Gluteal Tendinopathy

Evaluation

* 30-second Single Leg Stance Test
* Hip Lag Sign
* Resisted External De-Rotation Test

IMAGING RECOMMENDATIONS

Management

Soft Tissue

* STM- Gluteals

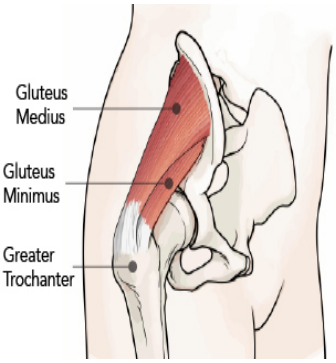
Phase I exercises

* Seated Clam
* Standing Weight Shift
* Glute Bridge w/ Band

Phase II exercises

* Step-ups
* Standing 3 Point Tap

Clinical Pearls



Introduction & etiology

Tendinopathy involving the gluteus medius or gluteus minimus is the most common cause of lateral hip pain. (1, 2) Gluteal tendon tears have been dubbed “rotator cuff tears of the hip” because of their similar prevalence, anatomy, etiology, and management. (3-5)

While the greater trochanteric bursa was once a focal point for lateral hip pain, current thought deemphasizes the concept of “bursitis.” Evidence now suggests that true “bursitis” is rare, and if present, generally occurs secondary to another underlying dysfunction, like gluteal tendinopathy. (6, 7) Only 10-20% of patients with lateral hip pain exhibit bursal enlargement. (2, 8) Even when the bursa is involved, histologic studies fail to show signs of inflammation. (9)

The gluteus medius originates on the iliac crest while the minimus originates on the center of the iliac surface. Both muscles converge to insert on the lateral greater trochanter. The gluteus medius and minimus tendon’s anterior fibers act as hip abductors and internal rotators, while the posterior fibers act as external rotators. (10) Most tears involve the undersurface of the relatively thinner and more vulnerable anterior tendon fibers. (8, 11)

Gluteal tendinopathy (GT) develops from a combination of excessive tension or compression. (12) Similar to rotator cuff tendinopathy, the primary trigger for gluteus medius tendinopathy seems to be compression of the undersurface of the tendon - as most tears begin in this region. (12) Hypothetically, if excessive tensile load were the primary mechanical trigger, tears of the outermost fibers would predominate. While the deeper fibers carry the least tensile load, they are also exposed to the highest compressive loads at their bony insertion. (12) Long-term compressive loads lead to a predictable continuum of ischemia, failed healing, matrix degradation, diminished load-bearing capacity, and failure. (12, 13)

Risk of GT increases with repetitive, ballistic, high-force, or eccentric gluteal contractions. Leg length inequalities would seem to be a predisposing factor; however, at least one study disputes that charge. (14-16) Alteration in the femoral neck - shaft angle has been suggested as a possible predisposing factor for GT (normal angle is 126 - 139°). Obesity is a known trigger for GT. One study found an increase in symptom severity in obese females with a normal femoral neck – shaft angle, but less than <134 degrees. (17)

Lateral hip pain is more common in the second half of life, affecting 6-15% of adults age 50-70. (18) GT affects females up to four times more frequently than males. (18) Lateral hip pain is more common in patients with concurrent lower extremity or lumbar spine dysfunction. Remarkably, 20-35% of low back pain patients suffer concurrent lateral hip pain; with gluteus medius weakness being the likely common denominator. (18, 19)

Clinical presentation

The classic clinical presentation for GT is a post-40-year old with insidious-onset, persistent lateral hip pain that may extend into the buttock and lateral thigh. (20) Muscle activation exacerbates symptoms during walking, climbing stairs or hills, standing on one leg to dress, and following prolonged sitting. (7) Nighttime pain is pervasive in patients with GT. (13) The presence of leg numbness or pain radiating significantly beyond the knee could suggest an alternate diagnosis. (21) Groin crease pain could indicate acetabular pathology, particularly osteoarthritis. (22) Symptomatic impact on ADL’s can be measured and tracked with the VISA-G disability questionnaire.

Clinical evaluation will demonstrate tenderness to palpation over the greater trochanter. Lack of tenderness over the greater trochanter suggests alternate diagnosis. (23) Pain along the posterior aspect of the greater trochanter may indicate gluteus medius involvement, while discomfort at the anterior aspect suggests a contribution from the gluteus minimus. (20) Hypertonicity and tenderness are common in the hip adductors, psoas, tensor fascia lata, gluteal, and lumbar muscles. Clinicians should pay particular attention to the gluteus maximus and TFL, as these muscles insert on the ITB and have the ability to enhance compression of the underlying gluteal tendons, particularly during hip adduction. (24, 25)

Patients may demonstrate limited hip range of motion with pain upon passive adduction or external rotation. (22) A significant limitation of passive internal rotation could suggest osteoarthritis. Resisted testing can help distinguish contractile tissue involvement from simple bursitis. (20) Resisted abduction is likely to reproduce GT complaints. (26) Modified Ober’s test is often positive.

The Hip Lag Sign demonstrates high sensitivity (89.5%) and specificity (96.6%) for detecting hip abductor tendon injury. (27) The Hip Lag Sign is performed with the patient in a side-lying position, affected side up. The clinician stabilizes the patient’s pelvis with one hand while using the other to passively move the patient’s hip and thigh into 20 degrees of abduction, 10 degrees of extension, and maximal internal rotation. The patient’s leg should remain relaxed with the knee bent at 45 degrees. After asking the patent to hold their leg in this position actively, the clinician releases support. The test is positive if the patient is unable to maintain this position and their foot drops more than 10 cm. (27)

The 30-second single leg stance test shows high sensitivity and specificity for gluteal tendinopathy. The test is performed by asking the patient to stand erect on the affected leg for 30 seconds. The clinician may stand in front of the patient to offer light stability. The patient should not be allowed to compensate by leaning toward the dependent side. Reproduction of pain during single leg stance suggests GT and is graded by the timing of onset- immediate (0-5 seconds), early (6-15 seconds), or late (16-30 seconds). (13, 28)

The resisted external de-rotation test also claims good sensitivity (88%) and specificity (97%). The test is performed by having the patient lie supine with their hip and knee flexed to 90 degrees. The clinician then passively rotates the patient’s thigh to full external rotation. If this movement is positive, the clinician slightly diminishes the degree of external rotation to achieve a comfortable position. Next, against clinician resistance, the patient is asked to de-rotate their leg inward, i.e., back to a neutral position. This movement tests the more vulnerable anterior gluteal tendon fibers that act as internal rotators. Reproduction of spontaneous pain suggests tendon involvement or tear. If the external de-rotation test is negative in the supine position, it may be repeated prone with the patient’s knee flexed to 90 degrees. The clinician then passively externally rotates the patient’s thigh by moving their ankle toward mid-line, and the patient is asked to de-rotate their hip outward against the clinician’s resistance. Performing the test in a prone position on a patient with a prior negative supine test increases sensitivity for gluteus tendinopathy. (28)

Patients with gluteal tendinopathy may demonstrate frontal plane movement imbalances. (6, 29) Hip abductor weakness is the most common muscle imbalance disorder in GT patients. Functional orthopedic testing for hip abductor weakness would include the Trendelenburg sign, overhead squat test, and single leg squat test. The presence of an “uncompensated” pelvic drop upon Trendelenburg maneuver suggests gluteus medius weakness. Long-standing weakness may result in a “compensated” response of lateral trunk flexion over the stance leg. Patients with significant tendon disruption may report pain on Trendelenburg testing. (30) Additional compensatory mechanisms for gluteus medius dysfunction include higher cadence, knee adduction, hyperpronation, and contralateral arm abduction. (31)

Imaging & differential diagnosis

Commonly encountered conditions that may produce lateral hip pain and mimic GT are many, and include osteoarthritis and other rheumatologic disorders, FAI, lumbar radiculopathy. In addition, mechanical low back pain, sacroiliac joint dysfunction, meralgia paresthetica, piriformis syndrome, iliopsoas tendinitis/tendinopathy, labral injury/tear and fibromyalgia. Other, more significant pathology in the adult patient includes avascular necrosis, stress fracture, acute traumatic fracture, primary bone tumor (benign and malignant), secondary bone tumor (metastasis), as well as soft tissue neoplasm (benign and malignant), and visceral somatic referral – particularly GI or GU.
In children, slipped capital femoral epiphysis (SCFE), Leg – Calve – Perthes (LCP) disease, as well as infection and primary neoplasms (benign and malignant) are other considerations.

When indicated, radiography includes minimum AP and frog-leg projections. A weight-bearing AP pelvis radiograph may be of benefit, particularly if significant biomechanical distortions are suspected. In children, bilateral AP pelvis and frog leg projections frequently are performed and may be helpful to identify subtle physeal changes or malalignment of the epiphysis. Generally, radiographs are not helpful for diagnosis of GT, but may reveal dystrophic tendon or bursal calcification, confirming clinical suspicion of this condition. Primarily, x-rays are performed to identify a secondary architectural cause for GT, including leg length discrepancy, acetabular hypoplasia, osteoarthritis, FAI and proximal femoral deformity from prior trauma, healed LCP or SCFE. In cases that are unresponsive to conservative measures, develop symptom progression or present with severe pain, inability to bear weight or limited passive mobility, x-rays are highly recommended.

Historically, MRI has been the standard imaging modality for confirming GT and associated inflammation involving the insertional tendon fibers (enthesitis), as well as other causes for lateral hip pain, including tendon tears, labral pathology and many other occult osseous and soft tissue abnormalities that may clinically mimic GT. MRI shows excellent specificity (92 – 100%). (27, 32, 33) However, diagnostic ultrasound shows a higher sensitivity (79-100%), with fewer false positives. (33) MRI is dependent on magnet strength and orientation of tendon fibers to the magnetic field, which may result in false positives. Ultrasound has the advantage of tailoring the examination by changing transducer placement and patient position during the examination but is highly dependent upon the technician performing the study. As always, advanced diagnostic testing should be reserved for cases of GT that are recalcitrant to conservative measures. Additionally, clinicians should not rely solely on advanced imaging findings, as many asymptomatic patients demonstrate ambiguous soft tissue changes that are often bilateral and do not correlate directly with presenting clinical symptoms. (33)

Management

GT treatment goals include:
1. Eliminating modifiable risk factors and faulty mechanics that cause excessive tensile or compressive loading.
2. Implementing early and progressive restorative loading to improve tendon integrity and load-bearing capacity. (13, 34)

ADL advice should include advising patients to avoid “hanging on one hip” and sitting or standing with their knees crossed. Patients should avoid prolonged periods of sitting, particularly on low-height seats. Keeping knees spread (like sitting on a horse) will ease tension when transitioning in and out of a chair. Patients will likely want to avoid side-lying sleep postures, as this triggers ipsilateral bed-side compression and contralateral traction-induced compression from thigh adduction. Placing a pillow between the knees can minimize excessive adduction traction. Sleeping in a ¾ prone position or using memory foam or egg-shell mattress toppers may reduce bed-side compression. When walking, patients should land softly on the front of their heels and take shorter quicker steps to avoid “over-striding”.

Athletes may need to temporarily avoid long distance or fast-paced running, hill climbing, and plyometrics (35) Lower stress alternatives would include cycling and water-based exercise. GT patients should avoid running on a banked surface, like the crown of a road or indoor track. Running on a small circular track causes the inner leg’s ITB to work harder to prevent it from swinging medially. Runners should reverse directions on a circular track each mile. Patients should avoid running on wet or icy surfaces, as these require greater TFL activation for stabilization. Patients with a “lazy” narrow-based running gait should be encouraged to increase step width to minimize stress on the iliotibial band. (36) Runners may need to consider new training shoes, particularly if the current shoes have over 300 miles or show any signs of wear on the lateral heel. Cyclists should make certain that their seat is not positioned “too high.” Overweight or obese patients should consider weight reduction programs.

Early rehab must minimize positions, activities, and exercises that involve sustained or repetitive compressive loading – particularly stretching the TFL/ITB in positions of hip adduction. (34, 37)

Sustained, low-intensity isometric contractions may provide analgesic benefit for tendinopathy patients. (38, 39) While there is no standard isometric exercise protocol for gluteal tendinopathy, a patellar tendinopathy protocol has demonstrated high success by performing four, 45-second contractions held at 70% maximum (MVC), repeated multiple times per day. (40, 41) Isometric contraction of the gluteal muscles can be achieved with the patient in a supine position with a pillow beneath their knees, and a belt strapped around their slightly separated knees for resistance.

Once the patient can tolerate isometric tensile loading, they may progress to eccentric exercises, with low repetitions of moderate effort. (34) Clinicians may use “change in nighttime pain” as a gauge for advancing or retreating exercise intensity. (34) High tensile load exercises should not be performed more than three times a week to allow adequate recovery time. (42)

Gluteus medius strengthening might include a combination of single leg stance, single leg squat, glut squeezes, bridging, side planks, lunges, band walk, side steps, step-ups, skater squats, and side plank with abduction exercises. Clam exercises are effective, however, patients may need to place a small pillow between their knees to avoid excessive adduction. Alternately, the exercise can be performed seated. While isolated hip stretching and strengthening exercises may be necessary to improve mobility and strength, they do not necessarily translate to improved functional movement patterns. For lasting improvement, patients must be subsequently taught to ‘groove” new movement patterns, via progressively challenging activity-specific exercises. (13, 43, 44)

Weight-bearing exercises, particularly single leg stance activities, trigger higher levels of gluteus medius activation (i.e., compression) and must be implemented strategically. (45) The functional rehab continuum begins by controlling frontal plane alignment during simple tasks like transitioning from sitting to standing, advances to functional bridging, stepping, and squatting, then culminates with more complex activities like running, cutting, and jumping. (13) Clinicians should teach patients to control excessive hip adduction while performing progressively higher load tasks. Instruction should include proper squatting and hip hinge techniques to limit hip internal rotation.

Clinicians must seek to identify and correct functional deficits throughout the lower body. (18, 46, 47) Arch supports or custom foot orthotics may be useful to correct excessive foot pronation. Manipulation and mobilization may be necessary to resolve restrictions in the lumbosacral spine and lower mechanical chain.

As with other degenerative tendinopathies, clinicians should employ treatments that initiate a controlled inflammatory response and trigger healing. IASTM may stimulate remodeling of the gluteus medius and gluteus minimus tendons and has shown benefit in managing chronic tendinopathies. (48) Therapeutic taping has been shown to stimulate gluteus medius function. (49) Various authors have recommended ancillary modalities, including massage, dry needling, and extracorporeal shockwave therapy (ESWT), AKA acoustic pulse wave therapy. (13, 50) Soft tissue manipulation and myofascial release may be necessary to resolve issues in the gluteal muscles as well as secondary sites of compensatory dysfunction.

Medical alternatives span the gamut from oral pain medications to surgery. Corticosteroid injections may provide moderate initial relief, but those results are not always sustained. (37) Research has shown that omitting cortisone when performing a tendinopathy injection leads to improved clinical outcomes. (51) Exercise and education have been shown to outperform cortisone injections. (52)Ultrasound-guided percutaneous needle tenotomy may be a useful tool for gluteal tendinopathy. (53, 54) Similarly, PRP injections may provide benefit for tendinopathy patients. (55, 56) Surgery is reserved for ruptures or tears that fail conservative management. (13) Surgical management includes minimally invasive endoscopic debridement, micropuncture, or excision with reports of good outcomes for appropriate cases. (57)

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