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Review

Current and future advances in practice: tendinopathies of the hip

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Abstract

Tendinopathy describes persistent tendon pain and loss of function related to mechanical loading. Two common hip tendinopathies seen in practice are gluteal tendinopathy and proximal hamstring tendinopathy. Both conditions can be frustrating for patients and clinicians due to the delay in diagnosis, significant disability caused and lack of response to common treatments. Tendinopathy is a clinical diagnosis and can most often be made using findings from the patient interview and pain provocation tests, without the need for imaging. Specific education and progressive exercise offer a low-risk and effective option for gluteal tendinopathy and result in greater rates of treatment success than corticosteroid injection, both in the short term (8 weeks) and at 1 year. Proximal hamstring tendinopathy is a common, but less researched, and under-recognized cause of persistent ischial pain. As research on proximal hamstring tendinopathy is limited, this review summarizes the available evidence on diagnosis and treatment following similar principles to other well-researched tendinopathies.

Lay Summary

This review summarizes the diagnosis and management of two common hip tendinopathies, gluteal tendinopathy and proximal hamstring tendinopathy. Gluteal tendinopathy, formerly trochanteric bursitis, is a common source of pain over the side of the hip. This condition is common in postmenopausal women and significantly affects physical function and quality of life. Advances in diagnostic testing and evidence-based treatment programmes have provided clinicians with promising options for the management of gluteal tendinopathy. Education aimed at improving patient understanding of aggravating factors, combined with functional rehabilitation, has been found to have greater treatment success than corticosteroid injection. Proximal hamstring tendinopathy is seen in both athletic and non-athletic populations, with the main symptom being pain at the sitting bone that interferes with sitting and day-to-day athletic function. As research on proximal hamstring tendinopathy is limited, this review summarizes the available evidence on diagnosis and treatment following similar principles to other well-researched tendinopathies.

Keywords: gluteal tendinopathy, greater trochanteric pain syndrome, GTPS, lateral hip pain, buttock pain, buttocks, proximal hamstring tendinopathy, hamstring tendons

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Key messages

- Hip tendinopathies are prevalent, impactful and not self-limiting.
- Palpation is not sufficient for the diagnosis of hip tendinopathies.
- Evidence informs that combining findings from the patient interview, palpation and active tests improves diagnostic accuracy.
- Cortisone injections are a common 'quick-fix' that are no better in the longer term than awaiting spontaneous recovery—and may have adverse tissue and behavioural effects.
- An active approach is preferable for tendinopathy management. High-quality evidence exists for education and exercise as the best first-line management for gluteal tendinopathy.

Introduction

Tendinopathy is defined as persistent tendon pain and dysfunction related to mechanical loading [1]. Studies from the Netherlands and Denmark have found prevalence rates of lower limb tendinopathies average approximately two per 100 patient presentations [2, 3], and increase with age, and female sex [3]. Gluteal tendinopathy (GT) is the most common lower limb tendinopathy, affecting up to 23.5% of middle-aged women [4]. Often a cause of moderate to severe pain that interferes with sleep and physical function, levels of disability and quality-of-life in people with GT equate to that of end-stage hip osteoarthritis [5].

Gluteal tendinopathy is inconsistently identified/diagnosed—commonly diagnosed as 'trochanteric bursitis'. This results in suboptimal management. 'Bursitis' implies underlying inflammation, which has traditionally been treated with passive strategies such as rest, electrotherapy, non-steroidal anti-inflammatory medications and corticosteroid injections (CSIs) [6]. Although it may co-exist with tendon pathology, isolated bursal pathology is uncommon, and inflammatory cells are rarely present [7, 8]. Structural change in the iliotibial band (ITB) also occurs in some individuals with trochanteric pain [9]. The primary pathology for GT is now understood to be a non-inflammatory insertional tendinopathy of the gluteus medius and/or gluteus minimus [7, 10, 11]. Despite this, many clinicians continue to target inflammation and deliver passive, 'quick-fix', low-value interventions [12].

The term Greater Trochanteric Pain Syndrome (GTPS) is commonly used as an umbrella term to encompass all soft tissue pathologies at the greater trochanter, but definitions in the literature are variable and treatment direction for a 'syndrome' is often unclear. Here, we use the term 'gluteal tendinopathy', a diagnosis that engenders more active treatment approaches, which will also benefit those with accompanying bursal or ITB change.

Proximal hamstring tendinopathy (PHT) is also common and, in the authors' clinical experience, may occur concurrently with GT. It affects both less active individuals [13], usually perimenopausal women [14], as well as athletes involved in running sports (e.g. prevalence of 12% in distance runners [15]). The primary pathology is insertional tendinopathy of the proximal hamstring tendon(s), predominantly semimembranosus [16]. This pain reduces the ability to sit for prolonged periods [17], can impact athletic performance [18] and has significant occupational and social impacts. PHT is often misdiagnosed due to the complex anatomy of the buttock region, referred pain from other structures and limited awareness of the injury [19].

This review highlights recent advancements and future directions in assessing and managing these common hip tendinopathies and the important clinical implications for the Rheumatologist.

Pathoaetiological factors associated with the development of hip tendinopathy

Pathogenesis of tendinopathy is a multifactorial process, and several theories exist regarding contributing factors [20]. For a comprehensive review, see Millar et al. (2021). Rather than a neat alignment, in tendinopathy collagen fibres become disorganized, with an accumulation of glycosaminoglycans, dysregulated extracellular matrix homeostasis, greater microvascularity and neo-innervation [21], leading to loss of tendon integrity. Mechanical loading is an important stimulus for catabolic and anabolic tendon processes, with overload and underload affecting tendon integrity.

Mechanical factors

Occupational and sport-related factors such as overuse, sudden increase in intensity of activity, inadequate recovery, highly repetitive movement and poor ergonomics predispose to the development of tendinopathy [20]. This may also occur in less active/sedentary populations.

Specific types of load may adversely impact tendon health. A combination of high tensile and compressive load is most detrimental [22], particularly in repetitive situations. Conversely, the combination of low tensile loads and compression can compromise load capacity and predispose to pain—even at low activity levels [23].

Tendon loads are influenced by joint position and bony morphology. In GT, a more acute femoral neck-shaft angle may contribute to compressive forces by altering trochanteric offset [24, 25], also reflected in greater differences between iliac wing width and trochanteric width in this population [26] (Fig. 1). ITB compression over the trochanteric soft tissues can also be increased by joint positions such as hip adduction (e.g., sitting with legs crossed) [24].

Loads imposed on the proximal hamstring tendons are influenced by sagittal plane hip joint position, as they are compressed against the ischium in positions of hip flexion, such as sitting and lunging [27]. The semimembranosus tendon is thought to be most vulnerable to compression due to its deep, lateral origin on the ischium [28].

Other factors

Age, sex and hormonal status are also risk factors [4], evidenced by a high prevalence in post-menopausal women. Advancing age is associated with slower tendon metabolism and poorer regenerative capacity. The ensuing poor collagen content impairs mechanical properties and healing. [29].
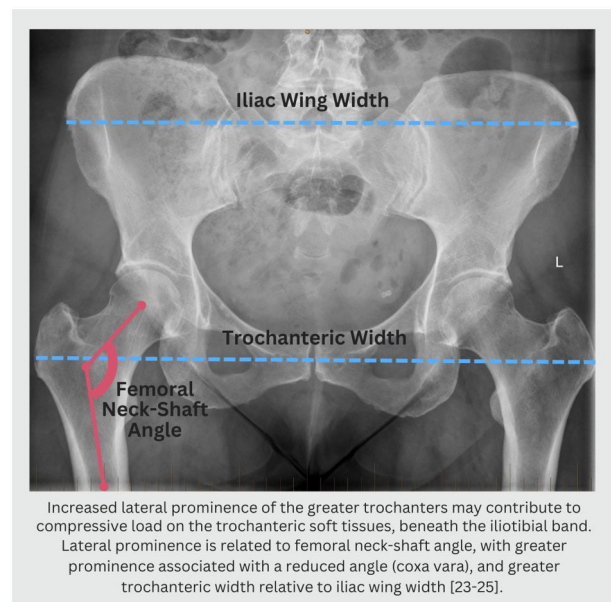

Increased lateral prominence of the greater trochanters may contribute to compressive load on the trochanteric soft tissues, beneath the iliotibial band. Lateral prominence is related to femoral neck-shaft angle, with greater prominence associated with a reduced angle (coxa vara), and greater trochanteric width relative to iliac wing width [23-25].

Figure 1. Bony features associated with gluteal tendinopathy

Reduced oestrogen levels are associated with reduced collagen synthesis and tensile strength, increasing the risk of tendon rupture [30].

Metabolic risk factors (e.g. obesity, hypercholesterolaemia and diabetes mellitus), have been associated with tendinopathy. Obesity may play a role both systemically and mechanically [31], and body mass index (BMI) is reportedly higher in people with GT than asymptomatic controls [32]. Genetic factors have also been associated with tendon injury; however, most studies have been performed in Achilles [33] and rotator cuff tendinopathies [20].

Various medications have been implicated. Fluoroquinolones can cause pathologic changes in tendon tissue, predisposing to tendinopathy and tendon rupture [34]. The Achilles tendon is the most affected site and is commonly affected bilaterally (44% of cases) [35]. Whilst incidence appears low (rates of 2.4 per 10 000 patient prescriptions for tendinitis and 1.2 per 10 000 for tendon rupture [34]), the risk may be higher in patients with certain comorbidities and magnify other known risk factors (chronic renal failure, diabetes, obesity and systemic corticosteroid use). Other drug types implicated in the development of tendinopathies include statins, glucocorticoids, aromatase inhibitors and anabolic steroids [35].

Assessment of gluteal tendinopathy

Assessment of patients with lateral hip pain involves making a diagnosis, negating other diagnoses, and an assessment of aetiological contributors and/or targets for intervention.

Differential diagnosis of lateral hip pain

The most common conditions associated with lateral hip pain are GT, hip osteoarthritis and lumbar radicular pain. The patient interview provides important clues for the differential diagnosis and directs the subsequent physical examination. Imaging may be required where the diagnosis is unclear, and the patient fails to progress.

Patient interview features

The features most useful in differentiating GT are the area of pain, behaviour of symptoms and absence of other features that are more indicative of hip osteoarthritis or lumbar-related pain (Fig. 2) [36]. Dermatomal pain distribution with paraesthesia is more likely to reflect radicular pain. Lumbar somatic referral may also occur in the lateral hip region (See Fig. 6 for features of somatic referral). GT is characterized by pain and tenderness over the greater trochanter, sometimes extending down the lateral thigh and upper leg, that impacts sleep and causes functional difficulties associated with pain on single-leg loading (stair-climbing, walking, dressing). It is important to note that patients may present with multiple sources of lateral hip pain, necessitating a comprehensive assessment to identify and address all contributing factors.

Diagnostic tests for gluteal tendinopathy

To diagnose GT, clinicians can perform palpation of the greater trochanter and various physical tests that load the gluteal tendons [37]. A recent meta-analysis of tests for GTPS [38] found that trochanteric tenderness alone is not adequate for diagnosis, but combining palpation with resisted hip abduction significantly increased the post-test probability of both a positive and negative diagnosis of GTPS. Adding the

30-second single leg stance test helps confirm a positive diagnosis (Fig. 3).

The role of imaging

Clinical examination is usually sufficient for diagnosing GT, with imaging reserved for cases of persistent symptoms. MRI is considered the gold standard for visualizing the gluteal tendons and adjacent soft tissues [7]. It can also provide information on abductor muscle size, quality and other potential pathologies. Ultrasound has also been shown to have good accuracy (US 72% vs MRI 65.2%) and sensitivity (US 89.5% vs MRI 64.7%), but poor specificity (US 16.7% vs MRI 66.7%) in detecting gluteus medius tendon pathology [39]. It is often user-dependent and focuses on an area of pain rather than a global assessment of the region of concern. It is important to note that imaging abnormalities are frequently present in individuals without symptoms [40], highlighting the need for matching imaging findings with the clinical findings.

Other clinical features

Recent work has highlighted impairments in muscle size, quality and function that may be targets for intervention, and psychological characteristics to be considered in the management plan. Patients with GT exhibit significant bilateral hip abductor muscle weakness, even in unilateral presentations [32, 41]. Abductor muscle weakness is accompanied by gluteal atrophy and fatty infiltration [42]. Fine-wire electromyography studies have reported alterations in abductor muscle behaviour during gait [41, 43] and biomechanical studies demonstrate altered gait characteristics that lead to substantially higher loads on the abductor tendons [44]. These impairments may be addressed through rehabilitative exercise.

Psychological factors may impact pain levels, disability, and treatment outcomes. Whilst isolated GT is not typically associated with high levels of psychological distress, patients with higher pain levels and poorer function are more likely to experience psychological distress [45]. Addressing pain, sleep disturbances and functional disability can help alleviate condition-related psychological distress, but some patients may require psychologically-informed rehabilitation or formal psychological intervention. Additionally, patients with multiple concurrent pain conditions may have greater distress [46] and a need for a multidisciplinary approach.

Management of gluteal tendinopathy

Early management of GT is important. Whilst passive interventions such as CSI can provide immediate pain relief, they may compromise tissue health, and the effectiveness of subsequent interventions, and alter patient expectations of care. Substantial advances in understanding and treatment approaches have recently culminated in the high-quality LEAP randomized clinical trial that showed education and exercise to be superior to CSI [47].

Education and exercise approaches

Educational content provided to those with GT aims to educate patients about their condition and implement specific load management strategies (Fig. 4) focusing on gradual progressive increases in load and reduced exposure to positions that compress the tendon. One specific target is reducing sustained or repetitive hip adduction in sitting, standing, sleeping, stretching, and dynamic function [48].

Figure 2. Patient interview features of three key differential diagnoses for lateral hip pain

Exercise therapy involves supervised exercise and/or a home programme, and commonly includes a combination of isometric hip abduction, exercises to improve frontal plane femoropelvic control, heavy slow abductor loading and lower kinetic chain conditioning [47, 49].

The LEAP trial compared the effects of education and exercises against CSI and wait-and-see [47]. The education and exercise group showed significantly higher success rates reflected in the Global Rating of Change measures, than the CSI and wait-and-see groups in both the short (8 weeks) and long term (52 weeks) (Fig. 5). CSI was superior to wait-and-see in the short term, but there was no difference in success rates at 52 weeks. Pain intensity was reduced most in the short term by education and exercise. In the longer-term, pain intensity was not different between exercise and CSI groups, but improvements in pain constancy, and quality-of-life were significantly better in the education and exercise group [47]. Economic analysis, using quality-adjusted life
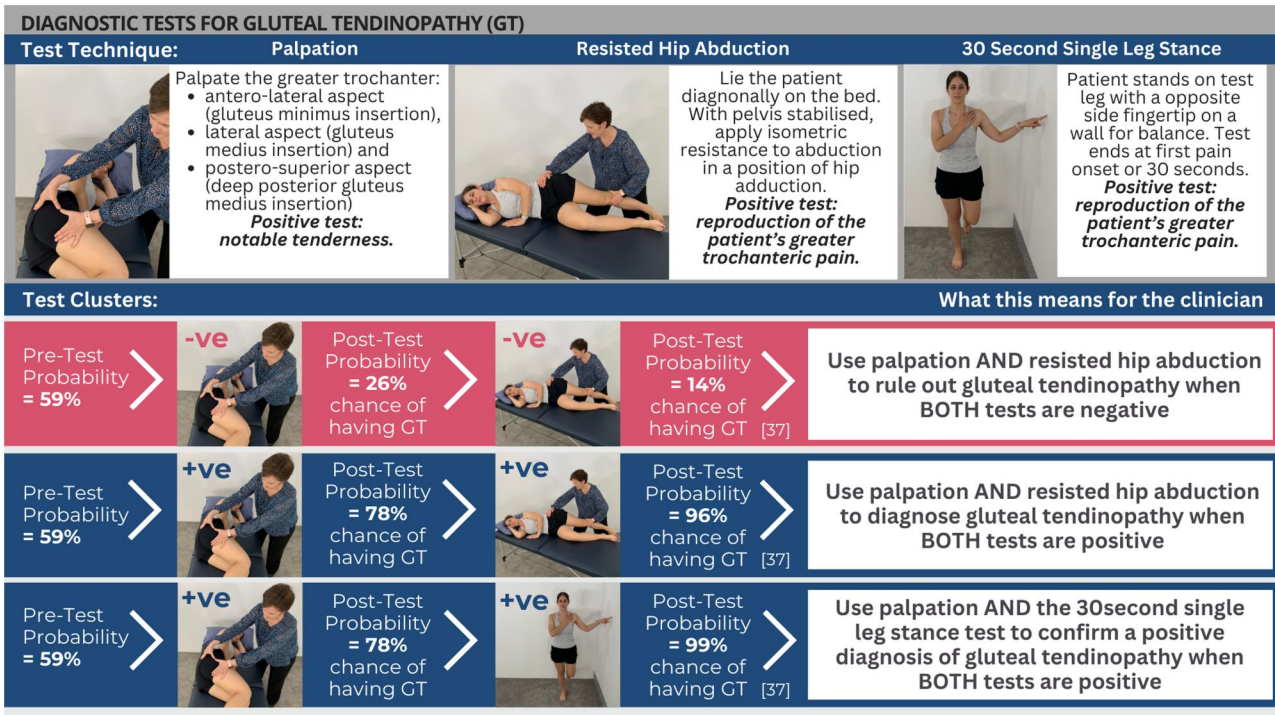
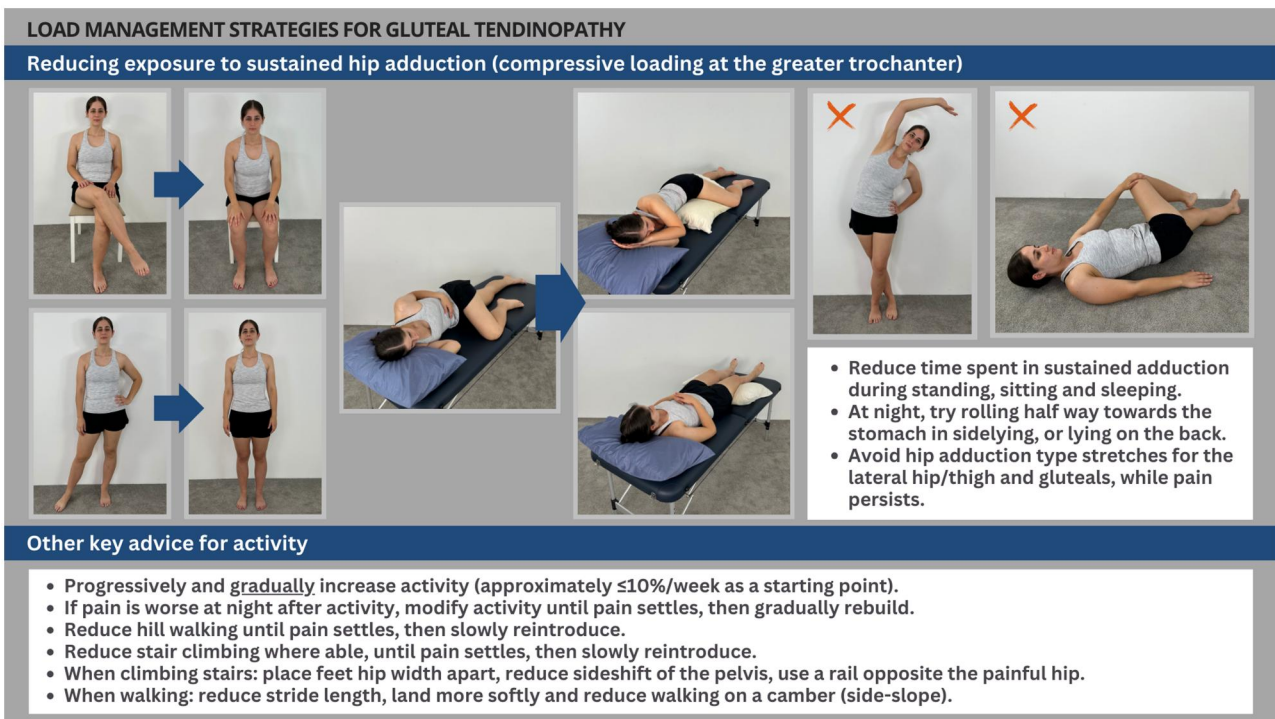**Figure 3.** Diagnostic tests for gluteal tendinopathy

Figure 4. Load management strategies for gluteal tendinopathy

years and total economic costs, also supports education and exercise as a superior cost-effective intervention compared with CSI and wait-and-see groups [50].

Injection therapies

Despite the high-quality evidence supporting education and exercise as first-line management for GT, our pilot data and observations from the clinic suggest that CSI remains a common initial medical treatment for trochanteric pain in general medical practice. The rationale for CSI has traditionally been to address inflammatory processes underpinning 'trochanteric bursitis', although histological studies have shown an absence of typical cellular inflammation [8]. Corticosteroids appear to be potent short-term analgesics,
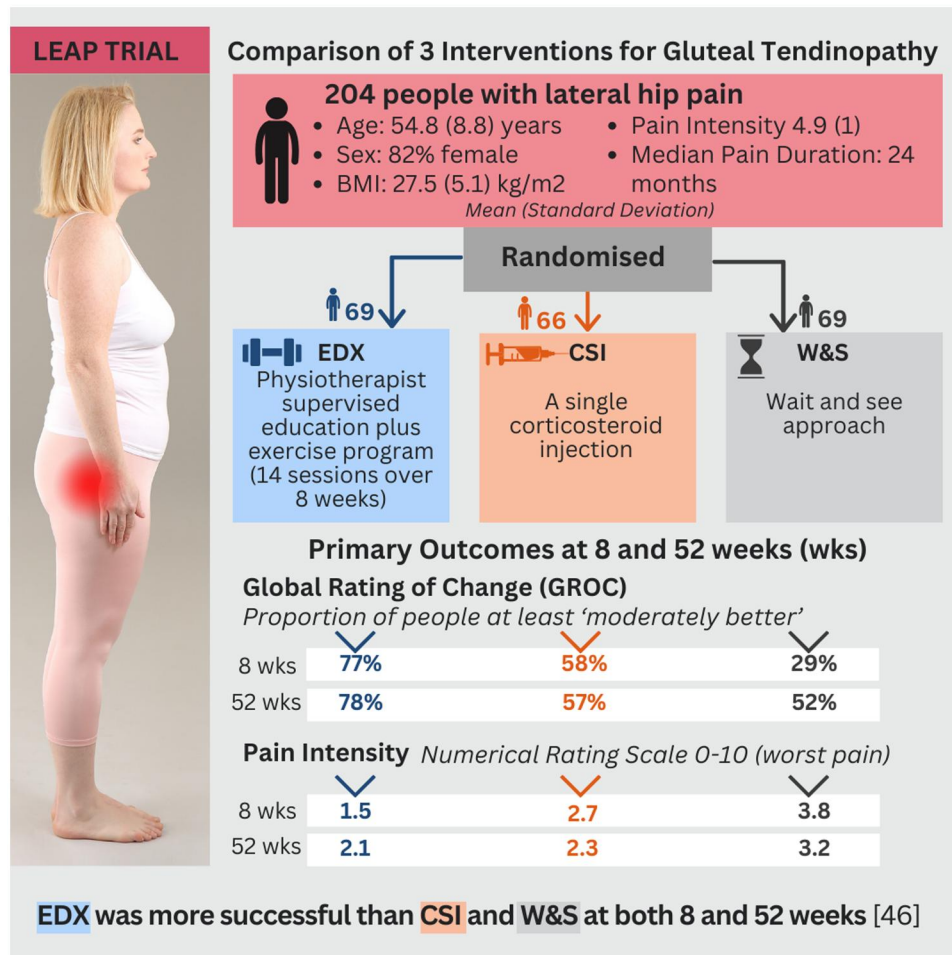
LEAP TRIAL **Comparison of 3 Interventions for Gluteal Tendinopathy**

204 people with lateral hip pain
- Age: 54.8 (8.8) years
- Sex: 82% female
- BMI: 27.5 (5.1) kg/m2
- Pain Intensity 4.9 (1)
- Median Pain Duration: 24 months

Mean (Standard Deviation)

Randomised

69 → **EDX** Physiotherapist supervised education plus exercise program (14 sessions over 8 weeks)

66 → **CSI** A single corticosteroid injection

69 → **W&S** Wait and see approach

Primary Outcomes at 8 and 52 weeks (wks)

Global Rating of Change (GROC)
Proportion of people at least 'moderately better'

	EDX	CSI	W&S
8 wks	77%	58%	29%
52 wks	78%	57%	52%

Pain Intensity *Numerical Rating Scale 0-10 (worst pain)*

	EDX	CSI	W&S
8 wks	1.5	2.7	3.8
52 wks	2.1	2.3	3.2

EDX was more successful than **CSI** and **W&S** at both 8 and 52 weeks [46]

Figure 5. LEAP clinical trial outcomes for management of gluteal tendinopathy

but long-term benefits are usually no better than wait-and-see or other treatments [47, 51, 52]. One study compared CSI against placebo saline injection [53], with no significant between-group differences at 4 weeks or 6 months, concluding that CSI is of limited benefit, considering poor long-term outcomes and potential side effects.

Corticosteroids have adverse biological effects on tendon health, reducing cell viability, collagen synthesis, and mechanical properties of the tendon [54], potentially impacting success of concurrent exercise-based interventions. Combining CSI with rehabilitation may delay and reduce long-term outcomes of physiotherapist-led interventions for tendinopathy [55]. Behavioural effects may also contribute to poorer outcomes. The immediate pain relief CSI provides appears to reduce mindful attention and priority patients place on implementing load management strategies, movement pattern modifications and their prescribed exercise programme. Furthermore, despite the effect waning after a few weeks, the rapid pain reduction imparted by CSI often results in the patient seeking repeat CSI over an active intervention. Although CSI is often provided as a 'helpful adjunct' to rehabilitation, it's likely to be more of a hindrance.

Platelet-rich plasma (PRP) injection has emerged more recently as an alternative to CSI [57]. PRP is believed to induce tendon healing through the delivery of platelet-derived growth factors [56], although evidence of tendon healing in humans is lacking. PRP injections have shown positive effects

on pain and disability in tendinopathy, but variable study methodologies make interpretation difficult [57]. Limited evidence, in the form of one trial, currently suggests that PRP for GT may be more effective than CSI in the medium to long term, when provided together with a home programme of advice and exercise [58]. No high-quality evidence exists for PRP against wait-and-see or education and exercise alone.

Shock wave therapy

Shockwave therapy (SWT) is another treatment for GT. This modality applies mechanical impulses to the trochanteric soft tissues to theoretically stimulate a healing response [59]. Low-level evidence exists for pain reduction in the medium-term, following SWT for GT [59]. However, associated pain and bruising often experienced with SWT and cost of multiple sessions should be considered. An education and exercise approach should be first-line management, with SWT reserved for refractory cases.

Hormone therapy

Hormone therapy aims to address the effect of waning oestrogen levels on tendon health in post-menopausal women. Only one study to date has explored the addition of transdermal hormone therapy, or a placebo cream, to an education and exercise intervention for GT [30]. Despite a lack of between-group effect, a responder analysis revealed that those with a BMI ≤ 25 who used the hormone cream had
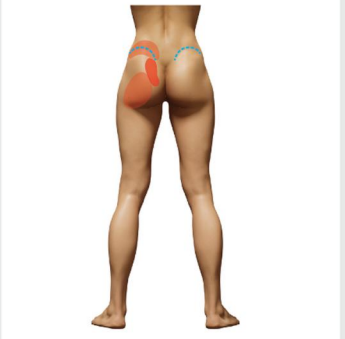
Figure 6. Patient interview features of three key differential diagnoses for buttock pain. (The information in this figure represents a combination of features outlined in the available literature and the authors' clinical experience.)

significantly better outcomes compared with the placebo cream group, suggesting that sublingual hormone therapy may be more effective due to higher levels of serum oestradiol and greater absorption of progesterone. Further investigation is needed, but this early evidence suggests that transdermal hormone therapy may benefit individuals with lower BMI when combined with an education and exercise programme.

Surgical interventions

Surgical interventions for GT are typically considered when non-surgical methods fail to provide satisfactory results.

Whilst surgical studies show promising outcomes [60], the level of evidence is low due to research design limitations. Based on available evidence, both open and endoscopic gluteal tendon repair yield similar outcomes in terms of patient-reported scores, pain relief, and improvement in abduction strength [61, 62]. Open techniques have a higher reported complication rate [61].

Further high-quality studies are needed to clarify the effects of surgery and identify patients most likely to benefit. Studies investigating common pathways to surgery—i.e., previous treatments and effects—are also required. Multiple CSIs are common before surgery [63, 64]. Whilst it is appropriate to exhaust non-surgical interventions before surgery, the question arises whether multiple CSIs increase the likelihood of subsequent surgery due to the potential reduction in tissue quality and effects of education and exercise interventions. Higher numbers of previous CSIs are also a prognostic factor for poor clinical outcomes following endoscopic surgery for GTPS [65]. Thus, healthcare professionals should consider longer-term implications when advising patients on early treatment options.

Assessment of proximal hamstring tendinopathy

The patient interview and subsequent physical examination drive diagnosis of patients presenting with buttock pain. Imaging is helpful where the diagnosis is unclear, or the patient's progress is stagnant.

Differential diagnosis of buttock pain

Common conditions associated with buttock pain include injuries to the proximal hamstring tendon-complex, somatic referral from the lumbar spine, hip joint or sacroiliac joint, radiculopathy, entrapment of the sciatic nerve and ischiofemoral impingement [66]. Stress fractures of the sacrum and pelvis, whilst rare, should also be considered, as PHT is common in runners and triathletes.

Patient interview features

The key concern raised by patients with PHT is localized ischial pain that interferes with sitting and restricts physical activity [67]. Symptoms can often be traced back to an increase in activity, particularly those involving significant hamstring activity in flexed-hip positions (e.g. hill-walking/running or gardening) [67, 68]. Symptoms are worse with higher hamstring loads, such as running at faster speeds [16]. Direct compression against the ischial tuberosity is also provocative, particularly when sitting on harder surfaces [18].

The well-localized nature of symptoms in PHT assists with differential diagnosis. Referred pain from the lumbar spine, hip or sacroiliac joints is typically more diffuse (Fig. 6) [69–71]. Symptoms extending from the ischium towards the knee may occur, but this is not a key feature of PHT [68]. Reports of morning or joint stiffness are not a feature of isolated PHT and should inform inspection of the spine, hip joint or consideration of systemic inflammatory disease. Enthesitis at the bone-tendon junction is less common in the pelvis than in the Achilles and plantar fascia; however, it should still be considered [72] and may be under-reported. Suspicion should be present in young adults reporting hip and/or back morning stiffness in combination with ischial pain.

Partial or complete proximal hamstring ruptures may occur, usually after acute trauma involving end-range hip flexion and knee extension, accompanied by an audible pop [73]. In younger athletes, avulsion fractures of the ischial tuberosity and apophyseal overuse injuries should also be considered [74].
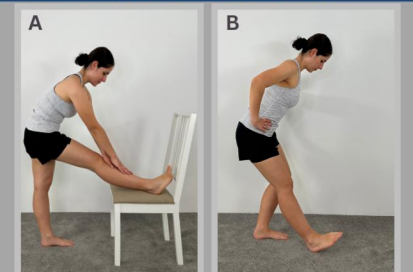

Figure 7. Diagnostic tests for proximal hamstring tendinopathy

Diagnostic tests for proximal hamstring tendinopathy

In those with PHT, clinical examination reveals localized is-chial pain with resisted knee flexion that worsens in greater ranges of hip flexion [67] (Fig. 7). Research on diagnostic utility of clinical tests is limited in PHT [75, 76]. A single study tested three passive stretch-based tests (Puranen-Orava test, bent-knee stretch test, modified bent-knee test) [75]. All tests demonstrated high positive likelihood ratios and low negative likelihood ratios. These findings are likely overesti-mated, as the comparison group were healthy participants. Further research is required using patients with mixed causes of buttock pain to understand the clinical utility of differ-ent tests.

Palpation of the hamstring origin is more challenging than the gluteal tendons, as the hamstring origin is deep to the overlying gluteus maximus. The diagnostic utility of palpa-tion in diagnosing PHT is unknown, but palpation is used frequently in the diagnostic workup [19, 77]. The ischium is more easily accessed in side-lying with hips flexed and should be performed methodically to ensure the entire footprint of the hamstring origin is palpated. The average width of the footprint of the conjoined tendon on the ischial tuberosity is approximately 3.4 cm and the semimembranosus 4.2 cm [28], with the sciatic nerve sitting 1.2 cm from the most lateral as-pect of the ischial tuberosity [78].

The role of imaging

Diagnosis of PHT doesn't always require imaging [68], but where necessary, MRI is most valuable in differential diagno-sis. MRI has also been shown to be more sensitive than ultra-sound in detecting abnormalities in the proximal hamstring complex [79]. As with GT, imaging findings must be inter-preted in conjunction with clinical findings, as pathology of the proximal hamstring complex is common in asymptomatic individuals [80], and older people [80, 81].

Other clinical features
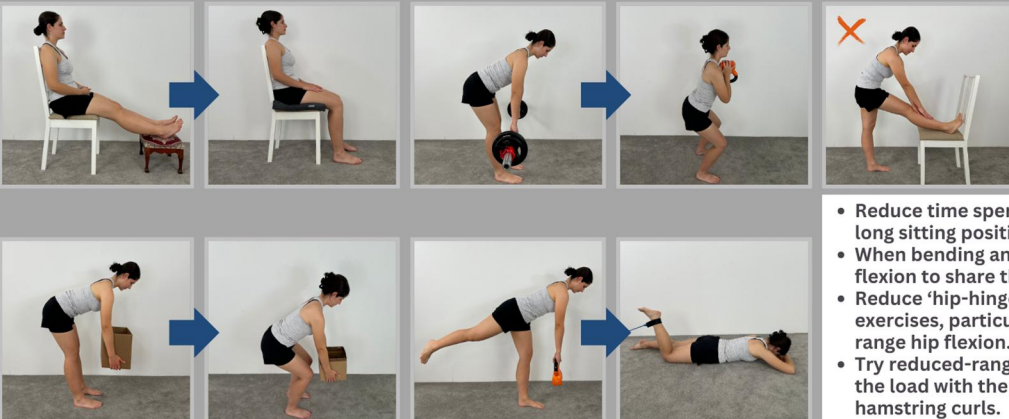
Restrictions in hamstring muscle length may or may not be present and relevance depends on individual patient require-ments. Reports on strength deficits accompanying the presen-tation of PHT are inconsistent [13, 77], but reductions in knee flexion and hip extension strength are often marked in long-standing cases. Running technique variations have been associated with PHT, including over-striding, poor control of anterior pelvic tilt and poor coronal plane stability [67]. Similar gait patterns may also exist, particularly over-striding, in walkers with PHT. Gait training, such as increas-ing cadence, can be a useful accompaniment to rehabilitation.

Management of proximal hamstring tendinopathy

Various interventions for PHT have been explored in the lit-erature, including exercise-based rehabilitation, SWT, inject-ables such as PRP and CSI, and surgery [82]. Unfortunately, the quality of evidence supporting any intervention is, at best low, with most studies limited to case series without control groups [82]. Hence, the current best practice treatment of PHT largely involves adopting evidence from other tendin-opathy research where higher-quality studies have been com-pleted [47, 83, 84]. As such, education regarding activity modification and the prescription of a targeted exercise inter-vention to improve the health of the affected tendon and mus-culature, and functional capacity of the individual is generally considered first-line management [67, 68].

Figure 8. Load management strategies for proximal hamstring tendinopathy

Education and exercise approaches

Early management aims to achieve symptom control, primarily through education regarding limiting high hamstring loads, particularly activities involving high hip flexion [68] (Fig. 8). Examples include reducing higher-speed running and removing or altering provocative exercises (e.g., deadlifts, lunges, deep squats). Maintenance of some running may be tolerated in less irritable cases, often at lower speeds. Ischial pain from direct compression during sitting may be relieved through adjustments in tilt and height of the chair, as well as sitting on softer surfaces/cushions. Whilst some improvements in symptoms and function are common over weeks and months, sitting pain is often slow to resolve and may persist for up to a year. Discussing appropriate expectations may reduce the pursuit of additional treatments [68].

In conjunction with load management and activity modification, early rehabilitation should include exercises that target the hamstring muscle-tendon unit [67, 68]. Several case series, providing low-quality evidence, have reported success with progressive strengthening of the hamstring unit, mirroring modern management options for more researched tendinopathies [85, 86]. A key consideration of exercise selection should be avoiding significant hip flexion range and performance of exercise in a slow, controlled manner.

Often resistance can be progressed quickly for exercises involving minimal hip flexion (e.g., prone hamstring curl), whereas increasing range into exercises involving significant hip flexion is often slower and generally takes months (e.g., lunge). Rehabilitation should also target the synergists of the hamstring, such as the gluteus maximus, and other muscles in the kinetic chain to assist with load sharing [68].

Injection therapies

The quality of research on PRP injection in PHT is low. A small RCT ($n = 15$) compared PRP to autologous whole blood injections [87] and found no significant difference between interventions at 6 weeks, 12 weeks or 6 months on physical function or quality of life. Additional limitations include small sample size and use of outcome measures traditionally designed for hip joint rather than tendon conditions [88]. Several other case series have reported on PRP, with the largest ($n = 29$) showing a small but not clinically meaningful improvement in physical function at 8-week follow-up [89]. Data on adverse events following PRP is limited in PHT, with rates varying from 0–10%, including high levels of pain post-injection and sciatic nerve irritation.

Two retrospective case series found a single CSI resulted in short-term improvement in symptoms that were not maintained in the long term [79]. Whilst no adverse events were reported, CSI should be used judiciously due to the potential negative effects on tendon health and rehabilitation discussed above [54].

Shockwave therapy

A single small RCT ($n = 40$) compared the effect of SWT to a 6-week multi-modal programme consisting of 3 weeks of exercise, NSAIDs and therapeutic ultrasound. SWT was found to have a large positive effect on symptoms and function over the multi-modal programme in both the short and long terms. Further research, with a larger sample size, is currently underway, which will help better understand SWT's efficacy in PHT [90].

Surgery

Surgical intervention is reserved for cases of recalcitrant PHT. Acute partial and full-thickness hamstring ruptures are distinct from PHT and are more commonly managed surgically [91]. Information on predicting who is likely to benefit from surgery for PHT is unknown [19, 92]. Several surgical studies report adhesions between the sciatic nerve and proximal hamstring region [19, 77]—in such cases, nerve symptoms [16] may be improved by surgical liberation of the sciatic nerve.

Surgical case series report promising changes in symptoms and physical function [19, 77]. As with GT, the quality of evidence is low, with a high risk of bias [82]. Current recommendations suggest that surgery should only be entertained after 12 months of an evidence-based loading programme has been trialled [93].

Future directions

Further studies are currently underway on GT and PHT [90], which will improve the understanding of the efficacy of non-invasive treatments, including the impact of the LEAP trial in other healthcare settings [94]. Recent studies suggest that patient-reported outcome measures used to assess the impact of GT and PHT [88] need urgent refinement (or re-development). Important work has started on the development of a core outcome set for use in all future clinical trials to ensure results can be pooled in meta-analyses and measures can be better trusted to capture changes in the condition.

Further research is also needed to improve understanding of the pathway to surgery. This may identify important milestones during a patient's journey and help determine who is most likely to benefit from surgical procedures. Future research on the impact of surgery should ideally involve control groups or comparisons with alternative interventions.

Concluding remarks from a rheumatologist's perspective

Hip tendinopathy is a common presentation. At present, hip tendinopathies are frequently misdiagnosed and poorly managed. There is a predilection for ordering expensive investigations (particularly MRIs) in a population of people that frequently will have pathology in areas that contribute to the differential diagnosis—such as hip osteoarthritis and lumbar spine disease. The lack of reliance upon our clinical skills, particularly an adequate history and physical examination, confuses the diagnosis and likely complicates patient care. A predilection to use injections, proven to be no better than saline or usual care, often at the expense of more effective and cost-effective education and exercise approaches is detrimental to our patient outcomes. How do we go about changing practice? The evidence is clear, many efforts at dissemination have been made, at present practice remains studiously either ignorant to this evidence or resistant to change. Measurement of clinical care standards and incentives to provide optimal care should occur.

Data availability

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