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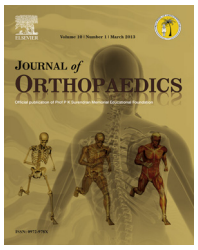

Review Article

The management of greater trochanteric pain syndrome: A systematic literature review☆

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ABSTRACT

Greater trochanteric pain syndrome (GTPS) is a common cause of lateral hip pain. Most cases respond to conservative treatments with a few refractory cases requiring surgical intervention. For many years, this condition was believed to be caused by trochanteric bursitis, with treatments targeting the bursitis. More recently gluteal tendinopathy/tears have been proposed as potential causes. Treatments are consequently developing to target these proposed pathologies. At present there is no defined treatment protocol for GTPS.

The purpose of this systematic literature review is to evaluate the current evidence for the effectiveness of GTPS interventions, both conservative and surgical.

1. Introduction

Localised lateral hip pain with focal point tenderness over the greater trochanter has for many years been clinically diagnosed as trochanteric bursitis.[1,2] The diagnosis of trochanteric bursitis may be inappropriate, given that three of the four cardinal signs of inflammation: rubor, erythema and oedema are uncommon with only pain being a feature.[3,4] Radiological findings for patients with greater trochanteric pain syndrome (GTPS) report variable incidence, with bursitis incidence ranging from 4% to 46% and gluteal tendinopathy ranging from 18% to 50%.[5–7] The preferred clinical term for lateral hip pain is therefore GTPS.[4] GTPS is the term that will be used for this paper.

GTPS encompasses a range of causes including gluteal medius and minimus tendinopathy/tears, trochanteric bursitis and external coxa saltans.[8,9] An exact cause remains unknown.[10] There is often co-existence of both bursitis and tendinopathy.[11] Treatment in the initial stages encompasses a range of conservative interventions including physiotherapy, local corticosteroid injection, PRP injection, shockwave therapy (SWT), activity modification, pain-relief and anti-inflammatory medication and weight reduction. Most cases resolve with conservative measures, with success rates of over 90%.[12,13] GTPS is self limiting for the majority.[14,15] A few cases persist despite treatment and time; these cases are known as refractory cases and may require surgical intervention in the form of bursectomy, iliotibial band (ITB) lengthening techniques or gluteal tendon repair.[2] At present, there is no defined treatment protocol for GTPS.[14,16] The criteria for when surgical intervention for refractory cases of GTPS is indicated are not presently well established.[17] The specific enquiry of this review is to determine the most effective treatment protocol for GTPS.

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1.1. GTPS

GTPS is a clinical diagnosis with typical presentation of chronic intermittent lateral hip/thigh/buttock pain, aggravated with activity and affected side lying position. There is a lack of valid/specific diagnostic criteria for GTPS. The most common examination finding is reproduction of the pain on palpation of the greater trochanter.[18,19]

GTPS affects between 1.8 and 5.6 patients per 1000 per year, more frequent between 40 and 60 years, predominantly female,[4,14] and possibly related to pelvic biomechanics. Females have a larger pelvic width relative to whole body width, with consequent greater prominence of the trochanters and associated increased tension of the ITB over the trochanter.[20] A lower femoral neck shaft angle may also be a predisposing factor, as this increases compression of the gluteus medius tendon on the greater trochanter[21]; increased acetabular anteversion may also be a predisposing factor.[22] The likely cause of GTPS is by repetitive friction between the greater trochanter and ITB, causing repetitive microtrauma of the gluteal tendons that insert into the greater trochanter. This in turn causes local inflammation, degeneration of the tendons and increased tension of the ITB.[23]

Approximately two thirds of individuals with GTPS have co-existing hip joint osteoarthritis or low back pain.[24] Having a higher than normal body mass index is also a likely contributing factor to GTPS.[21]

To determine the most effective management of GTPS, it is essential to have knowledge of the anatomy and proposed pathological processes.

1.2. Anatomy

The greater trochanter is a large quadrangular projection at the junction of the neck of femur with the shaft. It is the main attachment for the strong abductor tendons, which facilitate the complex movement achieved between the abductor mechanism and the bursae. There are approximately 20 bursae in the trochanteric area[25]; some bursae may be acquired due to excessive friction[26] or increased hip offset.[27] Three bursae are consistently present in the majority of individuals. These include the gluteus minimus bursa, located anterosuperiorly to the greater trochanter. The subgluteus medius bursa lies deep to the gluteus medius tendon. The subgluteus maximus bursa is the largest and often described as the 'trochanteric bursa'. This lies lateral to the greater trochanter between the gluteus medius and maximus (Fig. A1).[4,8]

The gluteus medius and minimus form part of the abductor mechanism of the hip joint. They are innervated by the superior gluteal nerve, L5 and S1.[25] The primary function of the posterior part of gluteus medius and gluteus minimus is to stabilise the head of the femur in the acetabulum during movement and gait. The anterior and middle fibres of gluteus medius have a cephalad pull assisting with initiation of abduction. The major hip abductor is tensor fascia lata.[29]

The anterior fibres of the gluteal tendon are under the most force and are consequently seen to separate from the bone first in tears, progressing from anterior to posterior, with the posterior tendon being involved in only the most severe cases.[30] Gluteal tears are present in around 22% of elderly patients.[11] The ITB and tensor fascia lata are another potential cause of GTPS. Together they work as a lateral tension band to resist strains over the greater trochanter.[14]

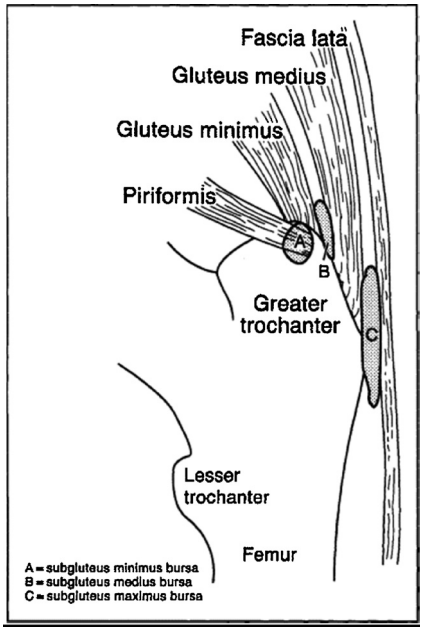

Fig. A1 – Peritrochanteric anatomy.[28]

1.3. Tendinopathy

Gluteal tendinopathy has been identified as a cause of GTPS.[31] Tendinopathy clinically presents as chronic activity related pain and impaired performance of a tendon with or without local tendon swelling.[32] Tendinopathy is characterised by hypercellularity, increased protein synthesis, neovascularisation, disorganisation of the matrix but no inflammation.[33,34] Although recent literature suggests a possible inflammatory component.[35]

The aetiology of tendinopathy is proposed to be multifactorial with both intrinsic and extrinsic components, the exact mechanism is unknown.[32] Repetitive activity is a main factor but tendinopathy can occur in patients without overuse. Different theories of tendinopathy have been suggested, the majority discuss abnormal mechanical loads and altered cellular responses.[36] Other models hypothesis that tendinopathy pathogenesis is related to a 'failed healing' response and is non-inflammatory.[34,37]

Chronic tendinopathies seen on imaging can be asymptomatic, therefore clinical assessment rather than imaging for initial diagnosis and treatment planning of tendinopathy is advocated.[34]

Chronic tendinopathic appearances on imaging show disorganised collagen bundles, neovascularisation and an increase in proteoglycan.[34,38] In tendinopathy there is reduced type 1 collagen and increased type 3 collagen, which has less cross-links and therefore reduces the mechanical strength of the tendon.[34] The chronic pain associated with the pathological changes of tendinopathy may in part be caused by