CLINICAL EVIDENCE

Clinical applications of PRP have become very popular. The initial case series all showed an improvement over time after injecting PRP in lower extremity tendinopathies (60, 61). In Achilles tendinopathy, two blinded and one non-blinded randomised placebo-controlled trials did not show an effect on patient-reported outcomes of a PRP injection on the intermediate and long term (62-65). A more recent randomised study showed an improved patient-reported outcome after 4 peritendinous PRP injections at 2-week intervals compared to placebo (46). The methodology of this trial seems robust, although the physician delivering the injection could not have been blinded as the location of these different injections was different.

In patellar tendinopathy, only one high-level three-armed RCT has been published (66). Combined with exercise therapy, both a single leucocyte-poor and a single leukocyte-rich PRP injection were no more effective than a saline placebo injection. A very small randomised trial compared the effectiveness of a PRP injection to dry needling in patients with patellar tendinopathy (67). The physician who delivered the treatment was not blinded. In this trial, the PRP group improved significantly more on patient-reported outcomes than the dry needling group at 3 months, but the between-group difference was not present anymore after 6 months. The number of systematic reviews on PRP treatment in Achilles tendinopathy exceeds the number of randomised trials. Overall, the quality of these systematic reviews is poor, with a risk of bias and conflicting conclusions (68). The two systematic reviews that are methodologically sound and (potentially) clinically useful concluded presence of insufficient evidence to support the use of PRP (69, 70).

RECOMMENDATION

Not recommended.

CELL-BASED INJECTION

The treatment of chronic tendinopathy would be greatly advanced with effective regenerative cell therapies. In vitro studies with different cell line therapies have shown the capability to enhance tendon tissue regeneration (71). The theoretical advantages are that the cells could contribute themselves to the healing process, because of their potency to differentiate and generate new tissue. In addition, tendon cells have the ability to produce growth factors and anti-inflammatory cytokines for a prolonged period, in contrast to single injection of growth factors. In contrast to mesenchymal stem cells, homologous (progenitor) tenocytes can be harvested relatively easily from another tendon of the patient using a biopsy.

CLINICAL EVIDENCE

Previous clinical evidence of the effectiveness of autologous cell therapy in lower extremity tendinopathies is mainly limited to case series (72, 73). Case series in 8-12 patients with patellar tendinopathy and gluteal tendinopathy, treated with autologous tenocytes or bone marrow derived cells, showed functional and structural improvement which remained at 2-5 years follow-up.

A randomised trial in 46 patients with patellar tendinopathy compared a skin-derived fibroblasts injection with a plasma injection (74). A statistically significant between-group difference in patient-reported outcome was found, favouring cell therapy. Several shortcomings of this trial included an improper power calculation, lack of minimum reporting standards at baseline, no adjustments for potentially influential baseline variables, unclear randomisation process, analysis of bilateral cases and absence of trial registration. The publication of a second randomised trial on skin derived fibroblasts in chronic Achilles tendinopathy from the same study group was retracted, due to problems with ethical approval (75). As robust studies are lacking, the use of cell therapy for tendinopathy is currently not advised, which is in line with the conclusions of a recent systematic review (76).

RECOMMENDATION

Not recommended.

TAKE HOME MESSAGE

A myriad of adjunct treatments have been proposed for athletes with lower extremity tendinopathies. Most trials performed in this field are flawed by substantial risk of bias, short follow-up periods and the small sample sizes result in large uncertainty in the comparative estimates of treatments (77). There is moderate to low level evidence for effectiveness of extracorporeal shockwave therapy in proximal hamstring tendinopathy, gluteal tendinopathy and Achilles tendinopathy. There is low level evidence for prolotherapy in patients with Achilles tendinopathy. There is insufficient evidence to support other adjunct treatments.

Barça Way:

- There are a plethora of treatments and therapeutic modalities proposed to effectively treat tendinopathy. Many of them have low or no scientific evidence, and research on their effectiveness and utility in the sports setting is needed. In FC Barcelona, the principal approach to managing tendinopathies is the manipulation of load and exercise interventions. Extracorporeal shockwave therapy and platelets rich plasma (PRP) are added to target poorly controlled symptoms and to contribute to the general management. The various methods of PRP preparation complicates their use and we are actively conducting research in these adjuncts to enhance our knowledge about these techniques.
- Shockwave therapy is used every second day, ideally following a training session and before a rest day. Radial shockwave of 1500 impacts, is used to decrease tension in the quadriceps and calf muscle and an additional 1500 impacts of focal shockwave is used on the affected tendon.
- We have found generally positive results with PRP injections in tendinopathy. We preferably use it before a rest day (a training session is cancelled if required). Ultrasound-guided injection into the pathology and the tendon itself is done 3-5 times, 4-8 ml re injected every 7-15 days.
- We have recently reported (78) the six month results of a prospective, double-blinded, randomized, phase 1/2 single-center clinical study investigating the effects of ultrasound-guided intratendinous and peritendinous injections of autologous expanded bone marrow mesenchymal stem cells (BM-MSCs) or leukocyte-poor platelet-rich plasma (Lp-PRP) on clinical outcomes in athletes with patellar tendinopathy. In this unique study we showed that injections of BM-MSC or Lp-PRP together with rehabilitation in chronic refractory patellar tendinopathy is effective in reducing pain and improving activity levels in athletes, and at six months, athletes who received BM-MSC treatment demonstrated greater improvement in tendon structure compared with those who received Lp-PRP. These are very promising results, but we understand that we need more studies and more time to apply this cellular treatment.

Summary:

- A myriad of adjunct treatments have been proposed for lower extremity tendinopathies, but trials evaluating these therapies are generally at high risk of bias.
- Extracorporeal shock wave therapy may be considered in proximal hamstring, gluteal or Achilles tendinopathy, but is not recommended for patellar tendinopathy. If no significant improvement is observed within five sessions treatment should be ceased.
- There is mixed evidence regarding the efficacy of NSAID use in tendinopathy. At this stage there is insufficient evidence to recommend their use.
- Local corticosteroid injections are widely used for lower extremity tendinopathies; however, their mechanism of action is unclear. Corticosteroid injections have been associated with complications including decreased collagen organisation and production and decreased tenocyte proliferation, which may pre-dispose the tendon to poorer long-term outcomes.
- Sclerotherapy has not been shown to be an effective treatment for lower extremity tendinopathies.
- High volume injections have been proposed to affect neurovascularisation of pathological tendons, however, in the absence of long-term studies this therapy is not recommended.
- Prolotherapy refers to the injection of a relatively small volume of an irritant solution at the site of painful tendon insertion. It is theorised to cause local irritation, subsequent inflammation and consequent tissue healing. It may be considered in the case of Achilles tendinopathy, but only if rehabilitation using an adequate loading program is not successful. A beneficial treatment effect should be observed after a maximum of five injections, otherwise treatment should be ceased.
- Platelet-rich plasma injections are hypothesised to assist in tissue regeneration, however insufficient evidence is available to demonstrate their effectiveness.

Clinical Implications:

- There is moderate to low level evidence for effectiveness of extracorporeal shockwave therapy in proximal hamstring tendinopathy, gluteal tendinopathy and Achilles tendinopathy. There is low level evidence for prolotherapy in patients with Achilles tendinopathy. There is insufficient evidence to support other adjunct treatments.
- Adjunct treatments are not a substitute for a well-constructed, tendon loading program, and their use should be carefully considered.

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— Lasse Lempainen and Håkan Alfredson

2.5. TENDON SURGERY

Conservative management is the first line treatment for tendinopathy. Successful treatment of tendinopathy relies on accurate initial diagnosis and surgical intervention is influenced by anatomical and tissue healing factors. Underestimation of the severity of the injury and diagnostic difficulties can delay optimal treatment.

The location of the injury in the musculotendinous unit; at the tendon insertion, in the tendon or in the myotendinous junction, is critical as the anatomical location of the injury impacts on treatment. For example, isolated complete adductor longus tendon rupture with minor retraction can heal well conservatively because the ruptured area is surrounded by intact fascia and is supported by the other adductor muscle tendon structures. Understanding the healing potential of tissues in these anatomical locations is important. For example, a distal Achilles tendon rupture or avulsion from the calcaneal bone requires surgical treatment because this injury heals poorly if treated conservatively.

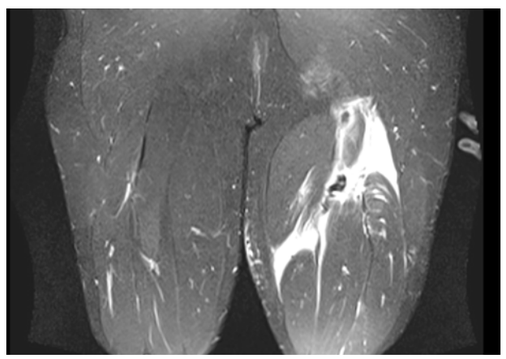
TENDINOPATHY

Tendinopathy is treated surgically when conservative management has failed. In the Achilles and patellar tendinopathy there is seldom need for intra-tendinous surgery, surgery can be effective if done outside the tendon using mini-invasive, ultrasound and Doppler-guided procedures. This extra-tendinous surgery does not require immobilisation and allows early weight-bearing followed by a short rehabilitation period before returning to full training and playing. The length of the rehabilitation period depends on the tendon affected, the capacity of the tendon, muscle, and kinetic chain (dependent on the duration of symptoms and unloading) and can range from 1 – 6 months.

TENDON RUPTURES

Complete tendon ruptures (Figure 1) in athletes are treated with surgery as soon as possible following injury. Re-insertion of the ruptured tendon or a direct tendon suture may be used, sometimes combined with reinforcement using fascia or other tendons such as the plantaris tendon. Restoring optimal tendon length is an essential goal of surgery. Total or partial immobilisation is followed by a period of structured rehabilitation. Gradual loading of the repaired tendon is essential to ensure that the tendon does not heal in a lengthened position. The time needed for healing and return to full training and play varies depending on the tendon involved. For example, an Achilles rupture repair is still healing up to 12 months, but most uncomplicated surgically repaired ruptures tolerate full training and playing after 4-6 months.

Most Achilles tendon ruptures are treated surgically in a football population because of the better post-operative strength level and end results. Patellar tendon ruptures must be surgically repaired. Isolated single tendon hamstring ruptures are relatively rare but can require surgical repair. All complete proximal hamstring avulsions should be treated surgically in high level athletes.



Partial tendon ruptures are usually treated conservatively. Some partial tendon ruptures can cause excessive scarring and tightness leading to reduced activity level and recurrent injuries and surgical treatment can be indicated. The time to return to full training and playing varies for different tendons.

Figure 1. Complete three tendon proximal hamstring rupture.
<

ACHILLES TENDON

Conservative management is unsuccessful in 24–45.5% of patients with Achilles tendinopathy (1, 2), although these outcomes are not known in (elite) football players. There are no absolute indications for surgical treatment in Achilles tendinopathy but surgery can be a good option if Achilles tendinopathy makes training impossible or symptoms reoccur after proper conservative treatment (3). If surgery is considered, shared decision-making is imperative about the type of surgery, the rehabilitation times, the likelihood for recovery and return to sport, and the best time for surgery.

SURGICAL TREATMENT OF MIDPORTION ACHILLES TENDINOPATHY

The traditional surgical treatment for midportion Achilles tendinopathy has been intra-tendinous revision followed by immobilisation and a long rehabilitation period (4-6 months). Extra-tendinous surgical treatment of Achilles tendinopathy is possible as midportion Achilles tendinopathy has multiple nerves located ventrally outside the tendon that are associated with neovascularisation (4). The use of ultrasound and Doppler examination can identify these vessels and guide a surgical procedure performed under local anaesthesia where the neural and vascular supply is disrupted outside the tendon. The rehabilitation is immediate weight bearing and a 4–6-week rehabilitation period. Both early return to sport (5), and long-term clinical results have been shown to be good (6).

ACHILLES TENDINOPATHY WITH PLANTARIS TENDON INVOLVEMENT

Some people with midportion Achilles tendinopathy have plantaris tendon involvement (7). The plantaris tendon can have different positions in relation to the medial side of the Achilles tendon, sometimes causing compression or friction with the Achilles tendon (8, 9). This occurs during activity that loads the tendon through range and causes sharp pain on the medial side of the Achilles. Rehabilitation with eccentric calf muscle training can worsen the medial Achilles tendon pain.

It can be challenging to identify the plantaris tendon on imaging as it can be fused with the Achilles (Figure 2), but careful ultrasound scanning can help identify its presence. If the plantaris tendon impinges on the Achilles then the treatment is surgical removal, together with the surgical procedure as per Achilles tendinopathy is performed (6). The rehabilitation period is the same as for the Achilles tendinopathy procedure alone.

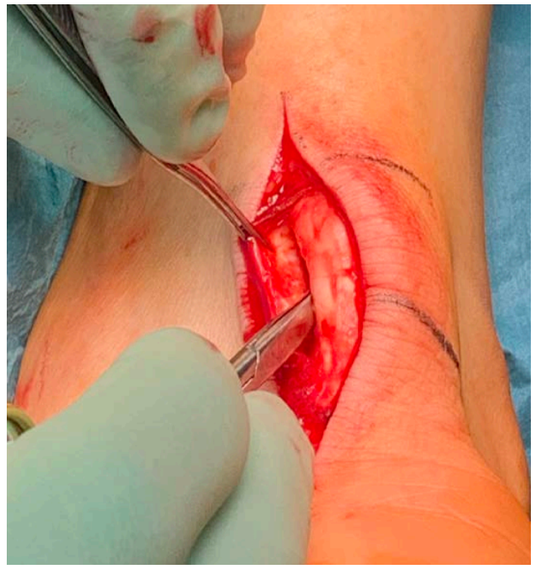


Figure 2. Adherent plantaris tendon.
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PLANTARIS TENDON RELATED PAIN

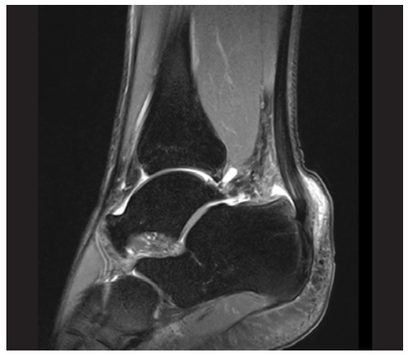
Load related pain from the plantaris tendon alone, without co-existing Achilles tendinopathy can occur (10). Sharp, medial side located Achilles tendon pain is present during rapid acceleration and sprinting, but the Achilles is normal on imaging. Dynamic ultrasound examination in the region for pain gives the diagnosis, and treatment is surgical removal under local anaesthesia (10).

CHRONIC PERITENDINOPATHY

Chronic painful constrictive peritendinopathy is a rare condition, and can occur after multiple episodes of acute peritendinopathy, or after trauma like a kick on the Achilles. Typically, there is tenderness and load related pain superficially and a feeling of pronounced stiffness that does not respond to stretching. Ultrasound is used for diagnosis, and treatment is surgical local peritendon removal, followed by a 6–10-week rehabilitation period. Limited loading during the first 4-6 weeks allows for formation of a new peritendon-like tissue.

INSERTIONAL ACHILLES TENDINOPATHY

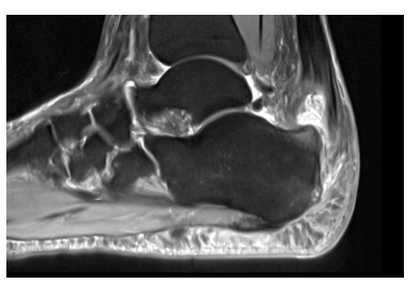
Evaluating insertional Achilles tendinopathy is complicated because there are multiple structures involved (Figures 3 and 4). The distal Achilles tendon, retrocalcaneal bursa, upper edge of the calcaneus, subcutaneous bursa and the distal part of the plantaris tendon can cause pain in this condition. The subcutaneous bursa is the most highly innervated tissue in the region (11).



^ **Figure 3.** Insertional Achilles tendinopathy with bony prominence and bursa involvement.

The use of diagnostic local anesthetic injections can guide surgical treatment. If pain completely disappears during provocative exercises after a local anaesthetic injection in the subcutaneous bursa alone, surgical removal of the bursa, despite other pathology on imaging, can resolve symptoms (12). This treatment is combined with a 6–8-week rehabilitation period and allows for a faster return to sport than more extensive surgery. Isolated tenderness and pain medial at the insertion may indicate plantaris tendon enthesopathy, after surgically removal a 6-week rehabilitation period is required. Some people have pain from several tissues; distal Achilles, both bursae, upper calcaneus, intra-tendinous calcification and plantaris insertion, and surgical treatment should then address all these tissues (13, 14). After that type of procedure a minimum 4 month rehabilitation period is required before return to sport (15).

PARTIAL ACHILLES TENDON RUPTURE



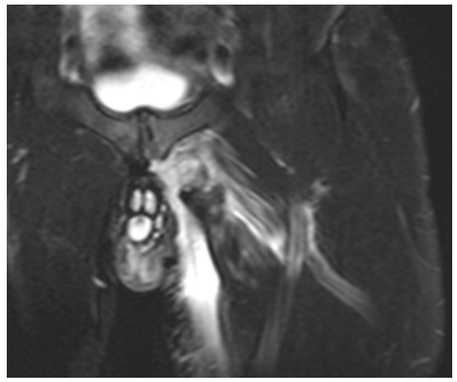
^ **Figure 4.** Insertional Achilles tendinopathy with partial tear requiring operative treatment.

Partial midportion ruptures in the Achilles tendon most often respond well with conservative management, using a heel lift and avoiding stretching for 10-12 weeks (Figure 4) (16). Partial rupture after intra-tendinous injections, especially corticosteroid injections, do poorly with conservative management, and surgical treatment with intra-tendinous revision of necrotic tendon regions is required. A 12–16-week rehabilitation period is required before return to Achilles loading sports.

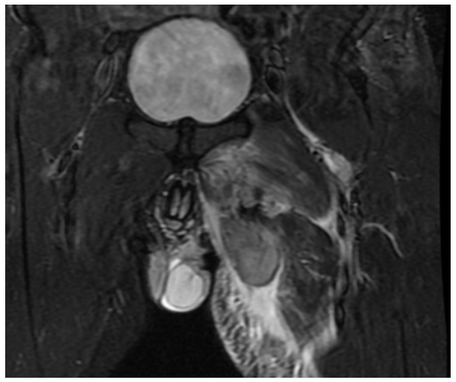
ADDUCTOR TENDON

Adductor tendon injuries are common in professional football players (17). Most of them can be treated conservatively but some of them need operative treatment to return to play. Adductor injuries can reoccur after conservative treatment and even though rehabilitation has recently advanced, there is no consensus on treatment of these more severe and recurrent adductor injuries (18).

Surgical indications for acute adductor injuries are rare. Surgery should be considered in severe, unstable adductor longus tendon injuries when there is a clear defect and tendon retraction from the pubic bone (19) (Figure 5 and 6). Complex injuries such as PLAC (pyramidalis + anterior pubic ligament + adductor longus rupture) injuries requires surgery in high level athletes (Figure 7) (19). In those cases, evaluation of the adductor strength is used as part of the decision making for operative treatment.

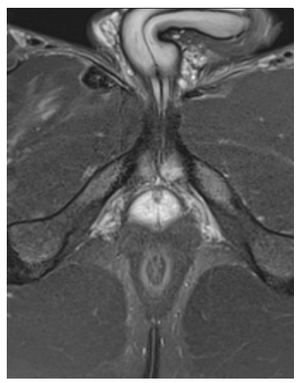


^ **Figure 5.** Unstable and retracted adductor longus tendon rupture.

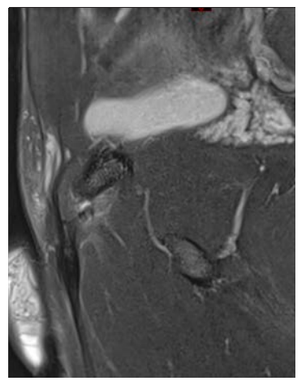


^ **Figure 6.** Severe adductor longus tendon rupture with concomitant structure lesions.

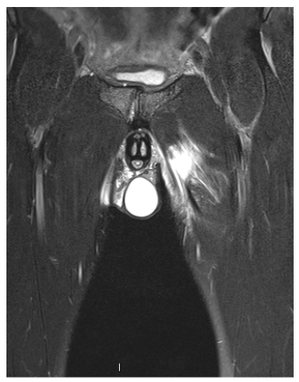
Following surgical repair of complete proximal adductor longus tendon avulsions (+ PLAC injuries), players can begin running and performing controlled drills with a ball (i.e., "return to field") after 8-12 weeks, and most have returned to optimal performance level after 4 to 5 months. In chronic adductor tendon related pain and surgical treatment, return to play can be even faster (Figures 7A - C).



^ **Figure 7a.** Chronic adductor longus tendon related pain requiring surgical management.



^ **Figure 7b.** Sagittal view showing thickening of adductor longus.



^ **Figure 7c.** Chronic adductor longus tendon tear at musculotendinous junction, coronal view MRI

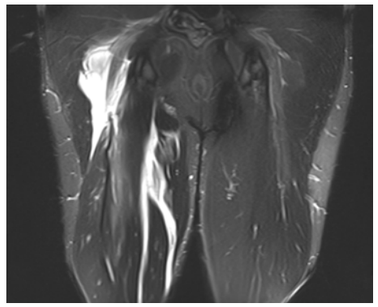
HAMSTRING TENDON

The goal of hamstring surgery is to restore the anatomy of the injured structure to allow for rapid recovery and a safe return to sports with low recurrence. Hamstring injuries among high-level athletes instead should be considered not just as hamstring injuries but as separate tendon injuries; biceps femoris, semimembranosus or semitendinosus injuries (20). Complete single tendon avulsions of one of these or a combined tendon injury in high demand athletes can result in a substantial loss of function and sporting ability (21-23). These single tendon injuries are in theory only partial hamstring injuries, but they are true complete tendon ruptures. The physician uses clinical findings (posterior thigh hematoma, pain, and decreased strength in hip extension / knee flexion) and MRI imaging to determine the severity of the hamstring injury.

INDICATIONS FOR EARLY SURGERY

COMPLETE PROXIMAL AND RETRACTED HAMSTRING (SINGLE) TENDON RUPTURE

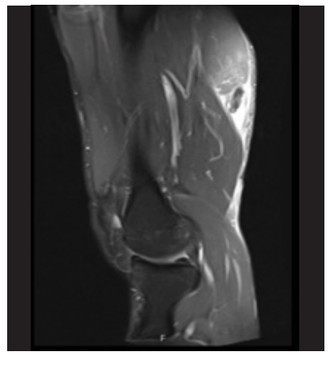
A proximal single tendon avulsion/rupture with a clear retraction should be treated surgically in athletes (Figure 8). If two or all three of the hamstring tendons are avulsed proximally surgery should be considered in all patients (23-25).



< **Figure 8.** Proximal BF free tendon rupture with a clear gap.

COMPLETE DISTAL AND RETRACTED HAMSTRING (SINGLE) TENDON RUPTURE

Distal tears of the hamstrings are rare (26). The biceps femoris (most common), semitendinosus, or the semimembranosus may rupture completely from the distal bony insertion or at the distal myotendinous junction (Figures 9 and 10). An acute complete distal hamstring tendon rupture with retraction should be repaired anatomically (27). Acute distal semitendinosus avulsion does not respond the same as harvesting the tendon for graft purposes (26, 28).

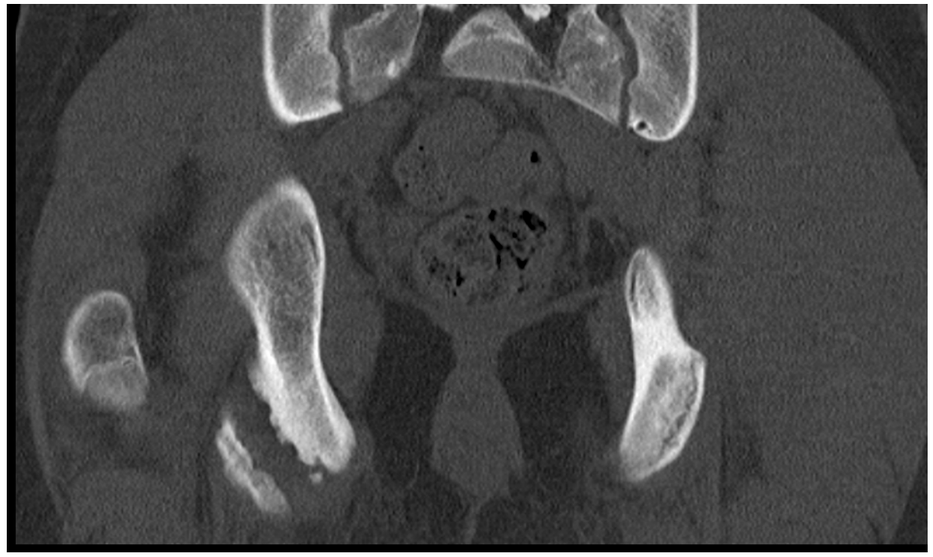


^ **Figure 9.** Distal ST rupture sagittal view mri.

^ **Figure 10.** Distal BF complete rupture.

APOPHYSEAL AVULSION

Apophyseal avulsions of the ischial tuberosity occur occasionally in adolescent athletes (Figure 11) (29). Surgical repair is traditionally recommended if the avulsed fragment is displaced by more than 10-15 mm.



^ **Figure 11.** Ischial tuberosity avulsion fracture (CT, coronal view)

INDICATIONS FOR DELAYED SURGERY

INCOMPLETE / RECURRENT HAMSTRING TEARS

Occasionally incomplete tears form scar tissue and adhesions and cause persistent symptoms and are not responsive to conservative treatment (30). This can occur in the proximal interface or in the proximal tendinous part or in the central tendon area. In proximal incomplete avulsions that remain symptomatic MRI may show fluid between the bone and the tendon indicating incomplete healing.

It has been suggested that paramuscular/central tendon injuries especially in the biceps femoris may have a higher risk of persistent symptoms and recurrent injury after conservative treatment (31). In these injuries there is often an incomplete tear of the paramuscular tendon typically in the area of 5 to 20 cm from the proximal origin. Often the muscle tissue is also torn away from the tendon. Surgery should be considered when symptoms remain after adequate conservative treatment or there are recurrences. Full continuity of the central tendon is restored with sutures and the attachment of the muscle to the tendon is reinforced. It is important to avoid overtightening of the repaired tendon. Scar tissue may be removed. Suture anchors may be used if the tear is located close to the bony origin.

HAMSTRING TENDINOPATHY / HAMSTRING SYNDROME

Surgical treatment may also be indicated in proximal hamstring tendinopathy or in chronic and/or recurrent hamstring injuries with symptoms of pain and tightness of the posterior thigh if conservative treatment has failed to change symptoms. These symptoms are called (post traumatic) hamstring syndrome or compartment syndrome (Figure 12)(23, 32). The surgical procedure may include excision of adhesions, fasciotomies, sciatic nerve liberation and liberation of the scarred tendons. After surgery, most of the athletes can return to the same level of sporting activity in a mean of 5-6 months.

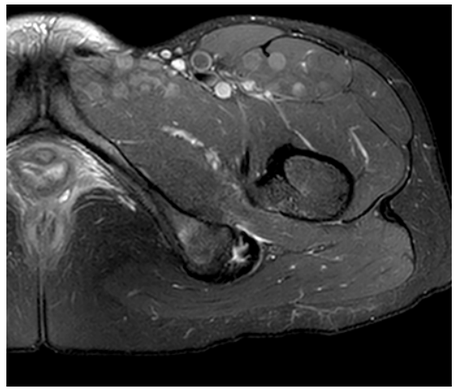


Figure 12. Proximal hamstring syndrome axial view MRI.
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PATELLAR TENDON

Surgery for patellar tendinopathy (Figure 13) is indicated when pain persistently disrupts training and playing and when adequate conservative management fails. Traditional surgical treatment for patellar tendinopathy involves open patellar tenotomy and excision of the pathological region of the tendon (33), and is associated with a prolonged recovery period and poor clinical outcomes (34).

Arthroscopic shaving does not involve surgery on the tendon, similar to that described in the Achilles tendon. Surgery is performed under ultrasound guidance on the region with neovascular ingrowth on the dorsal aspect of the tendon adjacent to the pathological region and minor resection of Hoffa's fat pad (33). Rehabilitation after surgery includes immediate weight bearing and a structured rehabilitation before return to play after 2-4 months (33). Longer term follow-up demonstrated a significant decrease in the anteroposterior thickness of the proximal patellar tendon, an improvement in tendon structure and a reduction in local blood flow (35). The average VAS score decreased from approximately 77 to 13, and 80% of patients reported being satisfied with the results of treatment (35).

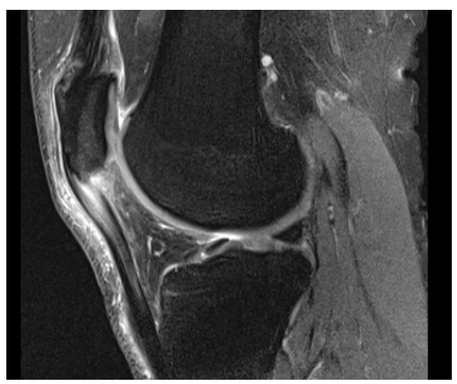

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Figure 13. Proximal patellar tendon tendinopathies.

PROXIMAL PATELLAR TENDINOPATHY AND PARTIAL RUPTURE

Tendinopathy in the proximal patellar tendon is occasionally seen together with a partial patellar tendon rupture, although the clinical importance of this is unknown. This is most often found among those who have been treated with intra-tendinous corticosteroid injections, and in patients where there is a sharp bony edge or spur in the patellar tip (36, 37). Ultrasound-guided arthroscopic shaving procedure is indicated, and rehabilitation includes immediate weight bearing, 4 weeks with light loading and then a structured rehabilitation before return to play after 3-4 months.

DISTAL PATELLAR TENDINOPATHY AND BURSITIS

The distal patellar tendon (tibial insertion) can be affected with tendinopathy, particularly in those that have previously had Osgood-Schlatter disease. There is often bursitis on the dorsal side of the tendon. When conservative management has failed surgical treatment including bursa removal, revision outside the dorsal and superficial side of the tendon, and often also removing of bone ossicles (sometimes inside the tendon) is performed. This surgery requires an 8-14-week rehabilitation before returning to sport.

COMPLETE PATELLAR TENDON RUPTURE

Complete patellar tendon rupture (Figure 14) among athletes is rare but it is a severe injury and early surgical treatment is always indicated.

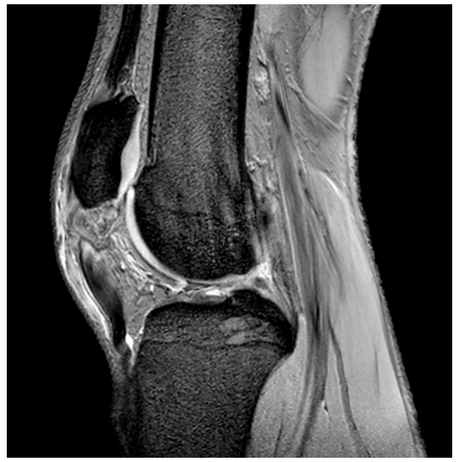

Figure 14. Complete patellar tendon rupture.
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Summary:

- Conservative management is the first line treatment for tendinopathy. Tendinopathy is treated surgically only when conservative management has failed.
- Successful treatment of tendinopathy relies on accurate initial diagnosis.
- Partial tendon ruptures are usually treated conservatively.
- The decision for surgical intervention is influenced by anatomical and tissue healing factors. The location of the injury in the musculotendinous unit; at the tendon insertion, in the tendon or in the myotendinous junction, is critical as the anatomical location of the injury and the healing potential of the tissues involved all influence whether surgical intervention is required.

Clinical Implications:

- In Achilles and patellar tendinopathy there is seldom need for intra-tendinous surgery. Surgery can be effective if performed outside the tendon using mini-invasive, ultrasound and Doppler-guided procedures. This extra-tendinous surgery does not require immobilization and allows for a shorter rehabilitation period before return to sport.
- Some people with midportion Achilles tendinopathy have plantaris tendon involvement. The location of the plantaris tendon medial to the Achilles tendon can cause friction or compression of the Achilles. It can be difficult to identify the plantaris tendon on imaging as it can be fused with the Achilles, but careful ultrasound scanning can help to identify its presence. Surgical removal may be considered if the tendon is impinging on the Achilles and the athlete is not responding to good conservative rehabilitation.
- The plantaris tendon may be a source of pain independent of Achilles tendon pathology. Sharp, medial side located Achilles tendon pain is present during rapid acceleration and sprinting, but the Achilles is normal on imaging. Dynamic ultrasound examination may be required for diagnosis.
- Surgical indications for acute adductor injuries are rare. Surgery should be considered in severe, unstable adductor longus tendon injuries when there is a clear defect and tendon retraction from the pubic bone.
- Surgery should be considered following rupture or avulsion of one or more of the proximal hamstring tendons in athletes, especially if multiple tendons are involved.
- Apophyseal avulsions of the ischial tuberosity occasionally occur in adolescent athletes. It is important that these injuries are accurately diagnosed as surgical management may be indicated if there is significant displacement of the avulsed fragment.

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3 Specific Section

— Karin Grävare Silbernagel, Haraldur Sigurdsson

3.1. ACHILLES TENDON INJURIES

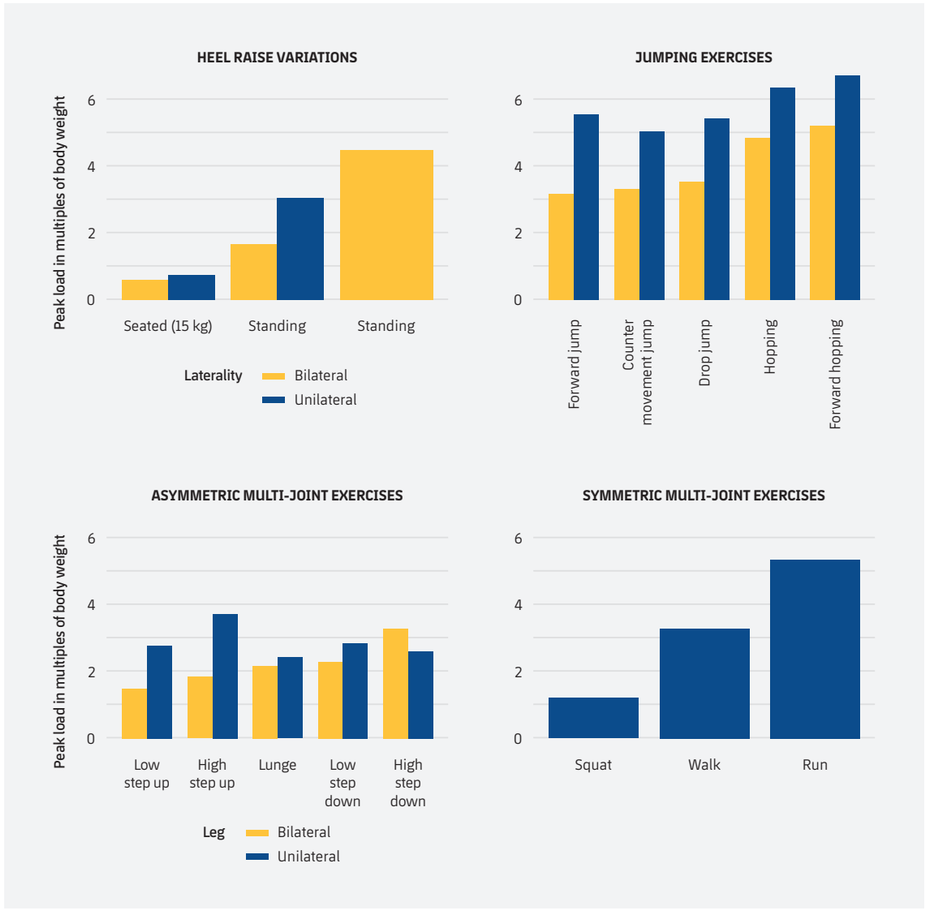
EPIDEMIOLOGY OF ACHILLES TENDON INJURIES IN FOOTBALL

There are two main types of Achilles tendon injuries that affect football players; Achilles tendinopathy, a painful overuse injury, and acute Achilles tendon rupture that is rarely preceded by symptoms. The most common is Achilles tendinopathy, a chronic overuse injury with a reported incidence of up to 0.3 injuries per 1000 hours (1). Given that Achilles tendinopathy may be present without time lost from training, the true prevalence of Achilles tendinopathy in football is likely higher (2). Achilles tendinopathy itself is composed of two clinical entities, mid-portion and insertional Achilles tendinopathy. Both can be slowly progressive after an insidious onset, but insertional Achilles tendinopathy tends to have a longer rehabilitation time. Peritendinopathy and bursitis are additional features that cause pain and can occur solely (peritendinopathy) or in combination (bursitis) with Achilles tendinopathy. Acute Achilles tendon ruptures represent 4% of Achilles tendon injuries (3).

ACHILLES TENDON LOADS

Understanding the load placed on the Achilles tendon during football and rehabilitative exercises is crucial to inform clinical decisions. The Achilles tendon is subject to two types of loads, tensile and compressive. During running, tensile load on the tendon can amount to 4-12 times body weight (4, 5). The tensile load is larger for faster, explosive movements such as jumping and sprinting where the tendon stores and releases energy. It is these movements that aggravate symptoms of Achilles tendinopathy, as well as cause ruptures.

The load on the Achilles tendon was recently estimated for a variety of tasks and exercises (Figure 1) (6). High force transmission through Achilles tendon are achieved either by slow movements with a heavy resistance (no energy storage and release loads), or from fast movements using body weight (energy storage and release loads) (6). The load placed on the Achilles tendon can therefore be finely regulated and can be progressed either by adding weight to a slow movement, or adding an exercise with energy storage and release (Figure 1) (6). The Achilles tendon load during running is also dependent on running speed, with faster speeds producing higher loads. With forefoot or midfoot strike patterns produce higher loads than rearfoot striking (2).



^ Figure 1. Achilles tendon loads for various exercises, adapted from Bater et al., 2020. Loads are represented as percentage of body weight. For the top row, colours denote unilateral or bilateral exercises. Bottom-left shows multi-joint exercises with asymmetric loads on the ankle joints. Bottom-right show multi-joint exercises with load distributed symmetrically on the ankle joints.

Although the exact aetiology of Achilles tendinopathy remains debated, animal models have demonstrated that repetitions of peak forces result in more tendon structural changes than fewer peaks with the same amount of total load (7). The number of repetitions performed at close to peak loads may be linked to the development of Achilles tendon pathology and tendinopathy. The higher the peak load, the fewer repetitions would be tolerable. In a football match, athletes perform multiple sprints and accelerations. This requires a high load capacity of the Achilles tendon to prevent rupture or the development of tendinopathy.

A second source of load on the Achilles tendon is compressive in nature. Compression between the midportion of the Achilles tendon and the plantaris tendon may contribute to midportion Achilles tendinopathy (8). The pressure is proposed to be increased by the combination of plantarflexion and hindfoot valgus (8), although the plantaris is stiffer than the Achilles and may also cause compression in dorsiflexion (9). Insertional Achilles tendinopathy develops on the deep aspect of the tendon where the superior posterior calcaneus compresses the tendon during dorsiflexion (Figure 2) (10). This compression is increased with greater ankle dorsiflexion and can be an issue when running on softer surfaces such as a wet grass field or with flat shoes.

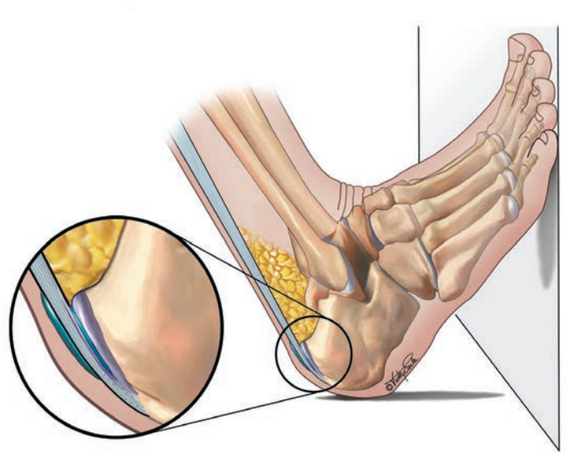


Figure 2. Compression in dorsiflexion.
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DIAGNOSIS AND TREATMENT OF ACHILLES TENDINOPATHY

CLINICAL PRESENTATION

The early primary symptoms of Achilles tendinopathy are pain during the warm-up and at the end of a training session (11). Morning stiffness or pain is a common complaint (12), especially the day following a game or intense activity. The onset is generally gradual and may start with a feeling of stiffness in the Achilles tendon prior to being warmed-up. It is important to be attentive to early symptoms and intervene before the injury becomes more severe.

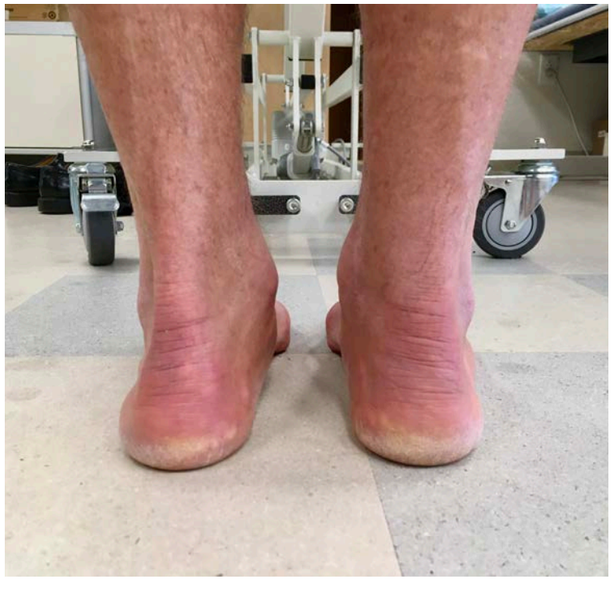

Figure 3. Enlarged Achilles tendon.
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DIAGNOSTIC TESTS AND CLINICAL EXAM TESTS

The term Achilles tendinopathy refers to chronic Achilles tendon pain and loss of function related to mechanical loading (13). The diagnosis is based on the person's symptoms and the clinical examination, aided by ultrasound or MRI imaging for differential diagnoses. There are several tests that will be positive in roughly half of patients (sensitivities ranging from 0.5 – 0.58 and specificity from 0.84 – 0.9) (14); pain with palpation, a positive Arc Sign, and a positive Royal London Hospital Test. The location of pain and the area of greatest tenderness with palpation can help differentiate between insertional and midportion Achilles tendinopathy. Insertional symptoms are also aggravated with loading at end range of dorsiflexion.

Pain provocation with loading has traditionally been assessed with tests such as pain on repeated hopping or jumping tasks. Using the incremental load progression (Figure 4), a clinician can have the athlete perform movements with sequentially increasing loads on the Achilles tendon. The movement where the onset of pain is noted, or where the pain exceeds 5/10 on a numerical rating scale can be considered the current load tolerance of the athlete.

INCREASING ACHILLES TENDON LOAD

^ Figure 4. Progressive loading test for the Achilles tendon. Localised Achilles tendon pain should increase in a dose dependent manner with each task.

PATIENT REPORTED OUTCOME MEASURES

Patient reported outcome measures (PROMs) may be used to assess the severity of symptoms. The Victorian Institute of Sports – Achilles (VISA-A) questionnaire (figure 5) (15) is an injury specific questionnaire useful for evaluating the severity of symptoms and response to treatment. This includes a question about morning pain and stiffness, this question can be used in isolation to monitor response to loading the previous day as the total VISA-A is resistant to short term change.

Athletes suffering painful conditions such as Achilles tendinopathy may develop a fear of movement – kinesiophobia. Education is an important intervention in those with fear of movement, emphasising that load is good for their tendon and that the chances of injuring it more with load are small. Kinesiophobia is associated with worse treatment outcomes in Achilles tendinopathy (16). As the main form of treatment involves loading exercises, evaluating and addressing kinesiophobia is important, the Tampa Scale for Kinesiophobia (17) or the modified version are commonly used questionnaires.

For professional, and aspiring professional athletes, the high-level functioning of their body is their livelihood. An injury represents a threat to that livelihood and can result in psychological consequences such as anxiety and depression (18). These can have widespread effects on treatment adherence and should therefore be screened for and then addressed. The American Medical Society for Sports Medicine Position Statement (19) has recommended screening instruments for signs of depression (20) and anxiety (21).

VISA-A

DATE __/__/____ INITIAL ASSESSMENT ☐ DISCHARGE ASSESSMENT ☐
NAME _____ SURNAME _____ AGE ___ WEIGHT ___ HEIGHT ___
SPORT _____ TEAM _____ PHYSICIAN _____

1. For how many minutes do you have stiffness in the Achilles region on first getting up?

100 mins ☐☐☐☐☐☐☐☐☐☐☐ 0 mins **POINTS** ☐
0 1 2 3 4 5 6 7 8 9 10

2. Once you are warmed up for the day, do you have pain when stretching the Achilles tendon fully over the edge of a step? (keeping knee straight)

Strong severe pain ☐☐☐☐☐☐☐☐☐☐☐ No pain **POINTS** ☐
0 1 2 3 4 5 6 7 8 9 10

3. After walking on flat ground for 30 minutes, do you have pain within the next 2 hours? (If unable to walk on flat ground for 30 minutes because of pain, score 0 for this question).

Strong severe pain ☐☐☐☐☐☐☐☐☐☐☐ No pain **POINTS** ☐
0 1 2 3 4 5 6 7 8 9 10

4. Do you have pain walking downstairs with normal gait cycle?

Strong severe pain ☐☐☐☐☐☐☐☐☐☐☐ No pain **POINTS** ☐
0 1 2 3 4 5 6 7 8 9 10

5. Do you have pain during or immediately after doing 10 (single leg) heel raises from a flat surface?

Strong severe pain ☐☐☐☐☐☐☐☐☐☐☐ No pain **POINTS** ☐
0 1 2 3 4 5 6 7 8 9 10

6. How many single leg hops can you do without pain?

0 ☐☐☐☐☐☐☐☐☐☐☐ 10 **POINTS** ☐
0 1 2 3 4 5 6 7 8 9 10

^ Figure 5. VISA-A.

7. Are you currently undertaking sport or other physical activity?

0	☐	Not at all
4	☐	Modified training ± modified competition
7	☐	Full training ± competition but not at the same level as when symptoms began
10	☐	Competing at the same or higher level when symptoms began

POINTS ☐

8. Please complete EITHER A, B or C in this question.

- If you have **no pain** while undertaking sport please complete **Q8a only.**
- If you have **pain while undertaking sport but it does not stop you** from completing the activity, please complete **Q8b only.**
- If you have **pain that stops you from completing sporting activities**, please complete **Q8c only.**

8a. If you have no pain while undertaking Achilles tendon loading sports, for how long can you train/practise?

0	☐	NIL
7	☐	1-10 mins
14	☐	11-20 mins
21	☐	21-30 mins
30	☐	> 30 mins

POINTS ☐

8b. If you have some pain while undertaking Achilles tendon loading sports, but it does not stop you from completing your training/practice, for how long can you train/practise?

0	☐	NIL
4	☐	1-10 mins
10	☐	11-20 mins
14	☐	21-30 mins
20	☐	> 30 mins

POINTS ☐

8c. If you have pain that stops you from completing your training/practice in Achilles tendon loading sports, for how long can you train/practise?

0	☐	NIL
4	☐	1-10 mins
10	☐	11-20 mins
14	☐	21-30 mins
20	☐	> 30 mins

POINTS ☐

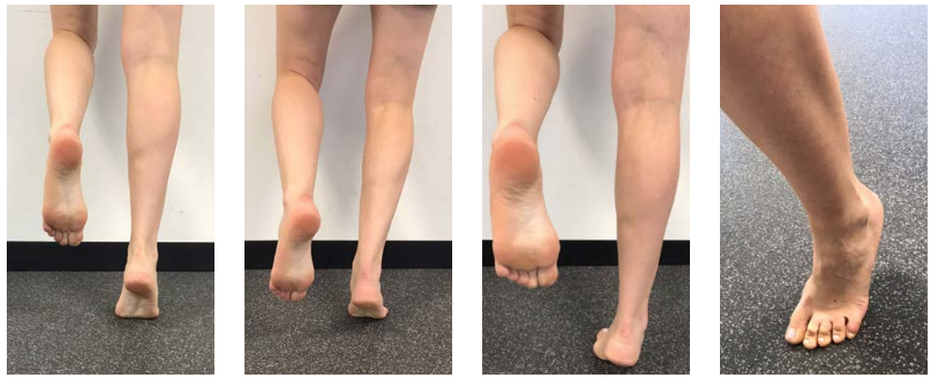
TOTAL SCORE: _____ /100 _____%

^ Figure 5. VISA-A.

FUNCTIONAL LIMITATIONS

A full evaluation will demonstrate the athlete's deficits in areas of range of motion, strength, endurance, functional tests such as jumping, and sport specific tasks. Individuals with Achilles tendinopathy have functional limitations over a range of different capacities from endurance to explosive power (22-24). These deficits should be evaluated and compared to the healthier side and deficits addressed as part of treatment.

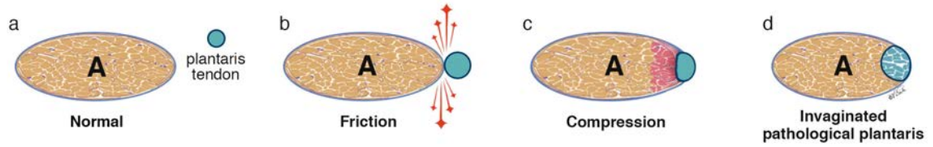
Hopping evaluates plyometric ability indicated by average jump height and the ratio of flight time to ground contact time, and pain indicates how well a load of approximately six times body weight is tolerated (Figure 1) (25). The heel-rise endurance test with good technique (26) can be used to evaluate endurance of the triceps surae muscles either by the number of repetitions or the total work completed, which takes into account that few people maintain full range of motion through the test. Pain during the heel-rise test also evaluates the ability to tolerate load of approximately four times body weight (Figure 6). Forward hopping is a good measure of maximum explosive ability, and tolerance of very high loads, approximately seven times body weight (Figure 4). Jumping and hopping tests are useful but do not isolate ankle joint function. If the athlete has been symptomatic for a long time or shows signs of shifting load away from the Achilles tendon when performing multi-joint movements, loaded heel-rises can isolate the ankle joint for more specific measures of strength and/or power.



^ Figure 6. Single leg calf raise. A: Good technique. B: Lacking full height. C: Excessive supination. D: Toe clawing.

IMAGING FOR DIAGNOSIS, PROGNOSIS, AND PREDICTION

Due to its superficial location, the Achilles tendon is easily imaged with ultrasound. The key findings in the tendon are thickening, increased vascularity and changes in echogenicity (how bright in colour the tendon appears) (27). The healthy Achilles tendon has uniform thickness across its length, no vascularity, and uniform echogenicity. Tendinopathic tendons on the other hand have an area of increased thickness, often contain neovascularisation, and have hypoechoic regions. In asymptomatic tendons, these same variables predispose a person to symptom development (27). Measuring the degree of thickening of the tendon, which is increased in tendinopathy, with ultrasound is reliable and in some studies has been found to be associated with symptom severity and prognosis of treatment (28-30). Ultrasound may be used to differentially diagnose other pathology and assess impingement of the Achilles tendon by other structures. If the site of a tendon pathology matches an area of contact to another structure, such as the calcaneus, or plantaris tendon (Figure 7), compression may contribute to the pathology. The compression may be visualised by moving the ankle into dorsiflexion (calcaneal impingement) or plantarflexion with calcaneal valgus (plantaris tendon).



^ Figure 7. Plantaris variants.

DIFFERENTIAL DIAGNOSIS

A thorough clinical examination is required to both diagnose Achilles tendinopathy, and examine for differential diagnoses (Table 1). Identifying red flags, systemic or serious diseases is important, the Achilles insertion is the most common bone tendon junction affected by enthesitis in sero-negative arthropathies.

Several clinical entities can mimic Achilles tendinopathy (Figure 8) by causing morning stiffness, pain in the heel region, and pain with plantarflexion.

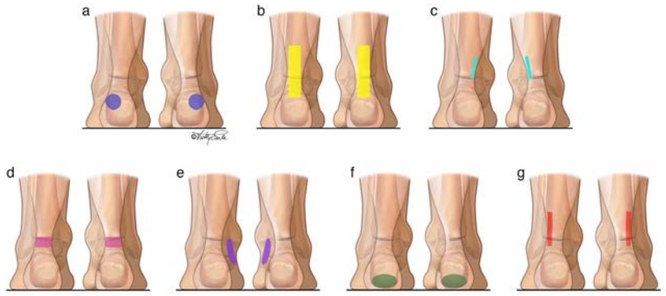


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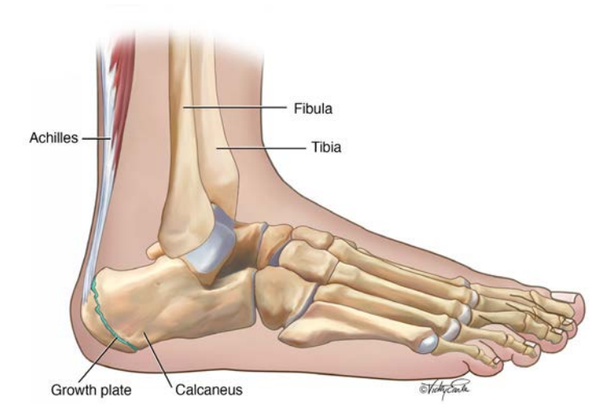
Figure 8. Achilles tendon pain map.

a) Insertional Achilles tendinopathy.
b) Achilles peritendinopathy.
c) Plantaris.
d) Mid-portion Achilles tendinopathy.
e) FHL/ tibialis posterior.
f) Superficial bursa.
g) Sural nerve.

Note: Pain locations are indicative of the patient reported location of pain, not palpation pain.



In adolescent athletes, Sever's disease (Figure 9) is a growth plate overuse injury analogous to Osgood-Schlatter of the knee. It is therefore exclusively found in athletes with an open growth plate, ages 8-15 (31). Pain with loading and tenderness on the calcaneal growth plate, on the medial and lateral aspect of the Achilles tendon are key signs of Sever's disease.

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Figure 9. Severs disease.

Posterior ankle impingement (32) is characterised by pain in the Achilles tendon region and is aggravated by plantarflexion. It can be differentiated from Achilles tendinopathy (that is painful with active plantarflexion) as posterior ankle impingement is painful with passive plantarflexion.

The flexor hallucis longus is an accessory plantarflexor muscle that can be affected by tendinopathy and (mostly) peritendinopathy that can mimic medial Achilles tendinopathy. It is mostly seen in dancers. Other medial and lateral ankle and foot tendons should also be ruled out as the source of pain.

Peritendinopathy is an inflammatory condition of the Achilles tendon peritendon and can occur as the sole injury or in combination with Achilles tendinopathy. Peritendinopathy has a more acute presentation, and crepitus may audible. Peritendinopathy responds to anti-inflammatory medication to a greater extent than Achilles tendinopathy.

Superficial calcaneal bursa can mimic insertional Achilles tendinopathy and is sensitive to type of shoes, those with a hard heel counter are provocative. Less commonly there can be a neural source of pain, either from proximal sources or the sural nerve itself.

PAIN	LIKELY SOURCE OF PAIN	CONSIDER THESE SOURCES
Posterior	Achilles tendon	Neural sources central or peripheral (sural nerve)
	Plantaris associated Achilles tendinopathy	
	Achilles tendon insertion	Seronegative arthropathies
	Superficial calcaneal bursa	
	Peritendinopathy	
	Posterior ankle impingement	
	Calcaneal apophysitis	
Medial	Medial tendons; FHL peritendinopathy, tibialis posterior tendinopathy or peritendinopathy	Neural; medial and lateral plantar nerve, posterior tibial nerve

^ Table 1. Differential diagnosis.

OTHER FACTORS TO CONSIDER

Numerous drugs and diseases affect the Achilles tendon however they are not often a factor in football players. Systemic and metabolic diseases such as rheumatoid arthritis, spondyloarthropathies, diabetes, hypercholesterolemia and hypercalcemia have all been associated with tendon pathology (33).

Fluoroquinolones are antibiotic medications and may be used to treat infections in football players. These drugs are associated with Achilles tendon ruptures and tendinopathies (34).

MANAGEMENT OF INITIAL PRESENTATION OF PAIN: WHAT TO DO WHEN THE PLAYER APPROACHES THE CLINICIAN

The first signs of Achilles tendinopathy are stiffness or minor pain that has no impact on the athletes' ability to perform and train as there is a warm-up tendency during activity. If these signs are recognised early, measures such as load management can prevent development into performance limiting Achilles tendinopathy. Minor symptoms, when consistent with Achilles tendinopathy, must be taken seriously by the coach and the medical team. Early load management and exercise therapy may keep the athlete performing at their peak.

When an athlete presents with performance limiting Achilles tendinopathy, the first priority is to educate them. Athletes can worry that their Achilles tendinopathy may progress to an Achilles tendon rupture and be fearful of loading. Kinesiophobia should therefore be addressed early through reassurance that further injury will not result from continued loading of the Achilles tendon. This is especially important since a period of complete rest will not help recovery and can waste valuable time.

The pain monitoring model should be used on the first approach to the clinician (Figure 10) (35). The pain monitoring model with a scale of 0 (no pain) to 10 (worst pain imaginable) requires the athlete to become familiar with expressing their pain on the scale. Moderate pain (up to 5/10) during both training and treatment for Achilles tendinopathy (35) is considered reasonable, although its use in elite athletes has not been determined. Evaluating the pain level the next morning on both a subjective scale (morning stiffness) and an objective test (hopping) is important to determine if the load the previous day was acceptable or excessive for the tendon.

PAIN-MONITORING MODEL
Numerical Pain Rating Scale (NPRS)

1. The pain is allowed to reach 5 on the NPRS during the activity.
2. The pain after completion of the activity is allowed to reach 5 on the NPRS.
3. The pain the morning after the activity should not exceed a 5 on the NPRS.
4. Pain and stiffness is not allowed to increase from week to week.

Figure 10. Pain monitoring model
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The athlete, and potentially the coach, should be informed on the prognosis and timeframe of recovery. The prognosis is generally good, with studies showing majority of players recover with treatment by the 12th week (36). However, the expectation of a longer duration from the start may prevent re-injury due to early return (3), as well as loss of motivation if recovery is not reached within 12 weeks. The greatest risk for reoccurrence/re-injury is returning to full participation too early. It is also important to not only rely on the symptomatic state since full resolution of symptoms does not ensure full recovery of function, and tendon capacity to withstand load.

The management plan must be athlete-centred at the initial evaluation. It may be possible to wait for the off-season to start progressive loading of the Achilles tendon. Planning for important competitions and setting realistic goals valued by the athlete and the team is important. Tapering Achilles tendon load as an important match approaches is a simple change that may extend recovery slightly but maximise the ability of the athlete to perform.

EXERCISE THERAPY

The cornerstone of treatment for Achilles tendinopathy is progressive loading, with a pain monitoring model. The loading on the tendon needs to be heavy and long enough to elicit a response (Figure 11a anad b) (37, 38). The loading then needs to be repeated often enough until the desired response is reached (39). If 'heavy enough, long enough, often enough' is reached, there is considerable flexibility to ensure that the loading program is optimal for the athlete's training and playing schedule.

Three varied protocols span the range of loading and frequencies have been published and show good results, however they have not been applied to football players or elite athletes. A meta-analysis found that the eccentric calf muscle training and the comprehensive Achilles tendon loading (40) protocols had equivalent results (41). The more recent heavy-slow resistance training program was published with a direct comparison to the eccentric calf muscle training protocol and showed compatible effectiveness (42). (Table 2).

^ Figure 11a and b. Weighted single leg seated and standing calf raises.

PROTOCOL	REPETITIONS PER SESSION	NUMBER OF EXERCISES	FREQUENCY
Comprehensive Achilles tendon loading	150+ (high load)	4	Every day (low load) plus Every other day (high load)
Exercises used: Bilateral heel raises, unilateral heel raises on a step, eccentric unilateral heel raises on a step, quick rebounding heel raises			
Heavy-slow resistance training (higher end)	135	3	3x / week
Exercises used: Bilateral seated calf raise, straight knee heel raise in leg-press machine, standing straight knee heel raise standing on a weight plate			
Eccentric calf muscle training	90	2	2x / day
Exercises used: Standing eccentric only unilateral heel raise, standing bent-knee eccentric only unilateral heel raise			

Repetitions per session is the total number of repetitions from all the exercises. The comprehensive Achilles tendon loading program numbers are the lower end of phase 3 of the program. The heavy slow resistance numbers are the high end of the program. The eccentric calf muscle training program is constant throughout.

^ Table 2 . Summary of various exercise loading programs for Achilles tendinopathy.

The programs can be followed precisely or used to guide exercise options. Out of the three loading programs the eccentric calf muscle training program (43) is the simplest to execute, consisting of two exercises performed twice per day. The program may not impact on capacity to train and play as the load is fairly low and frequent. This program is completed on each leg independently.

The heavy-slow resistance training program (42) starts with load equal to the athlete's 15 repetition maximum. Load is progressed to the athlete's 6 repetition maximum. These heavy loads may be more beneficial in an athlete with large strength deficits in the calf muscles.

The comprehensive Achilles tendon loading program (44) is a mixture of heavy loads and high repetitions with some exercises progressed with increasing repetitions and others with increasing weight. It is divided into four phases: symptom management, recovery, rebuilding, and return to sport phase. The program includes a plyometric component, starting with fast rebounding heel-rises and progressing to single leg hopping.

Quicker more explosive contractions resulting in higher peak tendon loads but a low time under tension are often the most aggravating. Figuring out a weekly dose threshold for high intensity activities is required on a per-athlete basis as no study has examined these limits.

For athletes with insertional tendinopathy, special training modifications can include using a heel lift in a shoe to decrease the dorsiflexion range of motion required during sports. Changes in playing surfaces also affect those with both midportion and insertional symptoms with softer surfaces causing increased symptoms especially at the insertion.

It is also important to perform a thorough evaluation of each athlete to ensure that there are no other weaknesses or limitation in range of motion of the ankle (especially dorsiflexion) or in joints proximally or distally that could result in increased or altered load on the Achilles tendon. There is no evidence that foot posture is associated with Achilles tendinopathy (45) but again this has not been evaluated in either football players or elite athletes.

TRAINING MODIFICATIONS USING PAIN MONITORING MODELS

Pain monitoring is an important tool to monitor and maximise training loads without overloading the Achilles tendon. When athletes present with minor symptoms of stiffness and/or minor pain, a pain monitoring model in conjunction with a progressive loading program may prevent progression to fully symptomatic tendinopathy.

In the early stages of Achilles tendinopathy, the pain might disappear when warmed-up but as the injury progresses pain might be present during training sessions. The question is therefore often asked; how much pain is 'OK' before progress is hindered? A study in recreational athletes compared continued and discontinued sports activity, where the continued activity group was instructed to not let pain exceed 5/10 on a numerical rating scale (35). At a 12-month follow-up there were no differences between the two groups. If a pain monitoring model is followed, total cessation of training is neither required nor beneficial. Modification of training load to maintain pain intensity below the threshold while maximising performance is beneficial, although its application to aspiring and elite athletes is unknown.

RETURN TO SPORT

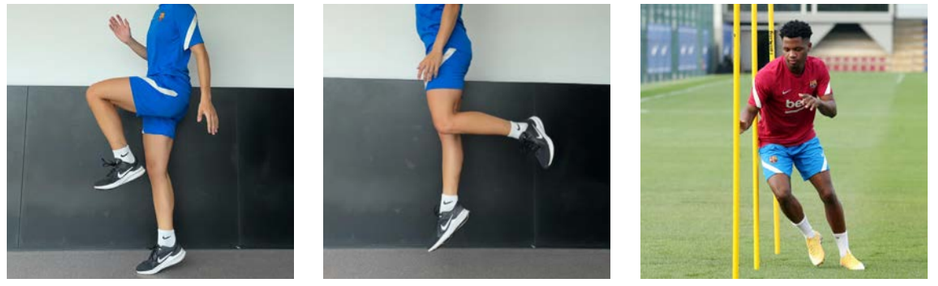
A return-to-sport program for Achilles tendinopathy can facilitate the decision making from rehabilitation to return to participation, return to sport and return to performance (46). The key aspects of this program are the use of the pain-monitoring model, monitoring morning stiffness and pain, and the rating of perceived exertion during training. These guide the progression of loading and the need for recovery days between heavy activity days. It is important to recognise that tendon capacity and musculoskeletal function may not be fully recovered even when the athlete is back training and competing at pre-injury levels. Changes in tendon properties or muscle-tendon function may persist for a year or more, predisposing the athlete to re-injury (47, 48). Therefore, it is critical that tendon loading exercises are continued and the athlete is monitored closely for recurrence of symptoms.

OTHER TREATMENTS

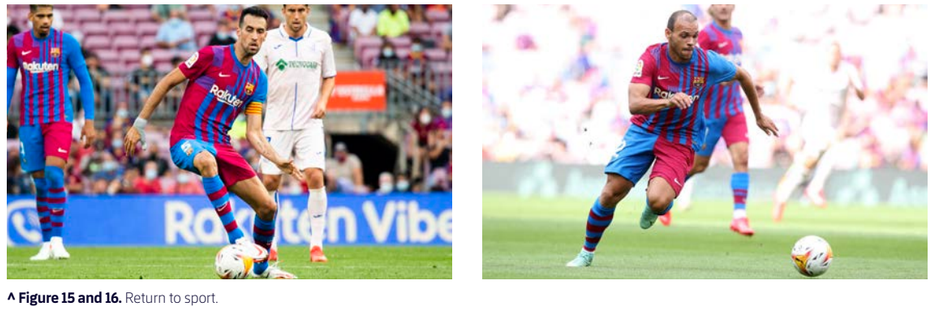
Exercise therapy is the mainstay of Achilles tendinopathy treatment, additional interventions such as extracorporeal shockwave therapy, when provided in addition to exercise, may provide benefits for some athletes (49). Rigid sports tape (not elastic tape) can provide benefits, especially when foot posture is corrected (49).

Injection therapies such as corticosteroid injections may offer benefit when exercise alone is insufficient (49). There is to date no evidence to support the efficacy of platelet rich plasma injections in the treatment of Achilles tendinopathy.

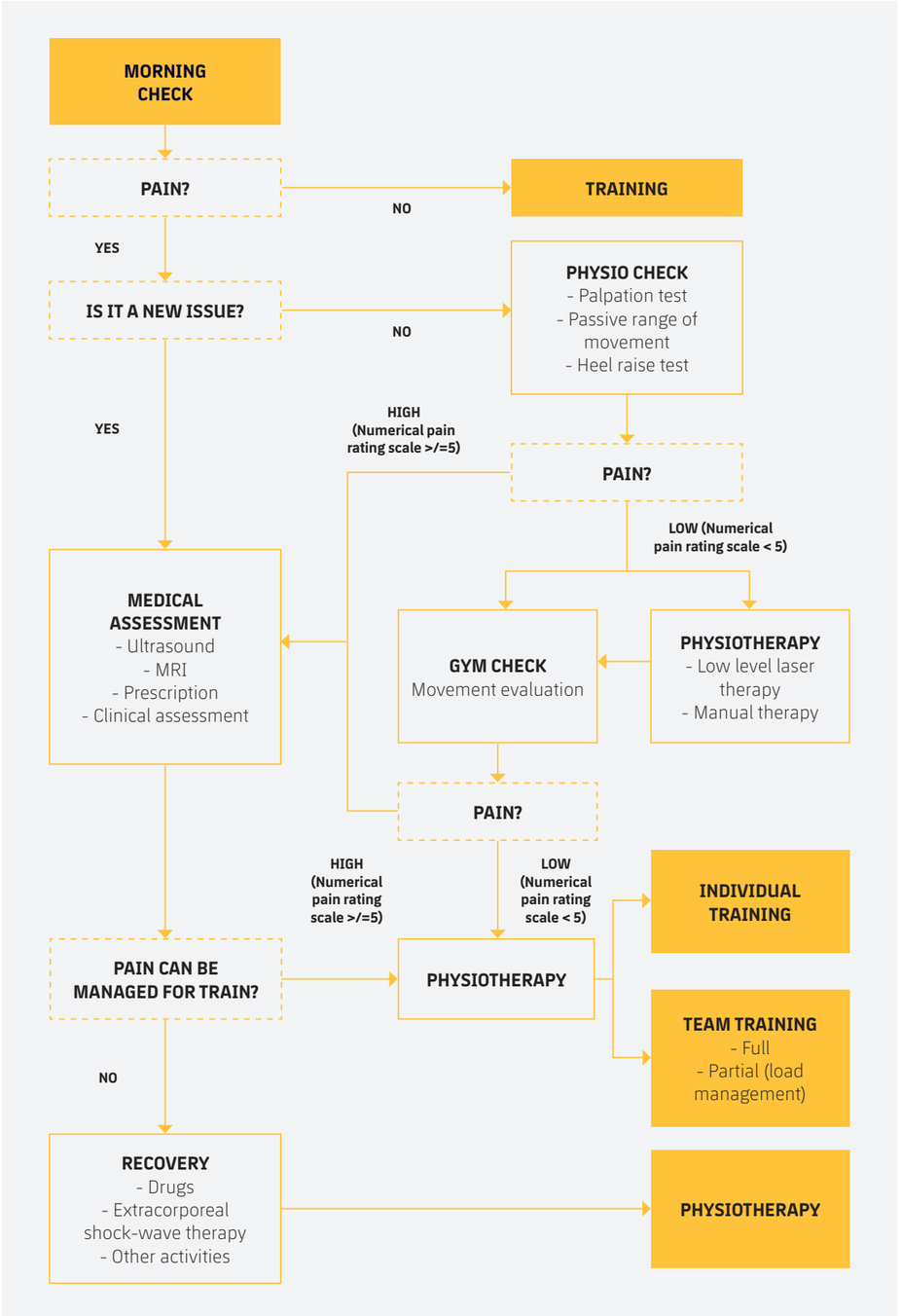
When treatment with exercise therapy fails, surgical interventions may be indicated (50). Most common surgical indicators are bony deformities or calcifications that can be surgically removed. Surgeries excising tendinopathic tissue or addressing neovascularisation can show positive results (51, 52) but high-quality evidence is lacking.



^ **Figures 12, 13 and 14.** Example return to sport drills.



^ **Figure 15 and 16.** Return to sport.



^ Figure 17. Flowchart of the daily evaluation process

— Maurizio Fanchini, Marco Barbato, Valerio Flammini, Walter Martinelli and Marco Esposito

MANAGING THE TENDON: THE ALGORITHM FOR DETERMINING CAPACITY TO TRAIN AND PLAY - ALLOWING THE PLAYER TO CONTINUE OR WITHDRAW FROM RETURN TO PLAY

ON FIELD MANAGEMENT DURING TENDINOPATHY

Managing a player with Achilles tendinopathy means balancing rehabilitation exercises and training and playing, to limit the cumulative effect of load that may increase symptoms and interrupt football training or competition. Pain monitoring before, during, after exercise, and the next morning, as well as in a daily football training routine is essential (Figure 16).

Players are checked when they first arrive at the training facility and activity is determined based on the pain and the results of objective tests during exercise in physiotherapy and gym (such as single leg hopping). The decision to withdraw or allow the player to train individually or with the team is then made and training participation is then managed with an on-field training plan.

Understanding load is just one aspect of the multidimensional management process. A training session systematically provides the stimuli to elicit internal responses (i.e. psychophysiological stresses) that ultimately improve performance, and is monitored using measures of both external and internal training loads. External training load (such as different running metrics collected with the global positioning systems) is the activity prescribed to the athlete and the internal training load (heart rate, rate of perceived exertion) is the physiological effect imposed by the external training load that varies with individual athlete characteristics (1). Therefore, a similar external load can result in different internal training load responses providing different stimuli for individual athletes and consequently different training outcomes (performance but also pain). For example, a player suffering Achilles tendinopathy can run the same distances (or high-intensity distances) with similar internal load compared to a healthy player but that external load may be enough to increase pain during/after training or the day after the session.

During planning practitioners should focus on both extrinsic and intrinsic risk factors that may influence the load tolerance (pain-response) of players suffering Achilles tendinopathy. These include:

- Age and previous injury are potential risk factors for calf muscle injury (> 26 years), fatigue from strenuous training sessions may result in an impaired muscle function to absorb shock which can put high stress on the Achilles tendon (3). These factors also increase the risk for Achilles tendinopathy
- Pre-season is where football players first participate in a high number of training sessions (frequently twice a day) and pre-season is the period of high incidence of Achilles tendinopathy (3).
- Changes in training load intensity and type compared with the preceding off-season should be considered and the reduction of exposure to training is an appropriate mitigation strategy. For example, allowing only one session per day or reducing the number of sessions during the week by checking loads and tissue response (i.e. pain monitoring model). Maintaining a load on the tendon and athlete in periods of unloading such as the off-season can mitigate the issues when transitioning back to pre-season loading.
- High-intensity running bouts, uphill runs (long or short distances) as well as jumps and sprints with change of directions are frequently used to improve player's fitness however such exercises should be carefully evaluated in players suffering Achilles tendinopathy due to the increased stress on the muscle-tendon unit (53, 54).

Weekly management should consider the days before and after match day as well as coach's tactical needs. Exercises performed on a full field allow players to reach more high-speed running distance compared to small pitches. Given the nature of football a player has to be able to accelerate and maintain speed, decelerate and change direction and change intensity with and without the ball (55, 56). These situations, requiring neuromuscular load (57), may provide high stress on the muscle-tendon unit and the Achilles tendon. However,

avoiding high-intensity activities may lead to under-preparation and ultimately limit the player's involvement in competition. The pain model can be useful to balance the need to cope with the match demands but also avoid pain provocation.

DIAGNOSIS AND TREATMENT OF ACHILLES TENDON RUPTURE

CLINICAL PRESENTATION

An acute Achilles tendon rupture is an unexpected acute injury that occurs with a forceful push-off on the weight-bearing leg with the knee extended, at the point of maximal energy storage. The Achilles tendon is strained both from the ankle/foot being forced into dorsiflexion and a strong calf muscle contraction. In football this often occurs during a quick change of direction. There is initially a sharp sensation and often an audible "pop". The patient frequently describes it as a feeling of someone kicking or hitting them in the back of the leg. The pain subsides quickly, but the patient has difficulty walking and stabilising on the leg. In non-weight bearing the patient can still produce some plantar flexion movement with the use of synergistic muscles but is unable to perform a heel-rise in standing on the injured side. There is degenerative pathology in the Achilles tendon prior to complete rupture, however the majority of the individuals have not had any previous symptoms (58).

^ Figure 18a and b. Calf squeeze or Simmond's test. The ankle should plantarflex when the calf muscle is squeezed if the Achilles tendon is intact.

^ Figure 19. Matle's test. A positive sign is that the foot on the injured side rests in a more neutral or dorsiflexed position.

DIAGNOSTIC TESTS AND CLINICAL EXAM TESTS

The patient's description of the injury is the strongest indication of an acute complete Achilles tendon rupture. During the initial inspection of the Achilles tendon there may be an observable and palpable gap in the tendon. After a few hours this might be less noticeable due to swelling in this area. The most common test to verify the diagnosis is the Thompson's test, also known as the calf squeeze or Simmond's test (Figure 18a and b), which has a sensitivity of 0.96 and a specificity of 0.93 (59). For this test the patient lies in prone and the examiner squeezes the affected calf muscle. If the tendon is intact the ankle will plantarflex but if the tendon has ruptured minimal plantarflexion of the ankle will occur. Another test is the Matles test (Figure 19) with a sensitivity of 0.88 and a specificity of 0.85 (59). For this test the patient bends both knees while in prone. A positive sign is that the foot on the injured side rests in a more neutral or dorsiflexed position.

MANAGEMENT OF INITIAL PRESENTATION OF RUPTURE

The initial treatment should be to place the foot in plantar flexion (to approximate tendon ends) and apply compression. Weight-bearing should be avoided to minimise separation of tendon ends. If an orthotic boot is available, then heel lifts should be used plantar flex the ankle within the boot. The injury is then assessed to determine whether surgical treatment is required. If surgical treatment is proposed, it is best performed within 72 hours of the injury (60). Most systematic reviews report that the re-rupture rate is lower when treated surgically and this may elicit greater improvement in symptoms and function (61, 62). However, surgical and non-surgical treatments are reported to have similar outcomes and re-rupture rates if followed by comparable early mobilisation and appropriate rehabilitation (62). There is no clear evidence of optimal surgical technique.

IMAGING FOR DIAGNOSIS, DETERMINING TREATMENT AND PROGNOSIS

Ultrasound imaging immediately following an Achilles tendon rupture may help determine the degree of tendon end separation, and guide whether surgical treatment is warranted to limit re-rupture or poor functional outcomes (63). . In a recent study it was reported that a tendon gap of >10mm was related to increased risk of re-rupture and a tendon gap >6mm was related to worse outcome if treated non-surgically (63).

REHABILITATION

The rehabilitation process following Achilles tendon rupture is fairly similar irrespective of the initial surgical or non-surgical management. Early mobilisation to progressively load the Achilles tendon is an important determining factor for successful outcomes (64). Early functional mobilisation is reported to be superior for decreasing calf muscle deficits and preventing excessive tendon elongation (64, 65). Early functional mobilisation most commonly includes weight bearing within 2 weeks, and the commencement of ankle range of motion and rehabilitative exercises at around 2 weeks (66). The purpose of early rehabilitative exercise is to minimise calf muscle atrophy and tendon elongation and promote tendon healing and recovery.

Historically, treatment protocols have been time dependent and exercises and activities were progressed based on the time post injury/surgery. However, recovery timeframes vary significantly between individuals, and therefore progression should be both time and criteria dependent (Table 1). Treatment and rehabilitation can be divided into four phases (67); controlled mobilisation, early rehabilitation, late rehabilitation, and return to sport. Each phase has its unique purpose and goals (Table 3).

PHASE	GOALS	ACTIVITIES AND EXERCISES	PROGRESSION CRITERIA
The controlled mobilisation phase: 0-8 week The specifics of this phase vary dependent on initial treatment (surgery or non-surgical, weightbearing or non-weightbearing) as well as physician preference. Progressive removal of heel-lifts in walking boot occurs during this phase	Promote tendon healing Minimise tendon elongation (avoid DF and stretch of tendon) Minimise muscle atrophy Avoid re-rupture, infection and DVT	Boot or cast to keep ankle in PF Weight bearing in boot Isometric plantar flexion in brace/boot Seated heel rises with starting position in the PF position Theraband ankle exercises	
Early rehabilitation phase 6-11 weeks Visit for physical therapy 2-3 times a week and home exercises daily. Progressive removal of boot and heel lift. Walk in shoes with higher heel height.	Avoid re-rupture, infection and DVT Avoid further tendon elongation Overcome fear of loading Recover walking Improve calf muscle – recover unilateral heel rise strength & endurance	Exercise bike Gentle ankle range of motion (not stretching of calf and Achilles tendon) Ankle strengthening using a resistance band or cable machine Sitting heel-rise with external load (25-50% of body weight) Standing heel-rise progressing from two legs to one leg Gait training Balance exercises Leg presses Leg extensions Leg curls Foot exercises Pre-running exercises Bilateral rebounding heel-rises Bilateral hops in place Gentle jogging in place	Criteria that can be used for starting pre-running exercises. To be able to perform five single leg heel-rises at 90% of height
Late rehabilitation 12-16 weeks	Recovery of tendon strength Recovery of muscle strength and endurance Recovery of function	Continue exercises at home and fitness facility with focus on gastroc-soleus strengthening and flexibility (if needed). Do not stretch gastrocnemius once DF (gastroc) equal to the opposite side. Add running based exercises as long as patient meets criteria	Criteria that can be used for starting a running progression program are: 1. To be at least 12 weeks after injury and be able to perform five single-leg standing heel-rises at 90% of the maximal heel-rise height of the injured side. OR 2. If unable to achieve the above criteria by week 14-15, the patient can start running progression if they are able to lift at least 70% of their body-weight during one single-leg heel-rise.
Return to sport phase	Return to team, field, competition Realistic expectation Adjust for compensation for lack of full triceps surae recovery	Start sports specific exercises and activities	Criteria for return to non-contact sports 16-20 Weeks and able to perform 85% of uninvolved number of heel-rises or >25-30 reps Criteria for return to contact sport 21-24 weeks and able to perform 90% of uninvolved side number of heel-rises or >30 reps

PF = plantarflexion.

^ Table 3. Rehabilitation protocol after a complete Achilles tendon rupture (67).

THE CONTROLLED MOBILISATION PHASE: 0-8 WEEK

The main reasons for lack of full recovery after an Achilles tendon rupture are tendon elongation during healing (usually occurs in the first 12 weeks) and calf muscle weakness. It is important to try to minimise the degree of tendon elongation as it is related to ongoing symptoms and poorer functional capacity (68-70). The foot should be maintained in plantar flexion early and plantar flexion gradually decreased by removing heel lifts. Calf muscle atrophy also occurs quickly, therefore encouraging the patient to maintain some calf loading through isometric plantar flexion exercises whilst in the boot or cast may promote tendon healing and reduce muscle atrophy (Figure 20).

^ Figure 20. Theraband ankle exercises.

THE EARLY REHABILITATION PHASE: 6-11 WEEK

During this phase, it is important to be aware that the risk of re-rupture is the greatest when starting to walk without the boot (71). Exercises should be progressed slowly, dosage of exercises and walking must be modified in accordance with pain and swelling in the Achilles tendon. Use of compression socks during the day can reduce swelling in lower leg. Stretching is not recommended in this rehabilitation phase to reduce tendon elongation. Recovery of calf muscle strength is a main goal during this phase and non-weight bearing exercises such as seated heel-rises and weight-bearing bilateral heel-rises may be introduced.

THE LATE REHABILITATION PHASE: 12-15 WEEKS

The goal in this phase is to strengthen and prepare the calf muscles for more demanding activities. The goal is to be able to perform single-leg heel rises as soon as possible. Additionally, strengthening exercises for the rest of the kinetic chain and core should be introduced, although in elite athletes additional supervised exercises should be included earlier. Studies have found that in order to run and jump despite ankle plantar flexion weakness, compensation occurs at both the knee and the hip (72), and therefore it is recommended that both knee and hip strengthening exercises are incorporated at this stage of rehabilitation. Appropriate timing to initiate running and jumping activities is individualised and depends on the recovery of calf muscle strength. It is important to choose valid and reliable test methods to be able to evaluate the patients' functional capacity prior to return to running and sport (Figure 21). The same testing battery as outlined previously for Achilles tendinopathy includes valid and reliable tests for calf muscle strength, endurance and jumping ability (25, 73). The majority of professional football players return to sport 7-9 months after injury/surgery (74).

> Figure 21. Jump test on force plate.

ON FIELD MANAGEMENT AFTER TENDON RUPTURE

In the return to performance phase the main aim is to increase specificity and load with the player exposed to activities that reflect the volume and intensity of training and match. During competitions, players are expected to cover a total distance of 10-12 km of which 1.7-1.8 km performed at high speed (>16 km*h-1), with 1000-1400 short movements such as change in speed and direction (75).

While prescribing exercise, practitioners should consider different types of high-intensity interval training. For example, interval training (4 x 4 min at 90-95% of maximum heart rate with 3 min of active recovery) provided the same effect in aerobic fitness and specific performance as small-sided games (3 vs. 3, 4 vs. 4, 5 vs. 5)(75). In addition, high-intensity interval training consisting of different running activities are performed at high, but not maximal, intensity as well as short to long sprints (10–30 s) performed at maximal intensity; both are spaced out by recovery periods. The high-intensity interval training can be organised as circuits with change of directions and technical exercises to stimulate coordination and agility.

A team training session can be considered an additional load for the player and a gradual exposure to team activity is needed. The cognitive (tactical) activity required during a team session places high mental load on the rehabilitating player. During the first weeks of team training only low-intensity activity are performed (warm-ups, technical and tactical exercises), in the subsequent weeks, more intense exercises such as small-sided games are prescribed. For example, a 2 vs. 2 showed higher intensity compared to competitive matches, while 4 vs. 4 and 6 vs. 6 showed similar and lower intensity compare to matches (75).

Gym activities should be organised to maintain the strength and power of the lower limbs and calf muscles with both concentric and eccentric overload contractions. During and after return to performance, players' load (i.e. exercise type, frequency, intensity and volume) should be managed in order to allow the increase or maintenance of demands on the neuromuscular system and minimise the risk of re-injury (76).

Summary:

- There are two main types of Achilles tendon injury that affect football players; Achilles tendinopathy and Achilles tendon rupture.
- Achilles tendinopathy is an overuse type injury, whereas Achilles tendon ruptures occur acutely and are rarely proceeded by symptoms.
- There are two main types of loads that the Achilles tendon is exposed to are tensile (energy storage and release) loads and compressive loads.
- Compression in the Achilles tendon may occur in the mid-portion, where the plantaris tendon is often implicated as a source of compression, or at the calcaneus.
- Achilles tendinopathy often exhibits a warm-up type pattern with activity in the early stages, or athletes may notice morning pain or stiffness, especially the day following intense training or competition.
- The diagnosis of Achilles tendinopathy is primarily clinical, with imaging only used when an alternate diagnosis is suspected.
- Clinical tests use progressively increasing tendon load are to diagnose Achilles tendinopathy. Clinicians should expect to see a graduated increase in localised tendon pain with increasing tendon load.
- Individuals with Achilles tendinopathy generally demonstrate significant functional limitations over a range of different capacities ranging from endurance to explosive power.
- Due to its superficial nature, the Achilles tendon is easily assessed with ultrasound imaging. Key findings include thickening, vascularity and echogenicity.

Clinical Implications:

- Several clinical entities can mimic the signs of Achilles tendinopathy, including Sever's disease in adolescents, posterior ankle impingement, medial and lateral foot tendinopathy and peritendinopathy. Clinicians should be familiar with the hallmark signs of tendinopathy in order to identify when a potential differential diagnosis is likely based on the nature and behaviour of symptoms.
- Load management and education are the key components of the early phase of treatment. It is important that athletes understand provocative loads and how to modify their training accordingly, as total cessation of loads are neither required nor beneficial for the athlete's recovery.
- Kinesiophobia is common and educating the athlete is critical to ensure they load the tendon as part of their rehabilitation.
- A pain monitoring approach should be followed, with training and match-play modified in order to maintain pain intensity at acceptable levels while still enabling the athlete to perform. Morning pain and stiffness the day after loading is a critical monitoring tool.
- Training and playing with Achilles tendon pain can affect the rest of the strength and power of the kinetic chain.
- Intrinsic and extrinsic factors that may influence the load tolerance of the tendon. These include age, previous injury, fatigue, time in season and exposure to high magnitude tensile loads.
- Initial treatment for an Achilles tendon rupture requires early unloading followed by a similar exercise progression to Achilles tendinopathy. Loads should be progressed slowly in accordance with patient symptoms.

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THE FC BARCELONA TENDON GUIDE

— Johannes Zwerver, Michel Brink, Jill Cook

3.2. PATELLAR TENDON INJURIES

Patellar tendinopathy is an overuse injury that is characterised by pain localised to the inferior pole of the patella during high tendon load activities (1). Prevalence rates of patellar tendinopathy in a football population have been found to be 12% in elite players and 2.5% in sub-elite players (2, 3). In the UEFA Elite Club Injury Study (ECIS) of top-level male European teams, patellar tendinopathy represents 1.1% of all time-loss injuries (4). The overall incidence of patellar tendinopathy is 0.08 per 1000 hours of football training and match play, equivalent to approximately one patellar tendon time-loss injury every second season (4). Patellar tendinopathy is more common in men compared to women, with a male prevalence rate in elite football approximately double that of female athletes (5).

The incidence of patellar tendinopathy varies seasonally, with the highest proportion of injuries occurring during the pre-season (3, 6). A higher prevalence is also seen after a mid-season break (3). Patellar tendinopathy usually results in short duration absence from training (less than 1 week), but has a high recurrence rate of 20% (3). Although many athletes are still able to train and play despite patellar tendon pain, their performance may be negatively impacted, even if no time-loss is registered (3).

Despite numerous hypotheses regarding the aetiology and pathogenesis of patellar tendinopathy, the exact pathophysiology remains unknown. Ultrasound and MRI can be used to visualise focal patellar tendon abnormalities, however there is a lack of correlation between symptoms and imaging findings (7). Complete patellar tendon ruptures are very rare and always require surgery, resulting in a longer rehabilitation period with an average return to play time of 10-months (8). A full discussion of the management of patellar tendon ruptures is beyond the scope of this chapter, the focus is on tendinopathy at the proximal attachment to the patella.

Figure 1. Jumping requires high tensile load to be transmitted through the patellar tendon.
<

PATHOLOGY

The pathological tendon changes in the patellar tendon are the same as described in other tendons, however there is evidence that the onset of pathology occurs earlier and may begin during adolescence (9). While pathology in other tendons has been linked to accumulation of pathology throughout the lifespan (10), patellar tendinopathy is highly prevalent in young jumping athletes between the ages of 14-18 (11), and the development of patellar tendinopathy after adolescence is less common (12). The patellar tendon matures through a cartilage plate without an apophysis and reaches full maturity 2 years after peak height velocity (9). Exposure to repetitive high magnitude tendon load throughout adolescence may disrupt the developing bone tendon junction (9), and excessive loading during this developmental stage may be the source of pathology that is likely permanent. Whether the athlete develops symptoms at this stage or later in their career may be more related to aberrant loading patterns.

During adolescence, tendons appear to be more capable of structural adaptation when compared with adult tendon tissue (13). As there is a correlation between tendon pathology and structural adaptation (14), this may be advantageous when rehabilitating younger athletes with patellar tendon pathology.

^ **Figure 2 and 3.** High patellar tendon load activities.

WHAT LOADS AFFECT THE PATELLAR TENDON?

Football is characterised by fast changes of direction, acceleration and deceleration, together with activities such as jumping and tackling. These activities place high load on the lower extremities (15). The patellar tendon is exposed to high energy storage and release loads during football, and the highest loads on the patellar tendon occur during the final stages of eccentric knee flexion prior to take-off in jumping or changing direction (Figures 1-3). Faster and more agile athletes may be predisposed to the development of patellar tendinopathy, as they are competent in storing and releasing large amounts of energy in the patellar tendon. There are no compressive or friction loads on the patellar tendon.

Quantification of training load has become popular in both football science and football practice that allows measurement of the individual load per session. The focus has been on physiological load indicators such as the total distance covered and the distance covered in certain speed zones (15). Furthermore, the frequency of activities can be recorded, the number of jumps in professional football is reported to be 15 per match (16). This is relatively low compared to jump sports such as volleyball where athletes jump up to 300 times in one single match (17). However, elite football players cover approximately 11 km on average per match, including 550m at high intensity (> 20 km/h), and 240 acceleration efforts (18). This is a substantial weekly load when training session and matches are combined. The sum of weekly load of the different load indicators can then be used to assess association with overuse injuries. In the case of tendinopathies in football, a stronger relationship can occur when accumulating loads over longer time frames, such as weeks to months.

Although these measures provide insight to the load players are exposed to, the biomechanical load of high intensity activities are largely neglected. This requires sensor set-ups that can quantify angular velocity of hips and knees (19, 20) and quantify changes of direction at high velocity (21). The biomechanical load truly related to tendon adaptation or maladaptation in football could be quantified, similar to what has been studied in volleyball (22).

RISK FACTORS

TIME OF SEASON

A higher incidence of both Achilles and patellar tendinopathy has been observed during pre-season compared to the competitive season in professional football (3, 6). There are several likely explanations for this variation including; pitch and weather conditions, a greater tendency to rest players with tendon pain during the off-season, and the overall higher volume and intensity of training encountered during the pre-season period, especially if there has been a period of unloading in the off-season (6).

TENDON LOAD AND GENERAL WORKLOAD

An association between workload, in particular spikes in workload has been suggested to increase the risk of soft-tissue injuries (23). In the UEFA elite football cohort, a relationship between spikes in workloads (measured using session-RPE) and non-contact injury was observed, although the predictive ability was low (24). No clear association between workload and injury is unsurprising given the complex and multifactorial nature of injury occurrence, however, it is reported as one of the factors to consider by football team practitioners (25, 26). For tendon disorders, an association between high total exposure to football (training and match hours) and patellar tendinopathy has been reported in elite players (27). Numerous risk factors including lower body strength, repeated sprint ability and speed training may further mediate the relationship between workload and the incidence of injury (28).

^ **Figure 4.** Change of direction drills.

BIOMECHANICAL FACTORS

Several studies have linked poor dorsiflexion with patellar tendinopathy (29, 30), likely due to a decrease in shock absorption at the ankle during landing that leads to increased knee loading during takeoff. It is not known whether this can be translated directly to football, although significant dorsiflexion demands are probable in change of direction (Figures 4 and 5) tasks, as better athletes tend to lower their centre of mass (31).

^ **Figure 5.** Change of direction during a game. Note: the significantly lower centre of mass of the player on the left.

CLINICAL PRESENTATION

HISTORY

The most specific and defining clinical features of patellar tendinopathy are (1) pain localised to the inferior pole of the patella and (2) load-related pain that increases with the demand on the knee extensors, especially in activities that store and release energy in the patellar tendon.

Patellar tendon pain is localised and does not spread with load. It is usually of gradual onset and symptoms often start after changes in training load or intensity, playing surface or shoes. Patellar tendon pain occurs directly when the tendon is loaded and usually disappears almost immediately when the loading is ceased. A misleading feature for many athletes is the so called "warm-up" phenomenon, where the patellar tendon pain may improve during training. They often continue to exercise and misunderstand the importance of this initial pain. Players may often experience increased pain after the training/match and the following day.

A key feature is that the pain is dose dependent; higher loads for the tendon result in more pain. Pain is rarely experienced in a resting state, although pain can occur with prolonged sitting (especially in a car), however this is also present in patellofemoral pain. Players will often report a change in function and performance noticing their speed and agility are decreased, due to both pain and dysfunction. Length of symptoms will amplify these performance issues.

Other possible risk factors like previous injuries especially ankle sprains where range and strength of the ankle can be compromised should be documented. It is rare to find co-morbidities in athletes with patellar tendinopathy.

PHYSICAL EXAMINATION

Athletes with patellar tendinopathy typically indicate with one finger where the most painful spot is. The proximal patellar tendon is often tender on palpation, but the clinical utility of palpation tenderness is limited as this can be tender in other conditions such as patellofemoral pain (32).

An examination of the entire kinetic chain is necessary to identify relevant deficits at the hip, knee, and ankle/foot region. Quadriceps and calf muscle weakness, loss of power and atrophy is commonly present, and gluteal muscle strength should also be assessed. Reduced ankle dorsiflexion (Figure 6) (33), hamstring and quadriceps flexibility (34), and foot alignment (35) should be assessed as some of these have been associated with patellar tendinopathy.

Figure 6. Knee to wall.
>

Localised tendon pain is reproduced with patellar tendon loading maneuvers like hopping or deceleration, and aberrant movement patterns may be evident. The athlete may avoid deeper ranges of knee flexion and have reduced leg power with hopping, illustrating deficits throughout the kinetic chain. These movement patterns can be measured with simple apps that can then become an outcome measure as strength and range improve. Contact mat hopping to analyse flight time to contact time can also be used to monitor progress.

The single leg decline squat (Figure 7) produces high levels of localised pain early in knee flexion range. This may be utilised in the clinical setting to increase the likelihood of the diagnosis patellar tendinopathy and also to define the degree of tendon irritability.

Figure 7. Single leg decline squat. >

OUTCOME MEASURES

The Victorian Institute of Sport Assessment-patellar (VISA-P) questionnaire (Figure 8) is a validated pain and function outcome measure that can also be used to assess severity of symptoms and monitor outcomes. The VISA-P, available in several languages is a 100-point scale, with higher scores representing improved function and less pain. The minimum clinically important difference is a change of 13 points (36). The VISA-P is less sensitive to small changes in the condition, and it is best used at monthly intervals.

VISA-P

DATE __/__/____ INITIAL ASSESSMENT ☐ DISCHARGE ASSESSMENT ☐
NAME _____ SURNAME _____ AGE ___ WEIGHT ___ HEIGHT ___
SPORT _____ TEAM _____ PHYSICIAN _____

1. For how many minutes can you sit pain free?

0 mins ☐☐☐☐☐☐☐☐☐☐☐ 100 mins **POINTS** ☐
0 1 2 3 4 5 6 7 8 9 10

2. Do you have pain walking downstairs with a normal gait cycle?

Strong severe pain ☐☐☐☐☐☐☐☐☐☐☐ No pain **POINTS** ☐
0 1 2 3 4 5 6 7 8 9 10

3. Do you have pain at the knee with full active nonweightbearing knee extension?

Strong severe pain ☐☐☐☐☐☐☐☐☐☐☐ No pain **POINTS** ☐
0 1 2 3 4 5 6 7 8 9 10

4. Do you have pain when doing a full weight bearing lunge?

Strong severe pain ☐☐☐☐☐☐☐☐☐☐☐ No pain **POINTS** ☐
0 1 2 3 4 5 6 7 8 9 10

5. Do you have problems squatting?

Unable ☐☐☐☐☐☐☐☐☐☐☐ No problems **POINTS** ☐
0 1 2 3 4 5 6 7 8 9 10

6. Do you have pain during or immediately after doing 10 single leg hops?

strong severe pain/unable ☐☐☐☐☐☐☐☐☐☐☐ No pain **POINTS** ☐
0 1 2 3 4 5 6 7 8 9 10

^ Figure 8. VISA-P.

7. Are you currently undertaking sport or other physical activity?

0	☐	Not at all	POINTS ☐
4	☐	Modified training ± modified competition	
7	☐	Full training ± competition but not at the same level as when symptoms began	
10	☐	Competing at the same or higher level when symptoms began	

8. Please complete EITHER A, B or C in this question.

- If you have **no pain** while undertaking sport please complete **Q8a only.**
- If you have **pain while undertaking sport but it does not stop you** from completing the activity, please complete **Q8b only.**
- If you have **pain that stops you from completing sporting activities**, please complete **Q8c only.**

8a. If you have no pain while undertaking sport, for how long can you train/practise?

0	☐	NIL	POINTS ☐
7	☐	1-5 mins	
14	☐	6-10 mins	
21	☐	7-15 mins	
30	☐	> 15 mins	

8b. If you have some pain while undertaking sport, but it does not stop you from completing your training/practice, for how long can you train/practise?

0	☐	NIL	POINTS ☐
4	☐	1-5 mins	
10	☐	6-10 mins	
14	☐	7-15 mins	
20	☐	> 15 mins	

8c. If you have pain which stops you from completing your training/practice for how long can you train/practise?

0	☐	NIL	POINTS ☐
4	☐	1-5 mins	
10	☐	6-10 mins	
14	☐	7-15 mins	
20	☐	> 15 mins	

TOTAL SCORE: _____ /100 _____%

^ Figure 8. VISA-P.

The decline squat (figure X) is an appropriate outcome measure to monitor daily and weekly changes in pain (37). The range of the squat will improve as pain decreases throughout rehabilitation, so recording pain at a consistent knee flexion angle can increase the reliability of this objective measure.

IMAGING AND ITS ROLE IN DIAGNOSIS AND PROGNOSIS

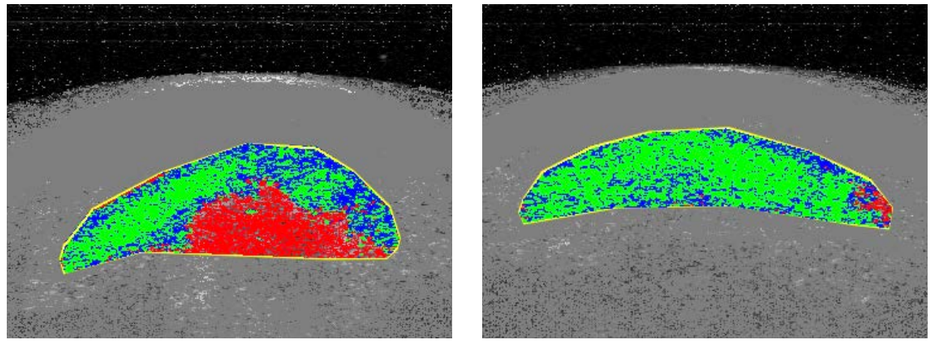
Both ultrasound imaging and magnetic resonance imaging can be used in the diagnostic work-up however, the role of imaging in the management of patellar tendinopathy should not be overestimated as it is a clinical diagnosis.

ULTRASOUND

Ultrasound in combination with colour/power Doppler can be used to visualise structural changes in the patellar tendon. (Figure 9) Characteristic findings of patellar tendinopathy are a proximal, central and posterior hypoechoic regions, tendon thickening and neovascularisation, and sometimes calcification in the proximodorsal region of the patellar tendon (38).

OTHER ULTRASOUND-BASED IMAGING TECHNIQUES

Ultrasound tissue characterisation and shearwave elastography are newer imaging techniques to assess and respectively quantify tendon structural abnormalities and elasticity. Although quantifying tendon changes seems promising for research purposes, the precise role in clinical diagnosis, monitoring improvement and screening appears to be limited (39).



^ Figure 9. UTC axial image of an abnormal patellar tendon at the inferior pole. Area of disorganisation is represented by the red pixels. UTC axial image of a normal patellar tendon at the inferior pole. Green and blue pixels represent aligned tendon structure.

MRI

The most characteristic MRI finding is focal T2 hyperintensity within the proximal tendon involving the central third of the tendon, medial tendon abnormality can also be present. In addition, an indistinct posterior tendon border may also be seen and oedema may be present within the adjacent Hoffa's fat pad.

DIAGNOSIS AND PROGNOSIS BASED ON IMAGING

Although ultrasound and MRI can clearly show focal patellar tendon abnormalities, they should not be considered a gold standard for the diagnosis of patellar tendinopathy. The prevalence of imaging abnormalities in sporting populations is high, and ultrasound and MRI abnormalities are not always associated with pain and loss of function. The role of serial imaging to monitor change in symptoms can also be debated, as symptoms and function often improve without corresponding changes in pathology on imaging. With these limitations in mind, imaging should not be used to confirm the diagnosis of patellar tendinopathy or monitor improvement but rather to rule out other coexisting pathology and assist the clinical reasoning process and differential diagnosis.

SCREENING

Tendon abnormality on imaging confers an increased risk of symptoms compared to tendons that are normal on imaging. In professional football players, an association has been found between ultrasound detected patellar tendon abnormalities at the beginning of the season and an increased risk of developing patellar tendon pain in-season (40). Players with patellar tendon abnormalities had 45% risk of developing symptomatic patellar tendon pathology, compared to 3% in players with no abnormalities (40). A cohort study with a 24 month follow up of professional ballet dancers showed that the presence of focal hypoechoic changes (not tendon diameter) was associated with the development of future tendon-related disability (41). Conversely in Australian football players, Docking et al showed that pre-season imaging was not able to predict the development of symptoms in-season, whereas simply asking whether the player had symptoms previously demonstrated greater predictive value (42). These findings suggest that imaging and history of symptom screening of players at the start of the season may be of use in identifying those who are at higher risk of developing tendon-related disability. This may be helpful to develop personalised modifications of training and match load or other targeted preventative interventions.

DIFFERENTIAL DIAGNOSIS

Anterior knee pain is a common problem in athletes and may present a significant clinical challenge to distinguish between a number of pain-producing structures including the bursae, fat pad, plica or patellofemoral joint (Figure 10).

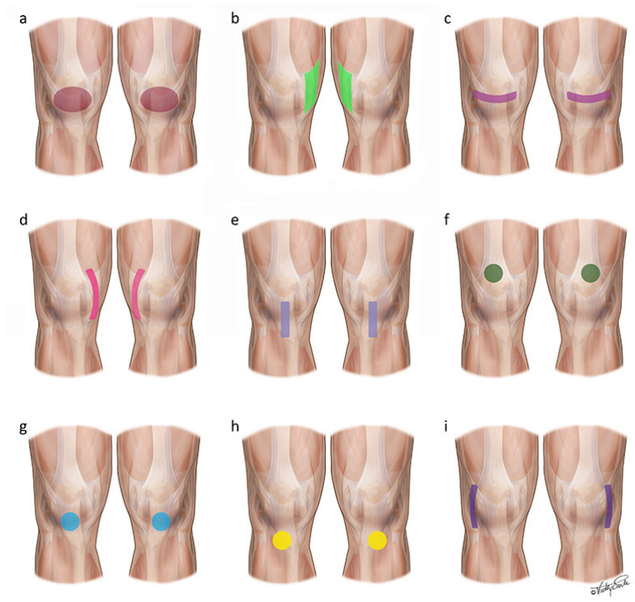


Figure 10. Patellar pain map. Localised pain indicates tendon, diffuse pain indicates patello-femoral joint or other source of pain.

a) Patellofemoral joint.
b) Patellofemoral joint.
c) Patellofemoral joint.
d) Patellofemoral joint.
e) Patellofemoral joint.
f) Quadriceps tendon.
g) Patelllar tendon.
h) Patellar tendon.
i) Patellofemoral joint.

Note: Pain locations are indicative of the patient reported location of pain, not palpation pain.

PATELLOFEMORAL PAIN SYNDROME

The patellofemoral joint is a common cause of anterior knee pain in athletes. Patellofemoral pain syndrome (PFPS) is primarily a diagnosis of exclusion, as there are no clear sensitive and specific clinical tests to establish the diagnosis. Localised osteochondral changes of the inferior region of the patella or of the trochlea may contribute to PFPS. These abnormalities can be visualised using MRI but may not be the cause of symptoms.

Patellofemoral-related pain is generally located diffusely around the patella or across the region inferior to the patella (diffusely over the tendon), in contrast to the localised, inferior pole pain typical of patellar tendinopathy. Athletes with PFPS often report aggravation of symptoms with activities that create low tendon load, such as prolonged sitting, walking or cycling. Reduction of pain when using patellofemoral taping (Figure 11) with provocative maneuvers, such as performing a lunge or a squat, may assist in confirmation of PFPS.

Examination findings that can differentiate the two conditions include analysis of hopping technique, where those with PFPS may have poorer hip control and more knee flexion. The decline squat pain in PFPS results in lower levels of pain in a deeper range than patellar tendinopathy.

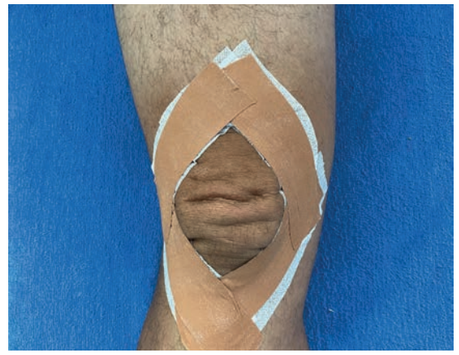


Figure 11. Patellofemoral joint diamond taping.

PRE– AND INFRAPATELLAR BURSITIS

The most commonly affected bursa is the pre-patellar bursa (Figure 12). Pre-patellar bursitis presents as a superficial swelling on the anterior aspect of the knee and can occur after direct trauma or kneeling for extended periods. Infective bursitis can occur if a wound exists and requires immediate medical management. An infra-patellar bursitis near the tibial insertion of the patellar tendon can also mimic patellar tendinopathy. However, pain from this presentation is typically more variable in nature and location compared to patellar tendinopathy. The history and examination will be helpful to diagnose these conditions, imaging may assist if the diagnosis is unclear.

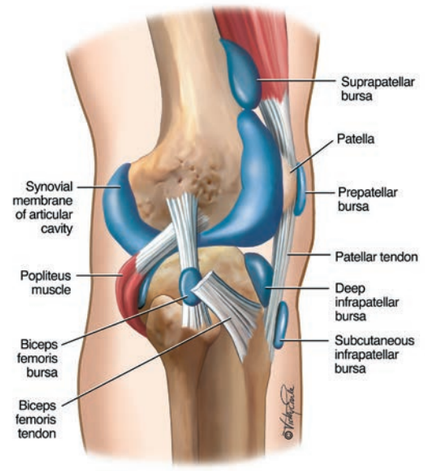
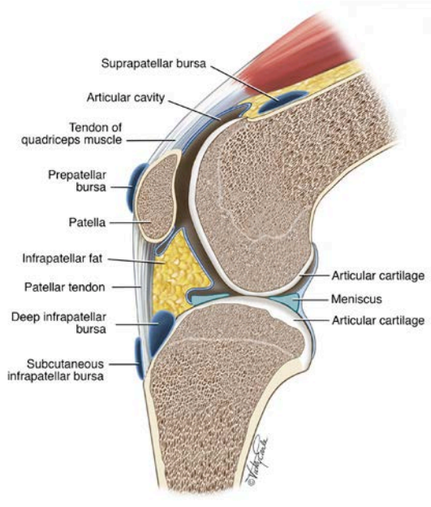


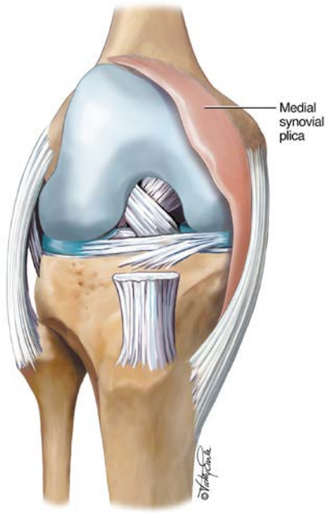
Figure 12. Knee bursa.



FAT PAD SYNDROME

Hoffa's fat pad (Figure 13) can become irritated and swollen with localised tenderness following hyperextension trauma to the knee, or after repetitive end of range knee extension. In contrast to patellar tendinopathy, pain is more diffuse and located in the anterior inferior to the knee. The pain is load dependent and is felt during end-range extension, or with palpation of the fat pad.

Figure 13. Knee cross section.



PLICA

Although still a matter of considerable debate, synovial plica (Figure 14) may cause sharp anterior knee pain and a snapping sensation (43). Sometimes a tender thickened band is palpable at the medial edge of the patellofemoral joint, and MRI can assist in the differentiation from patellar tendinopathy. It mimics quadriceps tendinopathy more than patellar tendinopathy because of its superomedial position.

< **Figure 14.** Synovial plica.

OTHER

Tumour, infections (especially in the prepatellar bursa), and referred pain from the hip (Perthes' disease, slipped capital femoral epiphysis in adolescents) can on rare occasions cause anterior knee pain.

OTHER TENDINOPATHIES ASSOCIATED WITH THE PATELLAR TENDON

Less common but also bothersome is patellar tendon pain located at the proximal patella (quadriceps attachment) or distal insertion of the tendon. Mid- or whole-tendon patellar tendinopathy is rare and generally the result of a direct blow, and nearby structures can also be injured with this mechanism (44). Pain location and history will be helpful to differentiate these conditions from proximal patellar tendinopathy.

QUADRICEPS TENDINOPATHY

Tendinopathy of the extensor mechanism of the knee can also occur at the quadriceps tendon. It is characterised by pain at the superior and usually the central to lateral margin of the patella. It is aggravated by a deep squat, where the tendon becomes compressed against the femoral condyle. This is more common in older athletes and seems to be related to activities requiring deep knee flexion such as weightlifting, however, can also occur in sports if an athlete uses deep knee flexion when changing direction and decelerating. The management is similar to patellar tendinopathy with the avoidance of compressive loads in deeper knee flexion until the tendon is tolerant to these loads.

DISTAL PATELLAR TENDINOPATHY

In younger athletes (10-15 years old) repetitive or excessive traction of the patellar tendon attachment to the tibial tuberosity can result in Osgood-Schlatter disease with local swelling and pain. Management of adolescent Osgood-Schlatter disease is similar to management of all tendinopathies, with load management and exercise to strengthen and improve power being the cornerstone of treatment. Decreasing load is more critical in the early and painful stages, but as with all tendinopathies complete rest is not recommended.

Changes in both the tendon and bone and can persist into adulthood. Overload during sport can aggravate symptoms, with distance running or prolonged kneeling appearing to be provocative from a clinical perspective.

MANAGEMENT OF INITIAL PRESENTATION

The first step in managing a player with patellar tendinopathy is to determine whether they can continue to train and play, or whether symptoms and performance impairment requires withdrawal from sport participation. Assessing irritability is a fundamental part of managing patellar tendinopathy and involves determining the duration of symptom aggravation following loading. Studies have suggested that up to 24 hours of pain provocation after energy-storage activities may be acceptable during rehabilitation. Accordingly, "irritable" tendon pain is considered to be pain lasting more than 24-hours following loading, and "stable" tendon pain as pain which settles within 24-hours (45). Usually, the aggravation of symptoms occurs during loading activities, such as walking downstairs, lunging or when performing a decline squat. Pain level can be rated on an 11-point numeric rating scale, where 0 is no pain and 10 is the worst pain imaginable.

Assessment of the athlete with patellar tendinopathy pain requires analysis of muscle bulk, strength, endurance and power, as well as assessment of the function of the musculotendinous unit in the context of the entire kinetic chain. Profound deficits in any of these areas may compromise the ability of the athlete to train and play and may compromise their overall performance. If deficits are particularly severe, withdrawal from sports participation may be required so that targeted rehabilitation may be undertaken.

REHABILITATING THE PLAYER

Once the decision is made that the player will cease training and playing, the emphasis is placed on restoring function to the tendon, muscle, kinetic chain and the neural system. Rehabilitation of patellar tendinopathy in young agile players requires a substantial strength program to be undertaken, in order to adequately prepare the tendon to withstand the high tendon loads required for sport.

^ **Figure 15.** Single leg extension.

^ **Figure 16 and 17.** Single leg standing and seated calf raise.

A progressive four stage loading has been shown to give better outcome that an eccentric program (46). The evidence for the first stage of the program (isometric exercise) is strongest in the patellar tendon so these can be utilised, both to reduce cortical inhibition as well as for pain control (47). Developing adequate strength in patellar tendinopathy requires a formal gym program as this is a condition of young jumping (mostly) men. Leg extensions, leg press, seated and standing calf raises and hip extensor exercises are essential (Figures 15-17), with sessions ideally completed three times per week. Other muscles are targeted if shown to be deficient in the assessment. All exercise should be completed single leg and each leg must be loaded independently. Seated calf raises selectively load the soleus muscle, an essential contributor to deceleration and change of direction movements. Once a strength base has been established, a functional strength endurance program including exercises such as stair climbing and walk lunges can be commenced.

When strength and endurance are adequate, the rate of loading is increased, with exercises such as low-level hopping, skipping, jumping and deceleration introduced every 2-3 days. These are progressed in intensity and quantity as the tendon becomes tolerant of these loads. Sport specific loading is then increased with the use of familiar football drills. The aim of these exercises is to increase the quantity and intensity of the earlier exercises.

RETURN TO PERFORMANCE CONTINUUM

RETURN TO PLAY

Training is increased until the player can sustain repeated deceleration and change of direction loads that mimic game play without an increase in symptoms the following day. The higher load skills (decelerations and change of direction) should be quantified and graduated in volume across this training block. Generally, other rehabilitation-based drills are reduced to avoid overload. At all times during rehabilitation the player must maintain strength using a gym program twice weekly.

IN SEASON MANAGEMENT

Improving and then maintaining strength is a key management strategy, and therefore a consistent gym program must be maintained throughout the season. Limiting provocative loads during training such as agility drills may be required to reduce the tendon load. Decreasing the frequency of on-field training to maximum of 3 times a week can also be of benefit in this stage. Isometrics before training and games can help in both pain and strength. These can be gym-based leg extensions or a Spanish squat (Figure 18) if gym access is limited (48). Adjunct treatments that enable the player to more effectively load the tendon during rehabilitation are encouraged, and these may be individualised. However, adjunct treatments aimed directly at tendon pathology are usually invasive and are discouraged.

< **Figure 18.** Spanish squat.

ADJUNCTS

A randomised controlled trial examining the effect of extracorporeal shockwave therapy has been shown to provide no benefit over placebo treatment in the in-season management of jumping athletes with patellar tendinopathy (49). A further randomised controlled trial compared the effectiveness of focused shockwave therapy and radial shockwave therapy, finding no significant difference between groups (50). Interestingly, both of these groups improved significantly, however it was concluded that this improvement was unlikely to be clinically worthwhile (50). A further study compared an eccentric exercise protocol with or without the addition of three shockwave sessions, and found no additional benefit (51).

The effect of using a patellar strap or sports tape for patellar tendon pain has also been investigated (52). A randomised controlled trial compared the effect of patellar taping compared to a placebo taping method and found that both patellar taping and the use of a patellar strap reduced pain in the short-term, however neither method was more effective than placebo (52).

Various injection therapies have been proposed to be of benefit in patellar tendinopathy. A systematic review of injection therapies found that steroid injections resulted in a decrease in pain in the short-term, but a relapse of symptoms was found with longer term follow-up (53). Other injection therapies were also analysed, but there was insufficient evidence available to confer superiority of any these treatments over the other or over placebo (53).

Summary:

- Patellar tendinopathy is an overuse injury that is characterised by pain during high tendon load activities such as jumping, deceleration and change of direction.
- The defining clinical features of patellar tendinopathy are pain localised to the inferior pole of the patella, and load-related pain that increases with the demand on the knee extensors, especially in activities that store and release energy in the patellar tendon.
- The incidence of patellar tendinopathy varies seasonally, with the highest proportion of injuries occurring after a period of unloading such as in the pre-season and after a mid-season break.
- While pathology in other tendons has been linked to accumulation of pathology throughout the lifespan, patellar tendinopathy is highly prevalent in young jumping athletes between the ages of 14-18 years, and the development of patellar tendinopathy after adolescence is less common.
- The single leg decline squat is a useful clinical test to increase the likelihood of a patellar tendinopathy diagnosis and to assess irritability. The test reproduces localised tendon pain early in knee flexion range and can be used to monitor changes in pain throughout rehabilitation. The range of the squat will improve as pain decreases, so it is recommended that pain be assessed at a consistent angle to improve the reliability of this objective measure.
- Patellar tendinopathy is a clinical diagnosis and imaging is not required as part of the diagnostic process. However, imaging can be useful to define co-existing pathology.
- Patellofemoral joint pain is a common differential diagnosis. Patellofemoral-related pain is generally located more diffusely around or inferior to the patella and is often aggravated by low tendon load activities such as prolonged sitting, walking or cycling.

Clinical Implications:

- Although many athletes are still able to train and play despite patellar tendon pain, their performance may be negatively impacted even if no time-loss is registered. Players will often report a change in function and performance, noticing their speed and agility are decreased, due to both pain and dysfunction. Deficits in performance are crucial when deciding to either manage an athlete in-season or to withdraw them from competition for rehabilitation.
- Faster and more agile athletes may be predisposed to the development of patellar tendinopathy, as they are more competent in storing and releasing energy in the patellar tendon.
- It is important to examine the entire kinetic chain for deficits. For example, poor dorsiflexion range of movement has been linked with patellar tendinopathy, likely due to a decrease in shock absorption at the ankle during landing that leads to increased knee loading during takeoff.
- A higher incidence of patellar tendinopathy has been observed during pre-season or following a mid-season break. This higher incidence is likely related to more rapid changes in training load. Appropriate monitoring of an athlete's training load is therefore vital in order to minimise these rapid load fluctuations.
- A misleading feature for many athletes is the so called "warm-up" phenomenon, where the patellar tendon pain may improve during training. They often continue to exercise and misunderstand the importance of this initial pain. This may lead to a delay in diagnosis and progression of pathology prior to intervention.
- The development of sufficient strength to rehabilitate patellar tendinopathy requires a formal gym program. Exercises including leg extensions, leg press, seated and standing calf raises and hip extensor exercises are vital. Sessions should ideally be completed 2-3 times per week. When strength and endurance are adequate, the rate of loading can be increased, with low level hopping, skipping, jumping and deceleration drills introduced every 2-3 days.
- Isometrics can be useful before training and games to reduce pain and cortical inhibition. Leg extensions can be used if a gym is available, or alternatively the Spanish squat can be used if gym access is limited.

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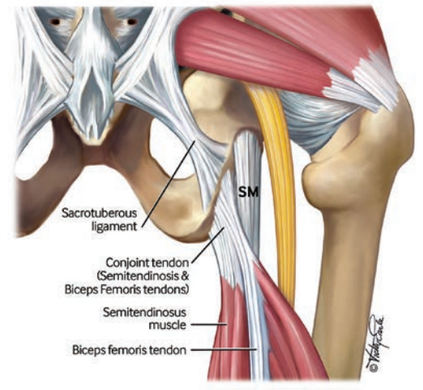
3.3. HAMSTRING TENDON INJURIES

EPIDEMIOLOGY IN FOOTBALL

Proximal hamstring tendinopathy is a cause of persistent ischial tuberosity pain (1, 2). It is difficult to estimate its incidence as most studies on hamstring injuries do not report tendon and muscle strain injuries separately. A single study found that the incidence of proximal hamstring tendinopathy in men's professional football (3) was 1.5 (95% CI 0.5-3.2) per 100 athletes, per season in professional football, and 0.4 (95% CI 0.1-1) in youth football. In women's football, the incidence was slightly lower, with 1.2 (95% CI 0.2-3.4) and 0 injuries (95% CI 0-0.6) per 100 athletes, per season, in professional and youth football respectively.

PATHO-ANATOMY OF THE HAMSTRING TENDONS

The proximal tendons of semimembranosus and biceps femoris long head are implicated in proximal hamstring tendinopathy. The semimembranosus tendon appears to be most vulnerable to compression in positions of hip flexion and adduction due to its deep and lateral origin on the ischium (Figure 1) (1). Compression might also occur in these same hip positions, where semimembranosus crosses under the common tendon to the anterolateral portion of the ischial tuberosity (4, 5). The semimembranosus may be more susceptible to injury after a period of de-loading, as it atrophies at a significantly faster rate than its synergists (6). Returning to high load training after a period off could place the this tendon at higher risk of tendinopathy. Tendinopathy of the three hamstring tendons at the knee, including ruptures of the distal semitendinosus and biceps tendon, have been recorded but are less frequent (7-9).



^ **Figure 1.** Sciatic nerve proximity hamstring origin.

HAMSTRING MUSCLE FUNCTION

The hamstring muscle group acts as both an extensor of the hip and flexor of the knee joint (10). The biceps femoris can also assist in external rotation of the knee (10). In upright running the hamstrings eccentrically decelerate knee extension in the terminal swing phase (11). Peak force occurs in late swing, with a second peak reported in the early stance phase of running (12). In late swing and early stance (Figure 2) phase during sprinting, tensile forces on the hamstring group are 8-10 times body weight (12, 13). Of this, around 50-60% of tensile force, negative work and peak power absorption are attributed to the semimembranosus, with biceps femoris long head the next highest at 20-30%. When kicking a ball, forces in the hamstring group in the kicking leg are lower than during sprinting, as this is mainly a trunk and hip flexor dominated activity (14). Higher hip and knee moments are found in the stance leg during kicking (14).

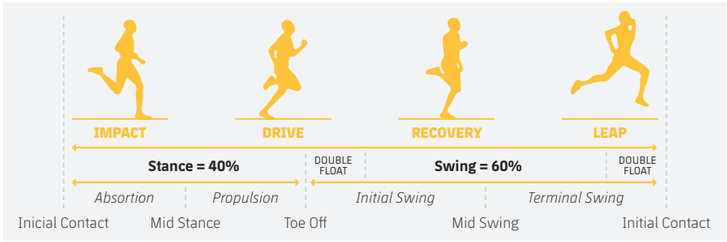


Figure 2. Running Gait. >

HAMSTRING TENDON LOAD

In the stance leg in maximal kicking (Figure 3), the pelvis is anteriorly rotated (anteverted) in the early phase and notably retracted by more than 30 degrees in the axial plane (14). Initiation of the kick is a result of posterior rotation (retroversion) of the pelvis, and on follow through the pelvis follows the leg and is protracted by up to 30 degrees. At the same time, the stance leg is adducted on the pelvis creating a combination of compressive and tensile loads on the semimembranosus and biceps femoris tendons and combinations of these sagittal, coronal, pelvic anteversion or axial rotation should be considered by the clinician in assessing contributing factors (15, 16).

The hamstring origin may be subject to higher energy storage loads in greater hip or trunk flexion. Rapid decelerations or forward movement of the trunk on the hip have the potential to place considerable tensile loads and a high rate of loading on the proximal tendon. In pure sagittal trunk flexion or exaggerated anterior pelvic tilt (1), the origin of the biceps femoris long head / semitendinosus tendon predominantly experiences a combination of tension and compression. Dropping of the contralateral pelvis in the coronal plane appears to chiefly affect the semimembranosus tendon due to its lateral origin on the ischial tuberosity. Hamstring muscle tensile loads during running and kicking have been described (12, 14, 17); however biomechanical modelling of factors such as increased trunk on hip flexion, pelvic anteversion or lateral pelvic drop and their effect on compression at the hamstring tendon origin has not been performed.

^ **Figure 3.** Maximal kicking.

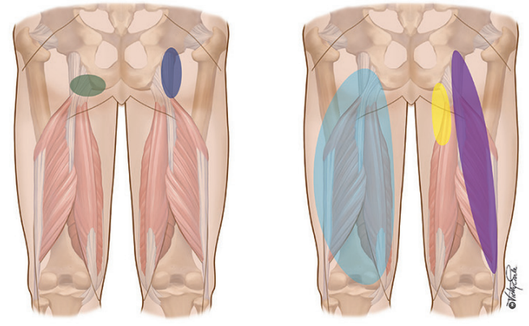
There is a reasonable demand on hamstring length in kicking, tackling (Figure 4) and accelerating with a flexed trunk. A widely recognised risk factor for tendinopathy is increased load due to high physical demand during training and matches (18), especially when there is a mismatch between tendon load and tendon capacity (19). If flexibility of the hamstring unit fails to meet these functional demands, it may increase load on the hamstring origin. Quantification of these demands and evidence that flexibility is protective for proximal tendinopathy are lacking.

^ **Figure 4.** Tackling.

CLINICAL PRESENTATION OF PROXIMAL HAMSTRING TENDINOPATHY

HISTORY

Proximal hamstring tendinopathy is a clinical diagnosis from the history and confirmed with pain provocation tests. The condition is characterised by deep buttock pain, often described as a dull ache, that is well localised to the hamstring origin (1) or with minor radiation (Figure 5). The typical onset is atraumatic, with symptoms arising in the hours after, or in the day following, high-tendon load activities. Classically the history describes a spike in tendon loading such as a sharp increase in training intensity or the introduction of unaccustomed drills that involve deep hip flexion, usually body on leg flexion.

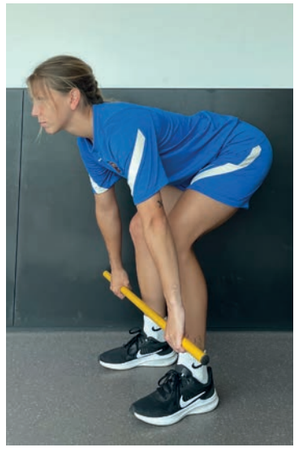


^ **Figure 5.** Hamstring pain map. Localised hamstring tendon pain (green circle) indicates tendon pain, more diffuse pain indicates other sources. Note: Pain locations are indicative of the patient reported location of pain, not palpation pain.

BEHAVIOUR OF SYMPTOMS

Symptoms are often worse when commencing activity after a period of inactivity, during and after sitting or in the morning, and ease with movement, but are aggravated again towards the end of athletic activity (1, 20). Running ability, particularly sprinting and acceleration are often impaired (Figure 6) (21), with symptoms worse when running at faster speeds (2) and during rapid change of direction (1). Pain is also provoked by activities that involve deep hip flexion range of motion (e.g. sitting, squat, deadlift, lunge) and stretching of the hamstring (1, 2, 22). In contrast, activities that involve minimal hamstring function or compression, such as standing or lying, are rarely painful (1).

Figure 6. Sprinting. >



^ Figure 7. Barbell back squat, lunge, deadlift.

CLINICAL TESTS

Pain provocation tests are used in the physical examination. Three passive stretch tests (the Puranen-Orava, bent-knee stretch test and modified bent-knee stretch test), have demonstrated high sensitivity and specificity compared to a localised hamstring origin pain and abnormal magnetic resonance imaging (MRI) as reference standard (23). Two active tests, performed in sitting, that involve resisted knee flexion in inner (A-90 test) and outer (A-30 test) range have also demonstrated high rates of sensitivity and specificity in a mixed cohort of proximal hamstring tendon injuries (24). Despite the high specificity of these tests, other co-pathologies or conditions that may be responsible for the symptoms should be considered as there are no gold standard diagnostic criteria for proximal hamstring tendinopathy.

Graded loading tests for the proximal hamstring tendon involve progressive loading of the proximal hamstring tendon, with higher loads being more provocative (5). Examples of lower level pain provocation tests include a unilateral hamstring bridge with the knee at 90º flexion (Figure 8), or a unilateral long lever bridge (knee at 0-20 deg flexion). Higher level provocation is achieved with an arabesque or dead lift. A more functional test may also be a rapid forward lunge with the trunk in 20-30 deg of flexion as the speed of the movement increases tendon load (Figure 9). These tests can be used as a diagnostic tool and as an outcome measure when applied 24 hours following an exercise bout to assess the response to an exercise intervention.

^ Figure 8. Single leg bridge.

The diagnostic value of proximal hamstring tendon palpation remains controversial, where the large gluteus maximus covers the hamstring origin. An absence of pain on palpation in gluteus medius/minimus is useful in ruling out gluteal tendinopathy in those with MRI positive tendon abnormality (25), the pain response to palpation in proximal hamstring tendinopathy seems more variable (21). An absence of pain on local palpation should not be used to rule out proximal hamstring tendinopathy.

^ Figure 9. Lunge test.

PHYSICAL EXAMINATION DEFICITS (STRENGTH, FLEXIBILITY, MOVEMENT PATTERNS)

Examination often reveals deficits in knee flexor and hip extensor muscle strength (21, 22). Weakness of the abdominals, gluteus maximus and gluteus medius have also been reported (22, 26). It is unknown if these deficits are present prior to the condition, or whether they are a consequence of unloading because of tendon pain.

Hamstring flexibility varies considerably in athletes who present with proximal hamstring tendinopathy (27). Shortness of the hamstring muscles that fail to meet the range requirements for football, such as during slide tackles and overhead kicking, may place increased strain on the proximal origin. Range of motion deficits in the joints of the lower extremity have the potential to increase demands on the hip and should be considered (e.g. loss of hip flexion or loss of knee flexion range of movement leading to increased flexion at the trunk or hip) (28).

Running biomechanics should be examined by assessing for overstriding, increased anterior pelvic tilt and excessive forward trunk lean, as these impact loads on the hamstring (1, 29). Some of these can be viewed during both assessment of single leg squat and sport-specific activities, and should assessed in frontal/coronal and sagittal planes. Assessment of running technique using video analysis is useful in resistant cases and in elite level athletes.

IMAGING AND ITS ROLE IN DIAGNOSIS AND PROGNOSIS

Tendinopathy is a clinical diagnosis and the diagnosis of proximal hamstring tendinopathy does not always require imaging. Where the onset of symptoms are traumatic, or not resolving, imaging is warranted to screen for tendon rupture, or avulsion in the skeletally immature. Magnetic resonance imaging (MRI) may assist differential diagnosis, or contribute to reasons for lack of progress with rehabilitation. Ultrasound can provide a dynamic image during active and passive movements to provide valuable information (for example to identify tethering of sciatic nerve to the proximal hamstring) (30).

Imaging findings are not linked to symptoms (31, 32). A review of 506 MRIs of the proximal hamstring complex in 253 asymptomatic participants found 65% of this older (median age 60) population demonstrated abnormalities (32), another study found changes in 90% of participants (mean age not reported) (31). Magnetic resonance imaging abnormalities in asymptomatic younger populations are less prevalent, a small study found proximal hamstring tendon changes in 25% (n=16) below 45 years. Exposure to loads in sport may increase the prevalence of tendon changes. One study showed that an increase in tendon size, peritendinous T2 signal with a distal feathery appearance, and ischial tuberosity oedema were more frequent in symptomatic participants (31). Further research in this field is clearly required (31).

Normal imaging appearance of the proximal hamstring tendon complex is often used to rule out tendinopathy and suggest other structures are the source of pain. Normal imaging evaluation was present in 23% of MRI and 65% of ultrasound studies of participants presenting with pain in the region (33), a proportion of whom subsequently derived benefit from peritendinous corticosteroid injection. If proximal hamstring tendinopathy is suspected but imaging is normal, additional clinical and imaging examination should be considered to exclude other sources of pain.

The presence of tendon pathology on imaging is a risk factor for the development of tendinopathy (34). The high rate of tendon abnormality seen in the asymptomatic population suggests that the hamstring tendons are able to adapt (31, 32). Most tendons with pathology can adapt to achieve similar levels of aligned fibril structure to healthy controls (35). The 20% of tendons that did not adapt were not more likely to develop symptoms (36) and monitoring tendon recovery using imaging modalities is not better than a symptom and function-based approach. Patient-reported outcome measures that monitor symptoms and function, such as the Victorian Institute of Sports Assessment - Hamstring (VISA-H) questionnaire (Figure 10) are preferable (37).

VISA-H

DATE __/__/____ INITIAL ASSESSMENT ☐ DISCHARGE ASSESSMENT ☐
NAME _____ SURNAME _____ AGE ___ WEIGHT ___ HEIGHT ___
SPORT _____ TEAM _____ PHYSICIAN _____

1. For how many minutes can you sit/can your drive a car pain free?

0 mins ☐☐☐☐☐☐☐☐☐☐☐ 100 mins POINTS ☐
0 1 2 3 4 5 6 7 8 9 10

2. How much pain do you have during or immediately after stretching your posterior thigh/hamstring (keeping knee straight)?

Strong severe pain ☐☐☐☐☐☐☐☐☐☐☐ No pain POINTS ☐
0 1 2 3 4 5 6 7 8 9 10

3. How much pain do you have during or immediately after normal running?

Strong severe pain ☐☐☐☐☐☐☐☐☐☐☐ No pain POINTS ☐
0 1 2 3 4 5 6 7 8 9 10

4. How much pain do you have during or immediately after sprinting?

Strong severe pain ☐☐☐☐☐☐☐☐☐☐☐ No pain POINTS ☐
0 1 2 3 4 5 6 7 8 9 10

5. How much pain do you have during or immediately after a full weight-bearing lunge?

Unable ☐☐☐☐☐☐☐☐☐☐☐ No problem POINTS ☐
0 1 2 3 4 5 6 7 8 9 10

6. How much pain do you have during or immediately after lifting an object from the floor (keeping knee straight)?

Unable ☐☐☐☐☐☐☐☐☐☐☐ No problem POINTS ☐
0 1 2 3 4 5 6 7 8 9 10

^ **Figure 10.** VISA-H.

7. Are you currently undertaking sport or other physical activity?

0	☐	Not at all
4	☐	Modified training ± modified competition
7	☐	Full training ± competition but not at the same level as when symptoms began
10	☐	Competing at the same or higher level when symptoms began

POINTS ☐

8. Please complete EITHER A, B or C in this question.

- If you have **no pain** while undertaking sport please complete **Q8a only.**
- If you have **pain while undertaking sport but it does not stop you** from completing the activity, please complete **Q8b only.**
- If you have **pain that stops you from completing sporting activities**, please complete **Q8c only.**

8a. If you have no pain while undertaking sport, for how long can you train/practise?

0	☐	0-20 mins
7	☐	21-40 mins
14	☐	41-60 mins
21	☐	61-90 mins
30	☐	> 90 mins

POINTS ☐

8b. If you have some pain while undertaking sport, but it does not stop you from completing your training/practice, for how long can you train/practise?

0	☐	0-15 mins
4	☐	16-30 mins
10	☐	31-45 mins
14	☐	46-60 mins
20	☐	> 60 mins

POINTS ☐

8c. If you have pain that stops you from completing your training/practice, for how long can you train/practise?

0	☐	NIL
4	☐	1-10 mins
10	☐	11-20 mins
14	☐	21-30 mins
20	☐	> 30 mins

POINTS ☐

TOTAL SCORE: _____ /100 _____%

^ Figure 10. VISA-H.

DIFFERENTIAL DIAGNOSIS

Many structures can refer pain to the buttock region including somatic referral from the lumbar spine, sacroiliac and hip joint, as well as radiculopathy and peripheral nerve entrapments (Tables 1-3). Referred pain typically results in a more diffuse area of symptoms than that found in proximal hamstring tendinopathy and aggravating factors are often less specific to high hamstring tendon loads in hip flexion (1). Co-morbidities such as spondyloarthropathy may be present in the player presenting repeatedly with low back and hip stiffness as well as proximal hamstring tendinopathy (38).

HAMSTRING MUSCLE-TENDON-BONE COMPLEX RELATED CAUSES	
Acute onset	Indirect muscle injury/muscle strain Direct muscle injury/muscle contusion Tendon avulsion injury Proximal semimembranosus rupture/partial rupture Ischial tuberosity apophysis avulsion fracture Reactive tendinopathy • Proximal hamstring tendinopathy • Distal biceps femoris tendinopathy • Distal semimembranosus/semitendinosus tendinopathy
Gradual or insidious onset	Chronic tendinopathy • Proximal hamstring tendinopathy • Distal biceps femoris tendinopathy • Distal semimembranosus/semitendinosus tendinopathy Traction apophysitis of the ischial tuberosity Myositis ossificans

^ Table 1. Differential diagnosis of gluteal and upper hamstring pain (39).

CAUSES FROM OTHER ANATOMICAL STRUCTURES	
Neural	Radiculopathy Peripheral nerve entrapment • Posterior cutaneous nerve of the thigh • Sciatic nerve • Inferior cluneal/pudendal nerve
Vascular	Iliac artery endofibrosis Thrombophlebitis Deep venous thrombosis Post thrombosis syndrome
Bone	Bone tumours Femoral/ischial/sacral stress reaction or fracture
Other muscle injury	Adductor magnus Gastrocnemius medial/lateral head
Joints	Referred pain from • Sacroiliac joint • Hip joint • Knee joint
Bursitis	Semimembranosus Ischiogluteal
Other	Chronic compartment syndrome of the posterior thigh Ischiofemoral impingement syndrome

^ **Table 2.** Differential diagnosis of gluteal and upper hamstring pain.

DIAGNOSIS	KEY FEATURES
Proximal hamstring tendinopathy	Pain during or after activities with hip flexion movements, particularly body on leg flexion Pain during prolonged sitting especially on a hard surface or in deep hip flexion (car) Localised tendon pain reproduced on resistance test
Sciatic nerve irritation	Can be localised or diffuse pain radiating in posterior thigh Pain during passive hip adduction Abnormal slump test/sitting piriformis test
Piriformis syndrome	Pain in the gluteal area with or without radiation in the posterior thigh Pain on resisted external rotation or passive internal rotation in sitting (40) Pain on piriformis muscle palpation
Ischiogluteal bursitis	Mainly pain during sitting Pain on localised palpation of the ischial tuberosity Ultrasound or MRI confirming diagnosis
Referred pain from the lumbar spine	Diffuse pain in the posterior thigh and/or lower leg Absence of injury pain during hamstring resistance tests and/or localised palpation
Ischiofemoral impingement	Pain on palpation of the quadratus femoris muscle Pain on passive external rotation with the hip in neutral-extension position MRI confirming diagnosis
Apophysitis or avulsion	Adolescent athlete Injury related to overuse (apophysitis) or an acute trauma (bony avulsion injury) X-ray confirming diagnosis (bony avulsion injury)
Stress fracture - posterior pubic, ischial ramus or sacral	History of overuse Female athletes at higher risk Pain on palpation over the posterior pubic or ischial ramus
Metabolic disorder, rheumatic disease or tendon abnormalities induced by medications	No response to usual care Family history of hypercholesterolaemia, diabetes, gout or seronegative conditions Use of specific medications (quinolones, statins)

^ **Table 3.** Key features of differential diagnoses of pain in the buttock region.

Several symptoms of sciatic nerve irritation, including pain with sitting and exacerbation with hip flexion and knee extension, are similar to those of proximal hamstring tendinopathy, and can make differential diagnosis challenging (41). The two pathologies may also coexist (24). Cadaveric studies have highlighted the close relationship between the sciatic nerve and proximal hamstring tendon complex, with the sciatic nerve on average 1.2cm +/-0.2cm from the most lateral aspect of the ischial tuberosity (42).

Sciatic nerve entrapment can occur at the level of the piriformis but may also occur anywhere through the buttock to below the level of the ischial tuberosity (figure 1) (1, 41). Provocation tests to identify sciatic nerve pathology in the buttock are a combination of the sitting piriformis test and the active piriformis test (40). Other tests include the straight leg raise and the slump test (1). The anatomical proximity of the sciatic nerve and the proximal hamstring complex (24) and enlargement of the proximal hamstring tendon in tendinopathy may compress the sciatic nerve, and tether the sciatic nerve (particularly the perineural structures) to the hamstring tendon (2, 21, 41, 43).

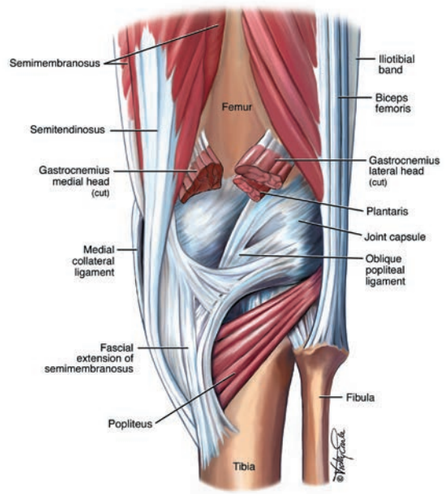
Ruptures of the proximal tendon are not common, but can occur as an apophyseal separation in adolescent football players, or tendon avulsion from overstretch due to a combined hip flexion and knee extension in older football players (44, 45). Intramuscular tendon injuries of the hamstring are distinct from injuries to the proximal tendon and present with a more variable pain location and traumatic onset (46).

Differential diagnosis should also consider stretch type hamstring injuries, resulting in full or partial tears to the semimembranosus free tendon, which have been reported in football during high kicking and sagittal splits (47). The conjoined tendon of semitendinosus and biceps femoris long head shares the ischial tuberosity with the most lateral, ischiocondylar portion of adductor magnus (48). Injury to the adductor magnus is rare (49) and they present with more medial ischial symptoms.

Other causes of buttock pain of insidious onset include ischiofemoral impingement (50, 51), stress fractures of the pubis or ischial ramus, and spondyloarthropathies such as ankylosing spondylitis (52). In the younger athlete apophyseal injuries such as avulsion fractures and apophyseal overuse injuries should also be considered.

DISTAL HAMSTRING TENDINOPATHIES

The three distal hamstring tendons of insertion are rarely affected, although cases of tendinopathy, associated bursitis or occasionally, tendon rupture are described. These presentations all appear to have overuse of knee flexion as a common feature in their development. Management of these atypical tendinopathies includes activity modification and reducing irritability, allowing symptom resolution prior to gradual reloading.



^ Figure 11. Hamtring insertion.

Semimembranosus has a complex insertion of a number of slips and a bursa that separates it from semitendinosus, the medial collateral ligament and the medial tibial plateau (Figure 11). Repetitive eccentric knee flexion is provocative (4). Isolated semitendinosus tendinopathy appears to be rare. More typically it is involved with gracilis and sartorius tendons in pes anserinus tendinopathy and/or bursitis (53), this bursitis is often seen in seronegative arthropathies. Rupture of the distal semitendinosus requires surgery, which gives better outcomes than conservative management (54).

Distal biceps femoris tendinopathy, enthesopathy and tears are described. The distal biceps femoris tendon bifurcates to accommodate the fibula collateral ligament, which because of its different echogenicity may lead to misdiagnosis on ultrasonography (55). Repeated inner range knee flexion may induce a combination of compression and friction between the two structures. Isolated tears or avulsion of the distal biceps are described predominantly through a mechanism of hyperextended knee and concurrent hip flexion (56).

MANAGEMENT OF INITIAL PRESENTATION OF PAIN

Initial presentation of proximal hamstring tendinopathy includes evaluation of intrinsic factors and documenting activity that may have contributed to the tendon overload. Clear differential diagnosis is critical as mixed presentations of tendinopathy with hip, low back pains or sciatic involvement are common.

PATIENT CENTRED TREATMENT PLANNING:

The clinician, athlete and coach should set priorities (competitions, time of year etc.) and align expectations early in management. Proximal hamstring tendinopathy management can require a lengthy rehabilitation process over several months, and there may be a need to balance rehabilitation (muscle strength and kinetic chain function), pain management and optimal training load (17).

PAIN AND LOAD MANAGEMENT:

The early focus is on symptom control by reducing excessive compressive and tensile loads. Reducing the total training sessions per week, or the volume of provocative activities within training and in the gym are both effective (57). Examples include reducing accelerations and decelerations in running, but retaining more steady state running, and reducing or removing provocative weights such as dead-lifts, weighted lunges or squats from a weights program. Adjuncts such as manual therapy including massage through hamstring muscle belly and other muscles in the region may also assist in reducing accompanying muscle tightness. Oral analgesics may be utilised to enable greater functionality.

PAIN EDUCATION AND SELF-MANAGEMENT:

Education of both the player and staff is critical to ensure they understand the nature of tendon pain (warms up/worse next day) and how to interpret changes in symptoms. This empowers the player to self-monitor and self-manage the condition. Key elements include regular pain monitoring on provocative tests the day after training, load management, and structured exercise to maintain control of symptoms. Education should also include awareness of sources of provocation, such as compression in the gym (e.g. squatting or lunging), or poorly controlled increases in volume/intensity of speed or change of direction work. Activities of daily living such as prolonged sitting associated with travel are also pertinent.

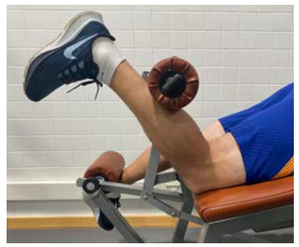
Compression in activities of daily living may be addressed through a well-placed rolled up towel to relieve direct compression of the tendon on seats. Reducing the degree of hip flexion through adjustments of the tilt and height of the chair may provide further relief.

LOAD MANAGEMENT:

Education of staff and coaches about provocative hamstring tendon loads and an optimal load window is critical to manage activity within a safe zone. Excessive load, rapid increases in load and the avoidance of prolonged periods of relatively low hamstring load are all provocative. One-off unaccustomed high intensity loads may also exacerbate the proximal hamstring tendinopathy (e.g. hard/low change of direction or acceleration drills, or big sets of deadlifts in the gym). Broad field-based training workload metrics (e.g. acute/chronic workload ratio) may not be specific enough to capture the high tensile or compression load placed on the proximal hamstring tendon.

In the weights room, it is essential to provide alternative hamstring strengthening exercises (e.g. bridges, prone hamstring curl, Nordic curls or Bosch holds instead of dead lifts or lunges) (Figure 12-14), as detraining of the hamstring group is deleterious to proximal hamstring tendinopathy and also increases hamstring muscle strain risk. It is critical to load both legs independently as bilateral exercises may provide opportunity for unloading the affected tendon.

^ **Figure 12.** Single leg bridge.



^ **Figure 13.** Single leg prone hamstring curl.

^ **Figure 14.** Nordic curl bosch hold.

^ **Figure 15.** Barbell hip thrust.

^ **Figure 16.** Sled push.

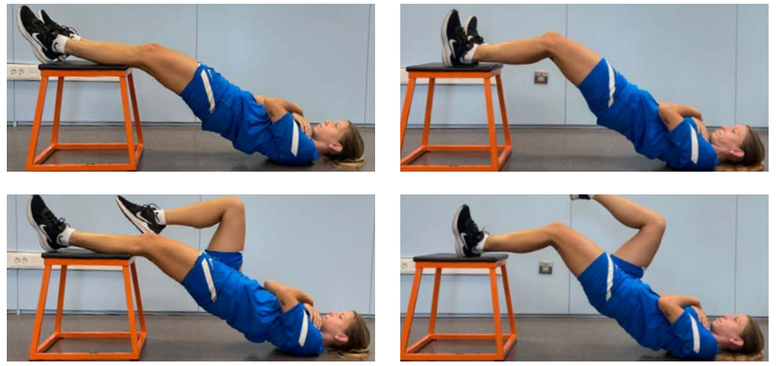
EARLY EXERCISE-BASED INTERVENTIONS – TENDON LOADING

Isometric exercises and slow isotonic exercises for pain management (58) are generally well tolerated in proximal hamstring tendinopathy if positioned in hip neutral (e.g. supine plank off roller (Figure 17), long lever bridge in minimal hip flexion, prone hamstring curl) (5). Isometric exercises have the best effect with 4-5 repetitions of 45 second holds (59). This is complemented by a heavy slow loading program (generally 2 or 3 bilateral or single leg resisted isotonic exercises) (60). Useful options here for proximal hamstring tendinopathy are leg curls, hip thrusters and Nordic hamstring curls. It is important this heavy slow loading regimen avoids deeper hip flexion range in early stages, this is progressed into greater hip flexion as irritability on compression eases (1). Early rehabilitation should target both limbs individually as sensory and motor deficits also exist on the non-injured side of patients with unilateral tendinopathy (61). This may also have the added benefit of a cross-education effect where strengthening one leg can substantially increase the strength of the other leg (62). Maintenance of some form of steady state running at lower speeds should also be tolerated in earlier stages that may enable maintenance of kinetic chain function and basic fitness.

^ **Figure 17.** Single and double leg supine plank off roller.

REHABILITATION PROGRESSION

Strengthening exercises are progressed through increasing resistance, generally with similar exercises to the early stage. Gradual introduction of deeper hip ranges as symptoms allow with exercises such as walking lunges, sideways lunges, graduated trunk flexion with sled push (Figure 16) and leg press or squat exercises. Greater single leg isolation is a key component to ensuring strength levels are normalised. Often resistance work can be progressed quite quickly, whereas increasing range into compression may be slower. Symptoms the next day and pain on a provocative test (e.g. lunge) will guide the progression of range and resistance (1). Heavy slow resistance work is generally applied three times per week (63) with aspirational targets of equalising side to side knee flexor and hip extensor scores in free weights.



< **Figure 18.** Bridge progression. Note the amount of contact between the lower leg and the box.

Top left- Double leg supported.

Top right- Double leg less Supported.

Bottom left- Single leg supoorted.

Bottom right- Single leg less supported.

Progression of running involves the graduated reintroduction of speed in steady state running and accelerations. Running drills and faster scooter work assist in this transition. It is important the speed attained and number of repetitions are quantified to enable fine tuning of workload tolerance, dependent on the 24 hour response of the hamstring tendon to its provocation test (1). During this re-introduction of speed, sessions are best limited to two sessions per week.

KINETIC CHAIN LOADING

Rehabilitation should also target the hamstring synergists (gluteus maximus, medius and triceps surae) in their role of sharing load across the kinetic chain, as well as specific individual findings – e.g. tightness of the contralateral hip flexors which has been shown to increase hamstring demand in running (64). Hamstring tendinopathy presentations will vary considerably in terms of contributing factors and are often related to individual past medical history - e.g. ankle injury with poor calf muscle capacity, ipsilateral hip stiffness (65) and reduced gluteus maximus strength which has been associated with proximal hamstring tendinopathy (66). Clinically, tendinopathy pain affects the kinetic chain that may result in a reduction of strength/endurance/power of the hamstrings and gluteus maximus and maintaining muscle strength and endurance of these muscle groups is essential to reduces the risk of subsequent muscle strain injury.

There are a number of biomechanical risk factors that may be present, including overstriding or lunging, excessive or poor control of anterior tilt of the pelvis and poor coronal plane stability i.e. dropping of the pelvis on the stance leg. Habitual trunk flexion in lunging activities and a crouching type of gait while running have the potential to

further increase demands on the proximal hamstring tendon. Many of these features only become apparent in a fatigued state, hence analysis should include assessment during fatigue. Technical re-education may be required to complement strength programs.

RETURN TO PERFORMANCE CONTINUUM: MANAGING THE TENDON PROBLEM: CONTINUE OR WITHDRAW SPORT PARTICIPATION

The decision to continue or withdraw from sport participation considers pain intensity, the effect of the tendinopathy on the athlete's performance and the time of season. Tendinopathies are rarely solely responsible for removal from competition, Players can often continue to play with tendinopathy however performance can be affected to a point where a period of recovery and rebuilding football fitness and skills may be required (3).

If maintaining training and competition, increasing tendon pain or pain that persists for 24 hours after activity indicates the need to reduce tendon load (57). If pain and function are acceptable, football can be continued. In Achilles tendinopathy running while maintaining pain within acceptable levels (maximum pain score of 5 points on a 0-10 scale) did not adversely affect outcome (67). However, if competition is continued, parallel rehabilitation should aim to maintain muscle strength and endurance without excessive tendon load (68) as it compromises tendon recovery (69). Scheduling multiple strength sessions with field training in-season can be difficult due to player fatigue. Strength exercises after team training may allow better recovery prior to the next training session. Rapid activities involving deep hip flexion such as deep squat plyometrics and select training drills may need to be ceased in the short term to settle symptoms and prioritise match performance.

If, despite these modifications and interventions, pain and function levels are not acceptable, removing the athlete from competition may be indicated. This should be based on symptoms and function rather than imaging findings and the athlete can return to football when the capacity to absorb repeated loads equivalent to training is regained.

ADJUNCT TREATMENTS:

Medication use may be approached in a stepwise manner starting with paracetamol and non-steroidal anti-inflammatory medication for pain relief. If the desired effect is not achieved, very occasionally a short course of oral corticosteroids in-season to gain symptomatic control might be appropriate, subject to FIFA drugs-in-sport guidelines. It is important to share potential side effects and complications of corticosteroid treatment with the player (70, 71).

Extracorporeal shockwave therapy is a treatment adjunct to consider. A recent meta-analysis showed moderate-level evidence for effectiveness of radial shock wave therapy in patients with proximal hamstring tendinopathy (27). This was based on a small randomised controlled trial in 40 patients, where four shock wave therapy sessions were applied at weekly intervals (2500 impulses at four bars, energy flux density 0.18 mJ/mm2 at 10Hz) (72).

Injecting corticosteroids, platelet-rich plasma and prolotherapy are potential adjunct treatments. Local corticosteroid injections in tendinopathies are effective in reducing pain in the short term, but pain is not improved in the longer term (73). Tendon rupture is a potential complication of local corticosteroid injections, especially when this is used repetitively (74) but specific data in proximal hamstring tendinopathy is lacking. Two case series in patients with proximal hamstring tendinopathy showed temporary effects on pain after local corticosteroid injections, but symptoms persisted in most patients in the longer term (33, 75). Clinicians should be cautious with applying local injections at the hamstring origin as efficacy in the longer term has disappointing results and the complication of a full tendon rupture is career-threatening for the professional football player.

Platelet-rich plasma injections aim to deliver growth factors locally with resulting regenerative effects. Several small case series have been performed on the effectiveness of platelet-rich plasma in patients with proximal hamstring tendinopathy with conflicting evidence for an improved clinical outcome (76-79). One RCT showed that platelet-rich plasma was not more effective in reducing clinical symptoms than autologous whole blood injections for patients with proximal hamstring tendinopathy (80). While platelet-rich plasma injections appear to be safe, the effectiveness of this treatment for proximal hamstring tendinopathy is not known when compared to a sham procedure or rehabilitation alone.

Effectiveness of prolotherapy has not been evaluated in patients with proximal hamstring tendinopathy. There is limited evidence for effectiveness of prolotherapy in other tendinopathies, although robust studies are lacking (81). Local injections of glucose or dextrose may result in temporary irritation of the sciatic nerve, but they appear to be safe with no reported complications.

Surgery of hamstring tendinopathy is considered when conservative treatment is unsuccessful. Retrospective case series studies including tenotomy, bursal and tendon debridement and removal of adhesions around the sciatic nerve, have demonstrated positive results on pain and physical function. Return to pre-injury level of sport post-surgery vary from 77-100% (82-84) with mean time to return to pre-injury level sport of around 5 months (82, 84). Adverse effects that have been reported in case series following surgery include paraesthesia (due to likely damage or irritation to the sciatic or posterior femoral cutaneous nerve), wound abscess and deep vein thrombosis (82-84).

Summary:

- Proximal hamstring tendinopathy is a cause of persistent ischial pain, with a reported incidence in professional football of 1.5 (95% CI 0.5-3.2) per 100 athletes per season.
- The condition is characterised by deep buttock pain that is well localised to the hamstring origin, occasionally with minor radiation. Symptoms are worse after periods of inactivity and a warm-up pattern is often evident with activity.
- Running, especially sprinting or acceleration, is often impaired, with pain generally worse at faster speeds or during rapid change of direction. Pain is also provoked by activities requiring significant hip flexion, such as sitting, squatting or lunging.
- Proximal hamstring tendinopathy is a clinical diagnosis based upon the history and confirmation with pain provocation tests. Imaging is not required for diagnosis.
- A number of potential differential diagnoses must be considered for athletes with pain in the ischial region, including somatic referral from the lumbar spine, sacroiliac and hip joints, as well as radiculopathy and peripheral nerve entrapment. Several symptoms of sciatic nerve irritation mirror those of proximal hamstring tendinopathy, which can make differential diagnosis challenging. There is also potential for coexistence of these pathologies.

Clinical Implications:

- Early management is focused on minimising provocative tensile and compressive loads while maintaining as much of the athlete's normal training load as possible. Options include reducing acceleration and deceleration while maintaining steady state running and removing provocative weights such as deadlifts, squats and lunges.
- Isometric or isotonic exercises are generally well tolerated if positioned in hip neutral. 3-4 repetitions of 45-sec holds are recommended for isometric loading. This is complemented by a heavy slow resistance training program. The degree of hip flexion during exercise can be gradually increased as irritability on compression decreases. The 24-hour response and pain on a provocation test will guide the progression of range and resistance. Heavy slow resistance can be completed three times per week, with the aim of equalising strength to the contralateral side.
- Alternative exercises in the gym that minimise hip flexion include prone hamstring curls, Nordic curls or Bosch holds. It is important to maintain strength of the hamstring group as detraining is deleterious to proximal hamstring tendinopathy and also increases hamstring muscle strain risk.
- When returning the athlete to higher speed running, it is important to quantify the speed attained and number of repetitions completed to enable workload tolerance to be determined. Speed sessions are best limited to twice weekly.
- The decision as to whether to continue or withdraw from competition should be based on symptoms and function rather than imaging findings. If an athlete continues to compete during the rehabilitation period, parallel rehabilitation should avoid high tendon load exercises, as this may exceed the capacity of the tendon when combined with the load encountered during competition.
- Extracorporeal shockwave therapy may be considered as an adjunct treatment for proximal hamstring tendinopathy. Local corticosteroid injections have been shown to be effective in the short-term but detrimental in the longer term. The effectiveness of platelet-rich plasma and prolotherapy in patients with proximal hamstring tendinopathy is not known.
- Surgery is rarely indicated and should only be considered in the case of repeated failure of rehabilitation.

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— Andreas Serner, Andrea Mosler and Christian Bonello

3.4. ADDUCTOR TENDON INJURIES

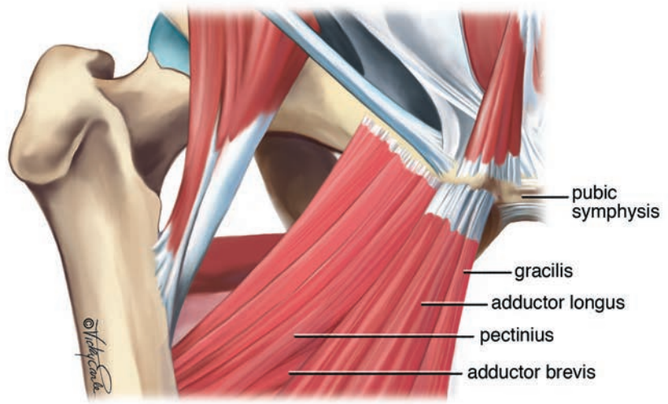
EPIDEMIOLOGY OF GROIN INJURIES

Groin injuries encompass pain and pathology in many different structures in the hip and groin area, one of these injuries is adductor tendinopathy. The diagnosis of adductor tendinopathy is however limited by a lack of valid examination tests, thus pain associated with the adductor tendons is currently classified as adductor-related groin pain (1, 2). Adductor-related groin pain is the most common entity experienced by professional male football players, accounting for at least 2/3 of groin injuries (3, 4). Groin injuries overall are common in football due to the high loads of running, direction changes, and kicking (3-7). It is the third most common time-loss injury experienced by male professional football players, accounting for around 13% (range: 4-19%) of all time-loss injuries sustained each season (7). Groin injuries are less prevalent in elite female football players (8), where they represent only 7% (range: 2-11%) of all reported injuries (7). Incidence rates for time-loss groin injury vary from 0.6 to 1.1/1000hrs (3, 4, 7, 9), and prevalence rates are high with 21% of professional male football players experiencing a time-loss hip/groin injury each season (3, 10). Most groin injuries have a gradual onset (3, 10), and result in less than 1 week's absence from football, but the risk of recurrence is high (3, 4). Furthermore, symptoms may persist following return to play, and be carried into the following football season (11).

Football injury surveillance studies have traditionally used a time-loss injury definition. However, hip and groin injuries often cause symptoms and reduced performance without forcing time-loss from training and match play. Therefore, this definition underestimates the true burden of groin injuries. Studies of groin symptoms in male football players (with and without time-loss) report prevalence rates as high as 59% (9, 12), with 20-30% of players experiencing some form of groin problem during any given week (12, 13).

ADDUCTOR ANATOMY

The adductor muscle group includes the adductor longus, brevis, magnus, and minimus muscles plus the pectineus, gracilis, and obturator externus muscles. The adductor longus is the most commonly implicated muscle in both acute and long-standing groin pain (14, 15). The anatomy of the proximal adductor longus tendon and muscle has been examined in several studies (16-18) (Figure 1). Unlike other common sites of tendinopathy (Achilles, patellar and hamstring tendons), the adductor longus does not have a free tendon, as the musculotendinous junction starts immediately adjacent to the insertion (16, 17). The proximal adductor longus tendon continues superficially on the muscle fibres, with the lateral part of the tendon transitioning intramuscularly at approximately 1-2.5 cm from the insertion (Figure 1) (18). The entire proximal tendon then becomes intramuscular at about 5.5-8 cm from the insertion, where it continues as an intramuscular tendon (18). The total length of the proximal tendon varies between 7-17 cm (17).



< Figure 1. Adductor Origin.

The distal adductor longus tendon inserts along the middle third of the femur on the linea aspera and appears to extend superficially and proximally as a tendon aponeurosis. The distal tendon is a rare cause of tendinopathy-related symptoms (19), although the muscle tendon junction of the distal tendon accounts for a large proportion of acute adductor muscle strains (14).

WHAT LOADS AFFECT THE ADDUCTOR TENDON?

The adductor longus works as both a stabiliser of the hip and pelvis and a prime mover for adduction of the thigh. The muscle is active in various movements, but two particular movements in football are consistently reported as associated with adductor-related groin pain; kicking and change of direction/cutting (20). While the name suggests the key movement of the muscle is hip adduction, the movements associated with injury characteristically are tri-planer with hip extension/flexion and rotation as additional components.

KICKING

The adductor longus is active throughout the kicking motion. During a maximal in-step kick (striking the ball with the top of the foot), the highest level of adductor longus activation and rate of stretch occurs in the backswing phase of the kicking leg- immediately after peak hip external rotation, and close to peak hip extension. The maximal length of the adductor longus is seen in the leg cocking phase, just after peak hip extension, and before peak abduction of the kicking leg (21). This implies that the peak adductor longus activation occurs eccentrically prior to the transition from hip extension to hip flexion, with muscle activity levels reportedly around 50-60% of an maximum voluntary isometric contraction (21). That in itself may not sound substantial, but this load occurs in less than 200 ms (22, 23), signifying a rapid onset of load on the muscle-tendon unit at a long length. Additionally, the peak ball impact reaction force is around 3000 N, and occurs over only 8–10 ms (24). Ball impact is therefore also associated with considerable load on the adductor longus muscle-tendon unit. Maximal instep kicking is of course not that frequent in football matches, but most variations of kicking will apply a considerable load on the adductor longus tendon. The side-foot kick (striking the ball with the inside of the foot), although not as powerful as the instep kick, has greater hip external rotation motion, leading to a comparable hip adduction torque (22). Even short side-foot passing, where hip abduction-adduction movement is minimal, adductor longus forces are reported to be around 200-260N (25). The repetitive nature of kicking leads to a repeated stress on the adductor longus tendon and load accumulation is a key factor in groin injury.

CHANGE OF DIRECTION

The adductor loads in change of direction is considered an important factor in the development of adductor-related groin pain (26). Football players perform numerous changes of directions during training and matches. The loading on the adductor longus is influenced by the amount of direction change, which varies from minor cutting angles (<45°) to full 180°. turns, as well as the cutting (change of direction) technique (e.g. side-step, crossover, split step) (27). Additionally, players can exhibit varying biomechanical movement strategies in the same sporting action (26), which can also influence load on the adductors.

The highest load on the adductor longus appears to be on the leg that is pushing off, as acute adductor longus injuries occur in the push-off leg (20). The push-off leg will have a typical pattern of hip extension and abduction often with the hip externally rotated. This increases adductor longus muscle-tendon unit length until the late stance phase, which is followed by with hip adduction and flexion in the early swing phase (28, 29). This requires high adductor longus muscle activity, which is highest during weight acceptance at the start of push off, and remains high through the final push-off phase, with a mean muscle activity of more than 100% MVIC (28).

CLINICAL PRESENTATION

SYMPTOMS

Athletes with longstanding adductor-related groin pain describe pain in the region of the medial upper thigh (equivalent to the proximal adductor tendon) and often on the pubic bone (Figure 2). The pain starts in one region and is unilateral, but can then gradually spreads to other regions, and can become bilateral (30). Groin pain may also be present around the pubic symphysis (pubic-related groin pain), over the inguinal canal area (inguinal-related groin pain), or at the anterior hip (iliopsoas- or hip-related groin pain), and pain location is important in categorising groin pain (1).

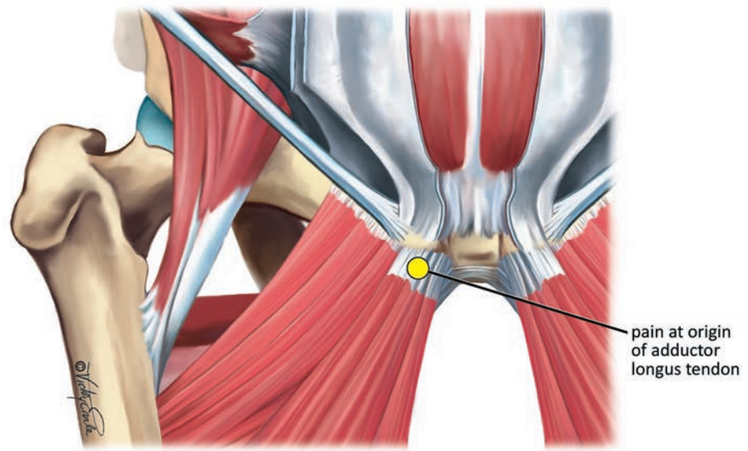


Figure 2. Adductor pain map.

Adductor-related groin pain is aggravated by specific movements, such as change of direction and kicking. Longstanding adductor-related groin pain in athletes usually has a gradual onset, but can also follow an acute injury that has not fully resolved (15). The athlete and coach often notice a gradual reduction in sports performance, particularly explosive actions. Classically, athletes experience pain after activity, often with pain and stiffness the following day. This then progresses to experiencing pain during activity in the typical load- and dose- dependent pattern associated with tendon pain. If the pain becomes more severe, activities of daily living can also be painful, such as turning in bed and getting in and out of a car. The natural history of groin pain is one of progressive increase with continued activity until symptoms eventually prevent participation in sporting activity.

QUESTIONNAIRES

Patient reported outcomes measures can provide a measure of the athlete's self-perceived symptoms. An easily implementable questionnaire is the Oslo Sports Trauma Research Centre's overuse injury questionnaire (OSTRC), which consists of four questions related to problems with participation, modified training/competition, performance, and pain (31). This can be focused on groin problems and provides a total severity score from 0-100. For a more extensive insight into subjective limitations, the Copenhagen Hip and Groin Outcome Score (HAGOS) can be used. This questionnaire consists of six subscales on pain, symptoms, activities of daily living, participation, and quality of life, each with a score from 0-100 (32). The HAGOS is freely accessible online (www.koos.nu) and has been translated into 14 languages. The questionnaire is not designed to diagnose hip and groin pain but applies well to patients with adductor-related groin pain. Clinicians can select the most relevant subscale to decrease questionnaire time. For an individual patient, changes of 10 to 30 points exceed the minimal detectable change, depending on the subscale and patient population (32, 33).

CLINICAL EXAMINATION

A comprehensive examination of the entire groin region is essential for athletes presenting with groin pain (34). The diagnosis of adductor-related groin pain is defined as "adductor tenderness and pain on resisted adduction testing" (Figure 3) (1). Most commonly athletes will present with pain localised to the adductor longus insertion on the pubic bone, both during palpation and with resistance testing (35). These two clinical examination findings have shown excellent intra- and inter-examiner agreement (36), though the diagnostic accuracy remains unknown. An adductor resistance test may provoke pain in areas other than the adductors, therefore it is important to ask the patient where they feel the pain during the test and whether this is their activity pain. Pain must be felt in the adductor region to be classified as being adductor-related groin pain (1). Palpation of the adductor longus, and other groin structures in general, can be uncomfortable and often painful even in absence of injury. Therefore, the clinical tests should always be compared with the uninjured side. It is also important to consider that adductor-related groin pain often co-exists with other entities of groin pain.

^ **Figure 3.** Adductor squeeze.

DIFFERENTIAL DIAGNOSIS

The recognisable pattern of symptoms exhibited by athletes for the four defined clinical entities of groin pain are outlined in the Doha agreement meeting consensus paper (1). Clear clinical examination findings are also described to assist the clinician in differentiating these clinical entities. Currently, the diagnostic accuracy of clinical testing to rule in hip-related pain is limited (37). Clinical suspicion based on the patient history can assist in differentiating between hip joint and adductor-related groin pain. Pain descriptions such as: deep hip joint pain where the pain is indicated by the patient's hand spanning the front, side and back of the hip region (known as the C-sign), pain with prolonged sitting, especially in deep hip flexion (38), can be suggestive of hip-related pain. A negative flexion, adduction, internal rotation test is helpful to rule out the hip joint as the source of groin pain in athletes (37). Various medical conditions can also mimic musculoskeletal groin pain in athletes, and clinicians should specifically question about intra-abdominal and gynaecological dysfunction in their differential diagnosis (Table 1) (1). Red flags that require screening include disorders or cancer of the reproductive organs such as endometriosis, urinary tract infection, prostatitis, or testicular can¬cer. Past history of prostate or breast cancer can also be associated with metastases in the hip and groin region (39). Therefore, the clinician should determine if there is a history of trauma, fever, unexplained weight loss, painful urination, or pro-longed corticosteroid use that could influence the presentation (39, 40). Other serious bony pathology can cause groin pain and must be considered in the differential diagnosis process (1, 34). These include pelvic or femoral stress

fracture, avascular necrosis, and in the young athlete with an immature skeleton, pubic apophysitis must be considered (41). A history of associated low back or buttock pain indicates that the groin pain may be referred from another site (such as the hip or the lumbar spine) (34). A full training history should be taken to determine if any recent changes in training (e.g. a generalised increase in volume or intensity, the introduction of a new exercise or an increase in a particular component of training) may have led to the development of the groin pain.

OTHER MUSCULOSKELETAL CAUSES OF GROIN PAIN	OTHER CAUSES OF GROIN PAIN
Bone and joint • Stress fracture neck of femur, pubic ramus, acetabulum • Head of the femur pathology - avascular necrosis/transient osteoporosis • Arthritis of the hip joint • Referred pain from spine	• Inguinal or femoral hernia pre or post surgery • Inguinal lymph nodes • Intra-abdominal abnormality - prostatitis, urinary tract conditions, gut *Tumours* • Testicular, bone, prostate, urinary tract , gut, soft tissue *Systemic inflammatory conditions* • Ankylosing spondylitis, gut related
YOUNG PLAYERS	
Bone and joint • Apophyseal - anterior superior iliac spine, anterior inferior iliac spine, pubic bone • Slipped capital femoral epiphysis, Perthes' disease	• Tumours
WOMEN	
	• Gynaecological conditions

^ **Table 1.** Differential diagnoses of groin pain.

IMAGING AND ITS ROLE IN DIAGNOSIS, PROGNOSIS AND AS AN OUTCOME MEASURE

The use of imaging in long-standing adductor-related groin pain is highly debated due to the complex anatomy in this area. The interpretation of imaging findings can be difficult even for specialised musculoskeletal radiologists. Additionally, there is large heterogeneity and varying methodological quality in the available literature (42). Magnetic resonance imaging (MRI) is considered the optimal imaging method to get an overview of the groin structures, however many MRI findings, which may be considered pathological, have been shown to be associated with sports-activity rather than groin pain (43). Imaging is helpful to rule out serious pathology in groin pain presentations; however, when the pain is clinically determined to be related to the adductor longus tendon, there is no evidence to suggest an improvement of diagnostic or prognostic indicators with imaging, except with avulsion injuries (42, 44). Adductor tendinopathy on MRI has been defined as "an increased signal intensity within the adductor longus tendon on fluid-sensitive sequences and/or bulging of the tendon" (45). This

finding however comes with very poor reproducibility (45), and is found both in asymptomatic football players and in football players with adductor-related groin pain, with prevalence of around 70% in both groups (43). Therefore, the MRI finding of adductor longus tendinopathy is currently not clinically useful. Abnormal imaging in structures close to the proximal adductor longus insertion may be relevant to adductor- and pubic-related groin pain, such as pubic bone marrow oedema (graded from 0-3) and symphyseal disc protrusion (43). However, it is still uncertain whether these findings influence prognosis. In adolescent athletes, pubic apophysitis is a differential diagnosis to adductor-related groin pain where imaging can assist (41). Computed tomography is considered the best imaging modality to confirm this diagnosis, but MRI with specific sequences may also assist with a relevant impression of the pubic apophysis to avoid unnecessary radiation (46).

Ultrasound of the adductor longus tendon and insertion can provide good visualisation of the different adductor muscles; (47), however, the relevance of findings, such as adductor longus tendon thickening or hypoechogenecity, are still unclear. As such, the main role of ultrasound in patients with adductor-related groin pain is to exclude alternative and/or potentially serious diagnoses (48).

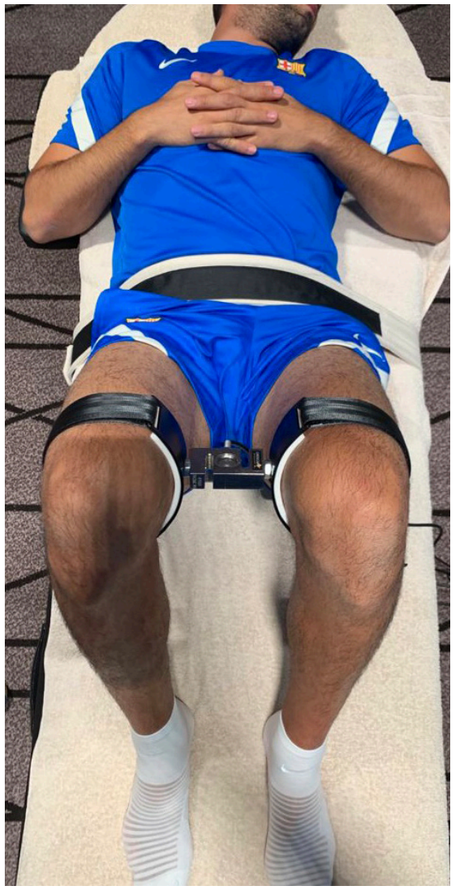
MANAGEMENT OF INITIAL PRESENTATION OF PAIN: WHAT TO DO WHEN THE PLAYER APPROACHES THE CLINICIAN

Symptoms have often been present for some time when an athlete with adductor-related groin pain first approaches the clinician. Therefore, a thorough medical history taking is essential to determine the severity and potential contributing factors to the current pain presentation. Asking the athlete to complete the HAGOS questionnaire on initial presentation, prior to commencing examination, is useful to establish their baseline symptoms.

In addition to assessing the diagnosis/categorisation of groin pain, the initial physical examination must determine the severity and irritability of the groin pain presentation. A traffic light approach to pain on provocation testing using the numeric rating scale can be particularly helpful to establish severity and develop an appropriate management plan (49). For adductor-related groin pain, the level of pain in the 5-second adductor squeeze test (Figure 4) correlates well with HAGOS subscale scores (49). Severity of pain on the numeric rating scale with adductor palpation, stretch, and functional tasks like kicking can also be helpful to establish symptom severity.

Depending on symptom severity, it may also be appropriate to examine impairments on initial presentation. Measuring adduction and abduction strength, and calculating the adduction:abduction ratio can assist in the evaluation of impairments and potential contributing factors to long-standing groin pain (5). Hip strength can be measured reliably with a hand held dynamometer, and can be compared to normative data in football players (50, 51). Abdominal strength, trunk endurance, quadriceps, and calf strength, muscle length, and functional tests, such as change of direction tests, may also be included in the examination of impairments.

Education about the pathology, contributing factors, and a discussion of realistic expectations and return to sport



^ **Figure 4.** Adductor squeeeze.

goals early in management is essential to success (52). Management of longstanding adductor-related groin pain can be complicated by the presence of multiple entities, contributing impairments, high recurrence rate, and potential for slow progress. Shared decision making necessitates an open discussion between the athlete, coach, parent (if applicable), medical, and conditioning staff to develop an appropriate management and rehabilitation plan with clear criteria for progression (53). The short-, medium-, and long-term goals of the athlete and the coach should be considered when developing this plan.

Pain management may be required in the early stage of treatment for adductor-related groin pain. Adjunct therapies are commonly used by clinicians for pain modulation and have varying levels of evidence of efficacy. Manual therapy and compression shorts have demonstrated efficacy for pain reduction (54-56), while other adjuncts such as taping, dry needling, and electrotherapy have little evidence. Pain management using these adjunct therapies must always be in conjunction with a graduated exercises programme, which has the highest level of evidence for the management of long-standing groin pain (55, 57).

MANAGING THE TENDON PROBLEM: CONTINUE OR WITHDRAW FROM SPORT

Players who can still perform despite pain will usually want to continue playing, but this comes with a risk of exacerbation potentially leading to prolonged absence from play. Additionally, adductor longus avulsions, although rare, can occur if participation continues with pain. Anecdotally the risk is higher in players taking pain medication to enable play. The clinician will therefore need to provide a clear risk assessment for continued participation. The decision to continue or withdraw from sport must consider multiple factors related to risk assessment (risk of further injury) and risk tolerance (how important the risk is). If the risk assessment of the groin injury is considered less than the tolerance of risk determined by the multi-disciplinary team, the decision should be to allow the player to continue with sport.

The decision will include an individual evaluation of the player's health status, including pain intensity, injury history, clinical and functional limitations, and the psychological state of the player (58). As part of this, clinicians can use the 5-second adductor squeeze test to monitor clinical pain intensity (49), and objective adductor strength measures should be included. A bilateral isometric squeeze tests can be used to track strength fluctuations over time (59), whereas a unilateral adductor strength test should be performed with an eccentric contraction, as this can detect muscle strength deficits better than an isometric test (60). A thorough subjective assessment (questionnaires) and objective testing of football specific movements should be included. The player's personality and psychological state may also provide an impression of whether the player is likely to disregard potentially severe pain or, in contrast, overreact to minor sensations in the groin. Another part of the risk assessment is evaluating factors related to the player's sport that can influence adductor loading, such as playing position, limb dominance, and the competitive level of play (58). For example, in a case of right-sided adductor-related groin pain, a left-dominant central defender from a recreational level could be considered to have a lower risk than a professional right-dominant winger, due to a lower amount and intensity of change of directions and passing and kicking.

A risk assessment should also consider different outcomes, and a percentage interval can be given for each outcome. For example, a player may be determined to have a 30-40% risk of increased adductor pain, a <5% risk of an adductor longus avulsion, and a 20-30% risk of reduced performance. Following this risk assessment, a decision on risk tolerance should be made considering all potential outcomes. Risk tolerance is then influenced by factors not directly related to the injury, such as time of season, current match schedule, pressure from the coach and the athlete's desire to play, as well as potential financial and other conflicts of interest (58). In the presence of risk modifiers resulting in increased risk tolerance, a shared decision-making process is essential, so all stakeholders are aware of the perceived risks and benefits of sports participation in order to make the best decision on whether to allow a player to continue to play unrestricted, to modify training, or to withdraw the player completely. As part of this process, it is important to remember that adductor-related groin pain in general has a considerable re-injury risk in elite football with 11% of players experiencing a re-injury resulting in time-loss within 2 months after returning to full participation (3, 4).

AN EXAMPLE OF A STAGED REHABILITATION PROGRAM

There is no evidence that a particular rehabilitation program is optimal. Therefore, different approaches may give similar results. The main objective is for the player to follow a progressive increase in load, with a focus on both increasing specific load capacity and progression to required sports-activity level. We provide an example of a structured 4-stage rehabilitation program focusing on progressive loading of the adductor muscles governed by symptom response (Table 2). It is important to also include a general training focus beyond adductor training to achieve the best results. Kinetic chain exercise should include the calf complex, quadriceps, hamstring, gluteal musculature and trunk strength. Many of these exercises can be initiated with close to the player's normal capacity for load even at the beginning of adductor rehabilitation. Inspiration for exercise selection for other muscle groups can be found in other sections in this guide. Additionally, cardiovascular fitness must be maintained through cross-training or stationary bike, until adequate running intensities can be resumed.

STAGE 1: ISOMETRIC

Isometric exercise may invoke exercise-induced hypoalgesia in local musculoskeletal conditions (61), and can be used for players with severe or irritable symptoms (62). These players may have a fear of movement and loading. Initiating loading in controlled positions without movement can therefore provide reassurance prior to progressing load and movement. Isometric exercises can maintain muscle activation and reduce muscle atrophy. Isometric exercise can be used several times a day if needed.

STAGE 2: SLOW RESISTANCE EXERCISES

When the player is ready to attempt loading through movement, slow resistance exercises can be introduced. A seated bilateral adduction machine functions well as a starting point for loading, as there are no stability requirements, and the external load easily can be monitored and progressed between or even within sessions. Some pain during exercise can be expected and the load should be adjusted according to the individual player's pain. A clear agreement on acceptable pain limit should be made, (using a numerical pain rating scale from 0-10 for example), with an acceptable cut-off level of pain during exercise set to between 2/10 and 4/10, depending on the individual athlete. The external load should then be as heavy as possible according to the set pain level, and the player can perform as many repetitions as possible.

Standing single leg adduction exercises with a heavy resistance band or in a cable pulley can also be initiated early- either in place of the adduction machine or as an addition. Strength work should be completed on each side separately, and the load adjusted according to the ability of each position (leg).

Once the player is able to withstand a higher load in these exercises, the Copenhagen adduction exercise can be introduced. This can initially be performed with a short lever (partner holding the knee or the knee is placed on a bench). When a player can perform 10 repetitions pain free, it is a good indication that they can progress to faster loading and kicking. We recommend maximum to heavy resistance exercises are performed, so the Copenhagen adduction should preferably take the place of the seated adduction machine.

STAGE 3: ENERGY STORAGE AND RELEASE

Once strength and endurance parameters are achieved, faster functional loads can be applied. An elastic resistance band or a cable pulley work well for this purpose, but the load should be lower than in the slow resistance exercise. It is important to register dosage and relative speed of movement. The exercises should only be progressed if the tendon is tolerating load, and pain is stable during exercise and for the 24-hours following the exercise. Pain levels can be assessed using something like the adductor squeeze test (63). Slow resistance is continued in this stage and volume is often progressed prior to speed. The exercise can be progressed from fast single plane adduction, to a multiplanar kicking motion as the adductor complex improves in load tolerance.

STAGE 4: SPORT SPECIFIC LOADING

Once the adductor tendon demonstrates an ability to manage controlled faster movements with stable and low pain (pain severity may not necessarily be zero), then sports-specific exercises can replace stage 3 exercises.

Stage 2 exercise should be continued. Stage 4 will involve an incremental progression of volume and intensity of football movements, such as side-steps, change of direction, running, jumping, sprinting, passing and kicking. . At this stage attempt to change only one or few components of the program at a time, as the tendon can react to these higher loads differently. It is important to count the number of repetitions and to build slowly in numbers and speed/intensity and always monitor pain response after load increases. As the athlete progresses through stage 4, more sport-specific drills can be substituted for other stage 4 exercises, and eventually the player can be included in modified team training sessions, before returning to full team training.

STAGE OF REHABILTATION	TYPE OF EXERCISE	EXAMPLES FOR ADDUCTOR TENDON			GOALS	ANTICIPATED TIMEFRAME
		Aim	*Example exercise*	*Exercise Parameters*		
1	**Isometric**	Pain relief Loading reassurance	Adduction squeeze with bent knees Adduction squeeze with straight knees	Frequency: Multiple times daily (set as pain relieving strategy) Intensity: According to set pain level (e.g. 2/10) Dosage: 5x30sec	<2/10NRS	1-2 weeks (can be continued through whole rehabilitation as required)
2	**Slow resistance**	Strength	Seated adduction machine Standing adduction against resistance elastic/ cable machine Copenhagen adduction exercise (short progressing to long lever)	Frequency: x3 weekly Intensity: According to set pain level (e.g. 3-4/10) Dosage: Increase from 2-3 sets initially to 4 sets if no considerable DOMS or pain exacerbation is present. (3 sec concentric, 3 sec eccentric)	Aim >3Nm/kg eccentric adduction strength 1.2:1 adduction /abduction strength ratio	8 -12 weeks (Continued after return to sport with adjusted dosage).
3	**Energy storage and release loads**	Progressing speed of movement.	Standing adduction against resistance - fast movements Kicking movements with resistance (elastic/ cable pulley)	Frequency: 3x weekly Intensity: Progress movement range, then volume, then speed. Dosage: 3-5 sets of 8-10 reps		2-4 weeks Strength exercises continued
4	**Sport specific loading**	Prepare for return to play.	Passing/kicking with ball. Change of direction drills on pitch (increasing in speed and volume)	Frequency: 3-5 times a week as tolerated. Intensity: Progress incrementally from easy/moderate short passes, to hard passes, and then position specific passes/ crosses/kicking Dosage: Activity and symptom dependant. A relative high volume of easy passing can be introduced early. Hard longer passes and kicking should be introduced later with a very low volume initially.		Until cleared for return to play, gradually phase in sport specific training (Strength exercises continue).

^ Table 2. Adductor rehabilitation programme design.

Summary:

- Groin injuries overall are common in football due to the high volumes of running, change of direction and kicking. Approximately two thirds of injuries are due to adductor-related groin pain.
- Groin injuries are the third most common time-loss injury in male professional football. However, the true prevalence of groin injuries is estimated to be even higher, as many players are able to continue training and playing while injured and therefore these injuries are not captured using a time-loss definition.
- The prevalence of adductor injuries in football has been estimated to be as high as 59%, with 20-30% of players experiencing some form of groin problem in any given week.
- The adductor longus works both as a stabiliser of the hip and pelvis and a prime mover of the thigh. The muscle is highly active during both kicking, cutting and change of direction, all movements commonly associated with adductor-related groin pain.
- Adductor longus is active throughout the kicking motion. During a maximal in-step kick, with the highest level of adductor longus activation and rate of stretching occurring in the backswing phase of the kicking leg.
- Change of direction of commonly implicated in the development of adductor-related groin pain. The loading of the adductor during change of direction is influenced by the angle of direction as well as the cutting technique.
- The highest load on the adductor longus occurs on the leg that is "pushing off" during change of direction tasks.

Clinical Implications:

- Athletes with adductor-related groin pain describe pain in the medial upper thigh and often on the pubic bone. Pain is generally of gradual onset but can also follow an acute injury that has become a longstanding issue. Pain is aggravated by specific movements such as change of direction and kicking. Classically pain is experienced after activity, with pain and stiffness present the following day.
- The criteria required for diagnosis of adductor-related groin pain include adductor tenderness on palpation and pain with resisted adduction testing.
- An adductor resistance test may provoke pain in areas other than the adductors, therefore it is important to ask the patient where they feel the pain during the test and whether this is their recognisable injury pain.
- Numerous other structures can refer pain to the adductor region, including the lumbar spine and hip joint. Additionally, various medical conditions may also masquerade as groin pain. Therefore, clinicians should always assess for a history of trauma, fever, unexplained weight loss, painful urination or prolonged corticosteroid use.
- Imaging is helpful to rule out serious pathology in groin pain presentations; however, when the pain is clinically determined to be related to the adductor longus tendon, there is no available evidence to suggest an improvement of diagnostic or prognostic indicators with imaging, except in the case of avulsion injuries.
- The level of pain in the five- second adductor squeeze test can be used to inform whether players should continue to participate in football training and play or stop activity and seek medical attention. A score on a numerical rating scale of 0-2 is considered safe for continued participation, a score of 3-5 signals need for clinical review before safe participation levels can be determined, and a score of greater than 6 indicates that activity should be ceased.

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Sign off

SIGN OFF

Tendinopathy is a complex diagnostic and management condition especially in football players training and competing at the elite level. The research and clinical pearls provided by the editors and expert contributors in this Guide will help clinicians to diagnose and manage tendinopathy in their athletes.

Despite the contributors in this Guide coming from a number of countries and continents and with varying research backgrounds and experience in managing tendinopathy in athletes, it is interesting to see that several central themes are consistent across all chapters. These consistencies are a great final message for clinicians reading and learning from this Guide to take with them into their own practice.

First a good assessment measures pain and function, not structure. Tendinopathy is a clinical diagnosis, and imaging is not always necessary, and can actually confuse the picture. Imaging cannot prognose, assess risk of rupture or be used as an outcome measure for gradual onset tendon pain.

Second, careful assessment is critical as many conditions can masquerade as tendinopathy. We cannot rely only on imaging and palpation soreness to diagnose tendinopathy, differential diagnosis is paramount.

Third, load is implicated in both the onset of tendon pain as well as its resolution. Excess or unusual load can initiate tendinopathy, and restoring capacity to cope with high loads is the cornerstone of management. Understanding the complex loads that a tendon experiences is one of the most important messages from this Guide.

Fourth, exercise is the critical intervention in the management of tendinopathy, it can take time to achieve the outcomes the athlete wants but providing an inadequate exercise program or not providing enough time for tendon, muscle, kinetic chain and brain adaptation can result in poor outcomes.

Finally, the role or adjuncts in tendinopathy is limited, few provide long term improvement, even if some can help pain in the short term. Choosing the right adjunct at the right time requires clinical expertise, using adjuncts as a means only to support an exercise program is essential.

The future of research into tendinopathy of athletes is exciting and an area that is a key priority and passion of the FC Barcelona Medical team as well as the expert contributors in this Guide. We look forward to progressing this field to understand how to more effectively treat and manage tendinopathy in athletes.

Thanks for taking this journey with us, the content editors are indebted to Dr Alan McCall for keeping us sane through this process. Go forth and treat tendinopathy!

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