

MANAGING THE ATHLETE WITH A STUBBORN PROXIMAL HAMSTRING TENDINOPATHY

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INTRODUCTION

Proximal hamstring tendinopathy (PHT) is an overuse injury that primarily affects running athletes (distance, sprinting, hurdling) and athletes involved in change of direction activities (e.g. football, hockey). Although uncommon, it is a stubborn injury that has a poor response to available treatments and often takes long to heal. In addition, the scientific evidence on PHT is very limited compared to other tendinopathic conditions. This makes it a challenging injury to manage, both for the athlete and the medical team.

The aim of this paper is to review the clinical management of PHT, based on both the limited available evidence and

our clinical experience in managing these stubborn injuries.

PROXIMAL HAMSTRING ANATOMY

A good understanding of the proximal hamstring anatomy (Figure 1) is paramount to effective clinical reasoning when managing an athlete with PHT. The proximal hamstring tendons attach to the upper posterior region of the ischial tuberosity¹. This bony region can be subdivided into a medial and lateral facet, which form the sites of attachment for the conjoint tendon and semimembranosus tendon, respectively. Due to this upper posterior insertion, the hamstring tendons are folded around the ischial tuberosity

during hip flexion, resulting in compression of the tendons. These compressive loads are thought to be an important factor in the aetiology of PHT².

The proximal semimembranosus tendon runs anterior to the conjoint tendon in close proximity to the sciatic nerve, which is located just lateral to the proximal hamstring tendon complex. In regards to differential diagnosis, it is helpful to note that the proximal hamstring attachment and tendons are located just anterior to the gluteus maximus, directly superolateral to the proximal adductor magnus tendon attachment (ischiocondylar portion)³, and lie in the posteromedial corner of the ischiofemoral space.

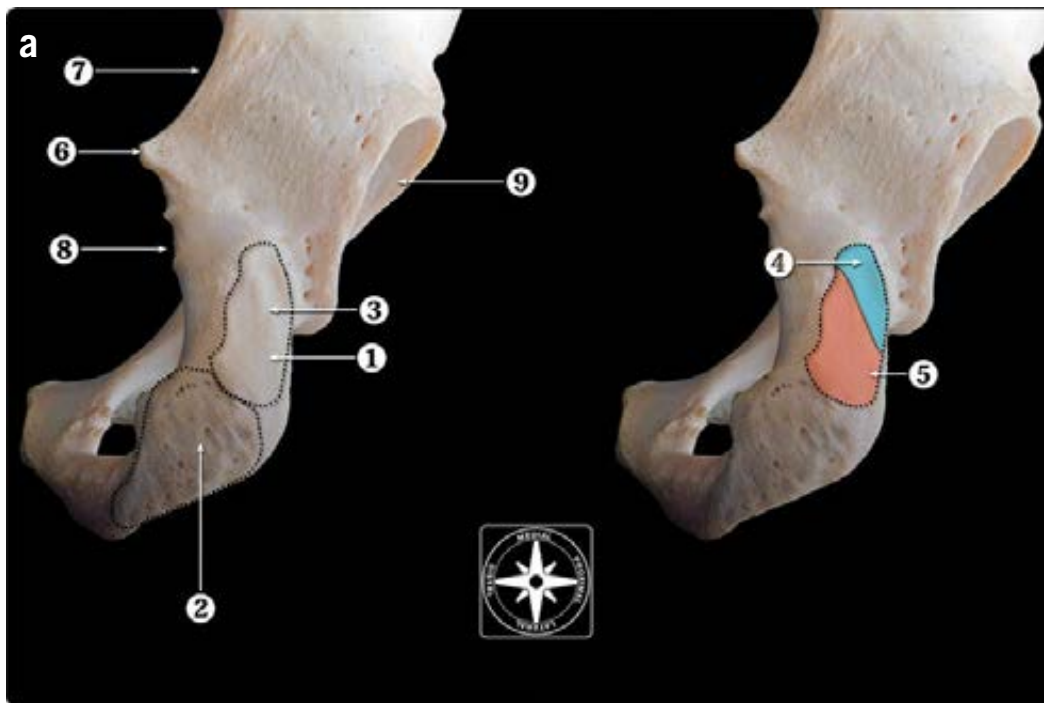


Figure 1: a) Posterior view of the right coxal bone showing the ischial tuberosity, subdivided into 1. Upper region and 2. Lower region. 3. Vertical ridge. 4. Lateral facet for semimembranosus attachment. 5. Medial facet for conjoined tendon of the long head of biceps femoris and semitendinosus attachment. 6. Sciatic spine. 7. Greater sciatic notch. 8. Lesser sciatic notch. 9. Acetabulum. b) Osteoarticular dissection with posterior view of the right proximal hamstring tendon complex. 4. Lateral facet with semimembranosus attachment. 5. Medial facet with conjoined tendon of the long head of biceps femoris and semitendinosus attachment. 6. Sciatic spine. 7. Greater sciatic notch. 8. Lesser sciatic notch. 9. Acetabulum. 10. Sacrospinous ligament. 11. Sacrotuberous ligament. 12. Adductor magnus.

DIAGNOSTIC CHALLENGES

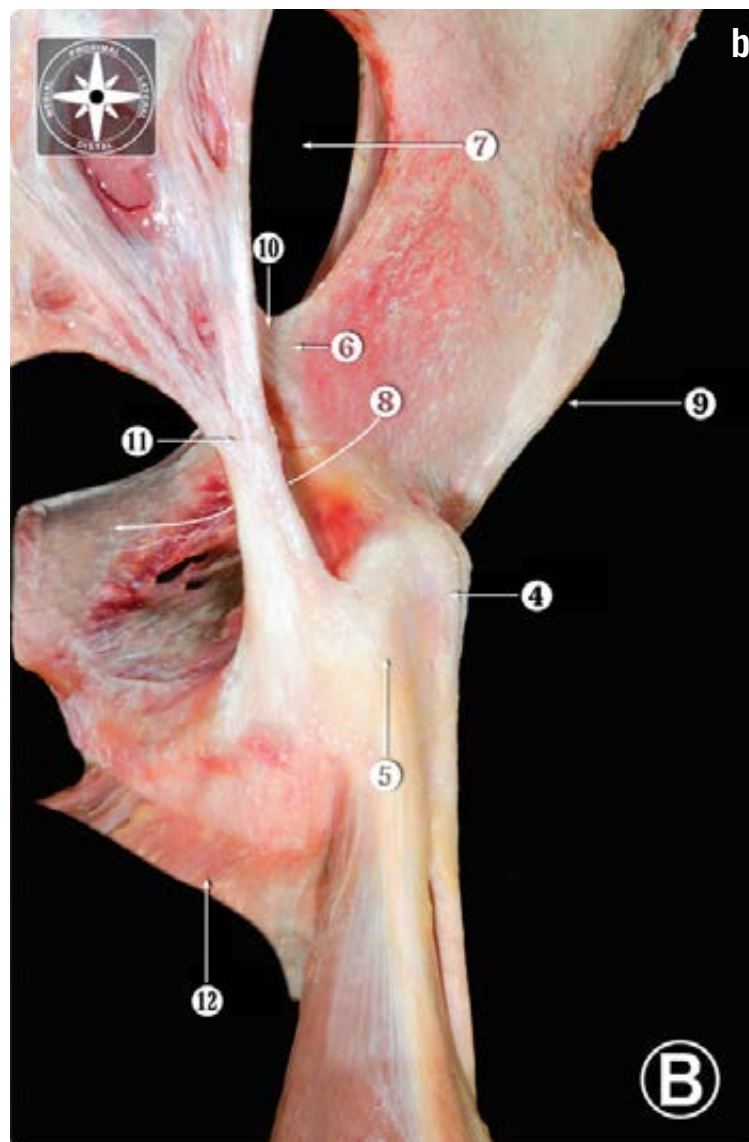
Although the clinical presentation of PHT is clear in some athletes with typical symptoms and signs, the diagnosis can be challenging in the absence of typical symptoms. PHT symptoms are quite similar to that of multiple other conditions. Additionally, there is very limited evidence to guide diagnostic management; a clear 'gold standard' as a reference point for PHT is lacking⁴, making the validity of diagnostic tests for PHT largely unknown.

History

The diagnostic cornerstone of PHT is a careful history and physical examination. The main symptom is pain in the region of the ischial tuberosity that may or may not radiate to the hamstring region. While symptom localisation at the ischial tuberosity is unique, the general clinical picture resembles that of a tendinopathy: insidious onset of symptoms, provoked or aggravated when commencing exercise, resolved or reduced after warm-up, and often returning or worsening symptoms for up to several days after exercise.

Local pain during sitting and driving is typical of PHT, likely due to the prolonged compressive forces on the proximal hamstring tendons. Excessive hamstring stretching involving sustained end-range hip flexion (e.g. yoga, stretching exercises) therefore often provokes symptoms.

Sciatic nerve related symptoms (e.g. numbness, burning or tingling radiating to



the lower leg and foot) may be present due to the proximity of the proximal hamstring tendons to the sciatic nerve, making the distinction between PHT and other nerve-related causes of pain (e.g. radiculopathy, sciatic nerve entrapment) challenging in some cases.

Physical examination

Physical examination is aimed at reproducing the pain by applying compressive and tensile loads to the proximal hamstring tendons. Basic hamstring tests, such as strength testing (knee flexion and hip extension against resistance), stretch testing (e.g. passive straight leg raise, passive/active knee extension test) and manually compressing the proximal hamstring tendons and its insertion at the ischial tuberosity, can be performed to provoke symptoms.

Scientific evidence is limited to three passive stretch tests (the bent-knee stretch, modified bent-knee stretch and Puranen-Orava test), which are reported to have moderate to high validity for diagnosing PHT⁴.

Imaging

Similar to tendinopathy in general, ultrasound- and MRI-scans are both good at visualizing tendinopathic changes in PHT. However, ultrasound has some important

limitations compared to MRI. Firstly, ultrasound is less sensitive in detecting tendinopathic changes such as partial tears and peritendinous fluid, and unable to detect bone marrow oedema⁵. Secondly, it is operator-dependent (the ultrasonographer's skill and experience influences image quality and interpretation). Ultrasound imaging of the proximal hamstring tendons is substantially more challenging and less reliable than that of other (more superficial) tendons, such as the Achilles and patellar tendons; even large tendon defects such as avulsion injuries are often missed or misdiagnosed by ultrasound imaging⁶. We therefore argue that ultrasound is not the imaging modality of choice for PHT, except in highly experienced/specialised centres.

The use of MRI as a diagnostic tool in PHT has been studied in several clinical series and provides some insight on the (limited) value of MRI that clinicians should take into account when considering diagnostic imaging⁷⁻⁹.

De Smet et al. studied the MRI appearance of the proximal hamstring tendons in patients with (n=21 tendons) and without (n=215 tendons) PHT symptoms (mean age of 41 year, range 4-87 years)⁷. Remarkably, they found that almost all tendons (>90%) had increased T1 and T2 signal abnormality. Peritendinous oedema was also commonly seen in asymptomatic tendons. Only the

presence of ischial tuberosity oedema was substantially different between symptomatic (19%) and asymptomatic tendons (3.3%), suggesting that this feature may be used to rule-in PHT, but has little value for ruling out PHT. The high prevalence of abnormal MRI appearance of the proximal hamstring tendons in asymptomatic subjects has been confirmed in two large series^{8,9}. In these series the prevalence and extent of abnormal MRI findings increased with age.

The association of MRI findings with PHT prognosis has not been studied. However, we know from other tendinopathies that imaging characteristics are usually not associated with the change in or the duration of symptoms and therefore of limited prognostic value¹⁰.

Imaging has limited value

Imaging is clinically relevant only when it provides additional diagnostic or prognostic information after a comprehensive clinical evaluation. We argued that PHT imaging currently has very limited value as: 1) it poorly discriminates between symptomatic and asymptomatic tendons, and 2) it has no proven prognostic value. Based on the literature and our clinical experience we suggest to consider the following aspects:

- In patients with typical PHT symptoms, imaging has no additional value;
- In patients with atypical symptoms,

Imaging is clinically relevant only when it provides additional diagnostic or prognostic information after a comprehensive clinical evaluation. In patients with typical proximal hamstring tendinopathy symptoms, imaging has no additional value.



imaging may be used to assess or rule-out pathology other than PHT;

- With increasing age, the presence of imaging abnormalities is of even less significance for diagnosis.

TREATMENT

High level scientific evidence on the best treatment of PHT is lacking. The following overview is the best available evidence-based approach taking into account evidence from other (lower limb) tendinopathies, and our own clinical experience.

Patient education and expectation management

Patient education and expectation management are key to a successful treatment partnership, especially in athletes with a stubborn PHT. We emphasize the following: 1) complete recovery usually takes months (sometimes up to two years) 2) the road to recovery is usually not a happy straight line pointing upwards; expect symptoms to be worse from time-to-time 3) progressive loading is the key; short cuts are counterproductive and 4) there is no 'quick fix' despite what expensive commercially offered therapies and their (guru) advocates may promise.

Modify load to manage pain

We aim to reduce pain to acceptable and stable levels (\approx VAS 0-3/10) during rehabilitation and avoid training loads that might increase or aggravate pain. Load modification is therefore very individual; some athletes may, for example, continue with a substantial amount of running, while others shouldn't run at all. However, there are some general principles to guide load modification and activity selection:

- Compressive loads to the proximal hamstring are usually more provocative. Activities and postures with increased hip flexion and/or anterior pelvic tilt provide more compressive loads and is easy to modify: e.g. cycling in a more upright position, avoiding long periods of sitting, and using a donut shaped-cushion to sit on.
- Tendon energy storage and release activities (spring like function) provide the highest tendon loads and can be avoided or reduced: e.g. cross-trainer, swimming or cycling instead of running activities.

Athletes are usually well aware of activities that might induce or aggravate pain. A pain diary is useful to guide further discussion on treatment plans.

Rehabilitation exercises

Based on evidence for tendinopathies in general, we consider a progressive loading rehabilitation program as described by the group of Cook and Purdam as the cornerstone of PHT treatment¹¹. We will provide a brief overview here. (A detailed description of the content of a program for PHT is outside the scope of this article, but can be found elsewhere in the article by Goom et al¹²). Some pain during and after exercises is acceptable (VAS 0-3/10) and progression through the phases should be based on symptoms (pain) and not time.

Phase 1: Isometric loading

Isometric resistance exercises reduce tendinopathy pain¹³. Exercises should be performed several times a day and preferably performed with the hip in a near-neutral flexion-extension position to minimize compressive forces to the proximal hamstring tendons.

Phase 2: Concentric-eccentric loading with limited hip flexion

Historically, eccentric exercises have been advocated as the exercise modality of choice for tendinopathy. However, more recent research indicates that heavy slow

resistance strength exercises (that include both concentric and eccentric components) have similar positive effects compared to isolated eccentric training in tendinopathy, and achieve greater collagen turnover than sub-maximal eccentric loading^{14,15}. Exercises should be performed with minimal hip flexion (e.g. single leg bridge, prone leg curl, Nordic hamstring exercises) to minimize compressive tendon loads.

Phase 3: Concentric-eccentric loading with increased hip flexion

In this phase, heavy slow resistance strength training is progressed to exercises with increasing hip flexion positions (e.g. lunges, the Diver, Romanian deadlift).

Phase 4: Energy storage and release loading

Exercises that require energy storage and release (e.g. plyometric exercises, running, jumping) put the tendons under the highest loads and should only be introduced in this final phase. The amount of hip flexion position in exercises can now be progressed gradually to increase the compressive loads to the enthesis.

Like every other injury the rehabilitation should be finalized with a sport-specific (on-field) phase that reflects the functional demands of the specific sport.

Non-steroidal anti-inflammatory drugs

Whether non-steroidal anti-inflammatory drugs (NSAIDs) affect tendon healing (either negatively or positively) is currently unknown. As a general rule we do not use NSAIDs in PHT in our clinical practise. The only exception is perhaps in athletes with a significant flare-up of tendon pain that compromises adequate loading. NSAIDs for several days and up to two weeks might support the (re)start of the exercise program.

Extracorporeal Shockwave Therapy

Extracorporeal Shockwave Therapy (ESWT) may be considered as an adjunct to exercise treatment. ESWT resulted in a superior decrease in pain compared to the control group that received traditional non-operative treatment in a randomised controlled trial by Cacchio et al. The mean pain score decreased from 7.1 (±1.1) to 3.2 (±1.6) in the ESWT group with a 80% return to sport rate at three months follow-up compared to no decrease in pain score in the control group and no athletes returning to



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sport¹⁶. The seemingly large positive effect of ESWT is compromised by a substantial risk of bias due to the lack of blinding.

Our clinical ESWT experience is only a moderate decrease in pain. As the exact mechanism and biological effect of ESWT remains elusive, we reserve ESWT for PHT patients with insufficient progression after three months of proper rehabilitation, emphasizing that ESWT is an adjunct to exercise, and not a short-cut to success.

More robust randomised double-blind clinical trials are needed to establish the effect of ESWT in PHT.

Avoid corticosteroid injections: a quick relief with bad consequences

We therefore strongly argue against the use of corticosteroids in PHT. Corticosteroids have been widely used for all tendinopathic conditions due to its possible short-term pain reduction. However, a growing body of high-quality studies provide strong evidence that corticosteroid injections have a deleterious mid- and long-term effects in tendinopathy¹⁷.

Platelet-rich plasma injections

Platelet Rich Plasma (PRP) injections have become increasingly popular as a treatment for tendinopathy, partly because of its possible effects on tendon healing in basic science studies. However, the current industry-driven popularity is mainly clever marketing strategies, rather than a solid

evidence base. There is no evidence that PRP improves imaging findings or patient-reported symptoms in tendinopathy¹⁸. Only one randomised trial compared PRP with whole blood injections in PHT and found no difference between the interventions¹⁹. There are no clinical trials comparing PRP with placebo or no injection in PHT.

Surgery

Surgery is suggested by some as an option in patients that do not respond to non-operative management. However, in PHT the evidence is limited to a single case series²⁰. We have therefore never referred or advised any patient for surgical intervention in our clinical practise, due to the lack of evidence for such an invasive intervention.

CONCLUSION

We described the clinical management of PHT, based on the limited available evidence and our clinical experience.

The diagnosis is primarily clinical, based on a history of high hamstring or gluteal pain that is reproduced during physical examination by applying compressive and tensile loads to the proximal hamstring tendons. Imaging has very limited value.

Patient education and expectation management are key. We consider pain management by load modification and progressive exercise rehabilitation as the cornerstones of PHT treatment. We restrict NSAIDs to patients with

aggravated symptoms that makes adequate rehabilitation loading impossible. ESWT can be used as an adjunct to the progressive load program.

We do not advocate the use of any other (invasive) treatments; there is insufficient evidence to support PRP injections and surgery, and strong evidence against corticosteroid injections due to its harmful mid- to long-term effects.

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