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Pains in a Butt – A Physiatric Approach to Buttock Pain

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Case Vignette

A 49-year-old female presents to the UPMC PM&R clinic for right buttock pain. The pain started 8 weeks ago and has been worsening over time. A few months prior to the onset of symptoms, she started playing golf. She was playing about once a week and was walking the course. She also jogs for exercise.

Her pain is localized in the right buttock region with no radiation. The pain is worse with sitting, prolonged walking, golfing, jogging, and turning in bed. Standing is not painful. The pain has become quite severe, causing her to stop golfing and jogging. She denies any subjective motor or sensory abnormalities.

She has tried over the counter NSAIDs and acetaminophen with limited relief. She has been in physical therapy for about 4 weeks performing basic core stability exercises (such as bird dogs and planks) as well as some gluteal strengthening exercises (clam shells and side-lying hip abductions). Because she has not seen improvement, she is asking for help to treat this problem.

Description of Problem

Determining the cause of pain localized in the posterior buttock region can be challenging. Pathology from many different structures – including the lumbar spine, pelvis, hip, and proximal thigh – may be implicated and can present similarly. The aim of this paper is to review three potential causes of buttock pain: atypical lumbosacral radiculopathy, gluteus medius tendinopathy, and proximal hamstring tendinopathy.

Buttock Region Anatomy

Osseous Anatomy

The bony structures that form the foundation of the gluteal region are the pelvis, acetabulum, and femur. The pelvis is composed of two iliac bones that join anteriorly at the pubic symphysis and posteriorly at the sacrum. Important pelvic and hip anatomy that may relate to buttock pain pathology includes the ischial tuberosity, greater trochanter, lesser trochanter, and sacroiliac joint.

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Disclosures

Drs. Bitterman, Brown, and Eventov report no relationships with proprietary entities producing health care goods and services.

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Ligamentous Anatomy

The hip joint capsule is a composite of three longitudinal ligaments that extend from the acetabulum to the femur. These include:

- Iliofemoral ligament: travels from the anterior inferior iliac spine and then splits, connecting to the intertrochanteric line of the femur anteriorly and inferiorly to resist anterior hip subluxation.
- Ischiofemoral ligament: connects the acetabulum's posterior ischial rim to the posterior femoral neck to resist hip internal rotation and adduction.
- Pubofemoral ligament: connects the pubic portion of the acetabulum to the femoral neck in order to resist hip external rotation.

In addition to the capsular ligaments, there is also the zona orbicularis, which encircles the femoral neck to prevent femoral head distraction and contains the ligamentum teres, which attaches the femoral head to the acetabular notch, mainly acting as a conduit for the obturator artery to the femoral head.

Hamstring Musculature

Hamstring muscles originate on the ischial tuberosity except the short head of the biceps femoris, which originates distally on the linea aspera. The semitendinosus and long head of the biceps tendon form a conjoined tendon that originates on the medial surface of the ischial tuberosity. The semimembranosus originates separately on the superolateral surface of the ischial tuberosity. Each muscle has a separate distal attachment: the semitendinosus to the medial tibia (as part of the pes anserine), the semimembranosus at the medial tibial condyle, and the biceps femoris at the fibular head. The hamstrings cross two joints and act as hip extensors and knee flexors. The hamstring muscles that originate at the ischial tuberosity are innervated by the tibial nerve (L5, S1, S2) and the short head of biceps femoris is innervated by the common fibular nerve (L5, S1). See Table 1 for a summary of gluteal and lateral hip musculature.

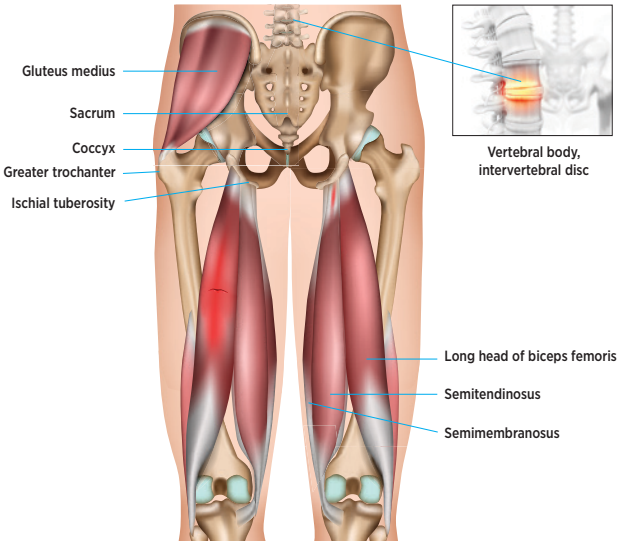

Figure 1: Lumbar Hernia

Neuroanatomy

Ventral rami of the lumbar and sacral spinal nerves form the lumbosacral plexus in the pelvis. These fibers compose the somatic innervation to the pelvis and legs. The lumbar plexus divides into several peripheral nerves. Of clinical relevance to buttock pain is the sciatic nerve (L4, L5, S1, S2, S3). The sciatic nerve travels adjacent to the piriformis while exiting the pelvis. In most humans, the sciatic nerve courses deep to the piriformis. However, there are anatomic variations where the nerve courses through the piriformis.

Buttock Pain Differential Diagnosis

Table 2 details the differential diagnosis for a patient with buttock pain. Three common causes of buttock pain — lumbosacral radiculopathy, gluteus medius tendinopathy, and proximal hamstring tendinopathy — are discussed in more detail in Table 2 on page 3.

Table 1: Clinically Relevant Gluteal and Lateral Hip Musculature

Muscle	Origin	Insertions	Action	Innervation
Gluteus Maximus	Ilium, coccyx, and sacrotuberous ligament	Gluteal tuberosity of the femur, iliotibial band	Hip extension	Inferior gluteal nerve (L5, S1, S2)
Gluteus Medius	Lateral superior ilium	Lateral and posterosuperior facets of the greater trochanter	Hip abduction, hip internal rotation, hip external rotation, stabilization of pelvis in single-leg stance	Superior gluteal nerve (L4, L5, S1)
Gluteus Minimus	Lateral ilium, sciatic notch	Anterior facet of the greater trochanter	Hip flexion, hip abduction, hip internal rotation	Superior gluteal nerve (L4, L5, S1)
Tensor Fascia Lata	Lateral surface of the anterior superior iliac spine	Iliotibial band	Stabilization of the hip in extension. Also weakly performs hip flexion, hip abduction, hip internal rotation	Superior gluteal nerve (L4, L5, S1)
Piriformis	Anterior sacrum	Greater trochanter	Hip external rotation	Nerve to piriformis (L5, S1, S2).
Quadratus Femoris	Lateral ischial tuberosity	Intertrochanteric crest at the medial femur	Hip external rotation, hip adduction	L4, L5, S1

Table 2: Differential Diagnosis for Buttock Pain

Disease	Clinical Presentation	Diagnosis	Pearls
Ankylosing spondylitis	- Chronic back, buttock, and/or groin pain. Often described as stiffness - 80% of patients record their first symptoms before the 4th decade - Male > female - May also present with peripheral arthritis, enthesopathy, and/or dactylitis	- 50 to 70% of patients with active disease have elevated inflammatory markers - A classic imaging finding in the late stage is "Bamboo Spine", referring to the fusion of adjacent vertebral bodies	- 6% of patients who are HLA-B27 positive have ankylosing spondylitis - Extra-skeletal associated diagnoses by prevalence: inflammatory bowel disease (50%), anterior uveitis (30%), psoriasis (10%)
Avascular necrosis of the hip	- Groin or buttock pain - Pain associated with walking, transitioning from sitting to standing, stairs, and inclines - Risk factors: AIDS, alcohol abuse, corticosteroids, infection, pancreatitis, sickle cell anemia, systemic lupus erythematosus, trauma	- Hip radiographs recommended for initial testing, include frog-leg lateral view to assess for subchondral collapse - Hip MRI recommended if high suspicion, sensitivity and specificity of 98%	- Risk of femoral head collapse can be classified into three groups using the modified Kerboul Necrotic Angles on MRI 1. Low risk: combined necrotic angle <190 2. Moderate risk group: combined necrotic angle is between 190 and 240 3. High risk group: combined necrotic angle is >240
Gluteus medius tendinopathy	- Posterolateral buttock pain, may radiate down lateral thigh - Pain worse with laying on the painful side and walking on uneven surfaces	- Clinical diagnosis, testing often not needed - Hip MRI can be ordered to confirm diagnosis if patient is not improving with conservative management	- Can occur secondary to other pathologies that affect hip biomechanics, in particular lumbosacral radiculopathy and hip osteoarthritis
Hip osteoarthritis	- Classically presents as groin pain, but may present as buttock pain - Pain worse with weight bearing activities and hip motion. - On exam, patient will have reduced hip range of motion with pain. They may have an antalgic gait and may shift their trunk over the affected hip during gait	- Osteoarthritis of the hip is often diagnosed clinically - Hip radiographs recommended to assess for extent of osteoarthritis - MRI and/or CT are typically not required for diagnosis, though may be used if there is high suspicion for intra-articular pathology and radiographs are unremarkable	Tonnis radiograph grading scale for hip OA: **Grade 0:** No signs of OA **Grade 1:** Slight narrowing of joint space, slight lipping of joint margin, slight sclerosis of femoral head or acetabulum **Grade 2:** Small cysts in femoral head or acetabulum, increasing narrowing of joint space, moderate loss of sphericity of femoral head **Grade 3:** Large cysts, severe joint space narrowing, severe femoral head deformity, avascular necrosis
Ischiofemoral impingement	- Due to narrowing of the space between the ischium and lesser trochanter. This results in impingement of the quadratus femoris muscle - Buttock pain that is worse with load dependent activities - Snapping, clunking, or locking sensation at hip joint while walking with a long stride - Pain may radiate down the thigh	- Plain radiographs of the pelvis should be obtained to evaluate hip configuration and generalized sense of the ischiofemoral distance - MRI is gold standard for evaluation of the ischiofemoral space. Edema or tears of the quadratus femoris strongly suggests this diagnosis.	- There are two validated exam tests used for ischiofemoral impingement: 1. Long Stride Walking Test: pain evoked by taking long strides (sensitivity 92%, specificity 82%). 2. IFI test: pain with passive hip extension and adduction (sensitivity 82%, specificity 85%)
Lumbar facet arthropathy	- Usually presents as low back pain, but may present as referred pain to the ipsilateral buttock and proximal thigh	- Oblique extension exam test has poor diagnostic accuracy - Gold standard is anesthetic block of facet joint. This should result in >80% pain relief. Two blocks recommended as single blocks have a false positive rate of 25-40%.	- The presence of facet arthropathy on imaging does not correlate with response to treatment. Facet arthropathy is common in the asymptomatic population. However, increased T2 signal on MRI or increased uptake on bone scan at a facet joint that correlates with the patient's pain may support this diagnosis.
Lumbar spondylolysis	- Usually presents with low back pain, but may present with buttock and posterior thigh pain -Typically seen among adolescent athletes	- Stork Test: Single leg hyperextension and rotation of the spine reproduces pain - MRI is the test of choice rather than radiographs or CT scan, to reduce radiation exposure to the adolescent population	- Bilateral spondylolysis at a single level can result in spondylolisthesis
Lumbosacral radiculopathy	- Typically presents as low back pain that radiates down the leg, but may atypically present as buttock pain - Associated neurologic abnormalities	- Clinical diagnosis, lumbar MRI recommended if persistent pain despite conservative management or the patient has significant neurologic abnormalities - Electrodiagnostic testing is specific but not very sensitive	- Red flag signs/symptoms: significant or progressive weakness, bowel or bladder dysfunction, saddle anesthesia, constitutional symptoms
Piriformis syndrome	- Proposed pathophysiology is sciatic nerve entrapment by the piriformis - Described as buttock pain that may radiate down the posterior thigh, associated with prolonged sitting and hip movements - May be seen in patients after trauma to the buttock region, resulting in injury to the piriformis	- Diagnosis of exclusion - Prior reported provocative maneuvers are: 1. Freiberg: Hip extension and forceful internal rotation 2. Pace: Resisted abduction and external rotation of the thigh 3. Beatty: Deep buttock pain produced by the side-lying patient holding flexed knee several inches off the table 4. FADIR: flexion, adduction, and internal rotation - EMG may demonstrate an abnormal H-reflex	- The prevalence of this syndrome is disputed
Proximal hamstring tendinopathy	- Pain in the inferior gluteal region, may radiate down posterior thigh - Exacerbated by exercise, prolonged sitting, squats, lunges, and hamstring stretches - Seen in runners, hurdlers, soccer, football and other sports with rapid acceleration or rapid changes in direction	- Radiographs may show ischial tuberosity avulsion. Recommended in younger athletes where the apophysis may not be fused - MRI may be considered to confirm diagnosis	- Spectrum of injuries, from benign strain to chronic degenerative disorder to tendon rupture
Sacral stress fracture	- Intractable low back, buttock, groin, and/or thigh pain - History of trauma, especially ground level falls in the elderly population	- Pelvis MRI recommended, highest sensitivity test	- One study found 78% of patients with sacral stress fractures had a coexisting pubic rami fracture
Sacroiliitis	- Buttock and/or low back pain, may radiate to lower limb - As with ankylosing spondylitis, may be described as a stiffness	- Provocative tests such as FABER and Gaenslen's have low specificity - Radiographs show significant sclerotic changes along the SI joint, particularly at the inferior iliac margin - Pelvis MRI shows edema at the SI joint during inflammatory period - Recommended labs: WBC, ESR, CRP, HLA-B27, rheumatoid factor, ANA	- Associated with ankylosing spondylitis and other HLA-B27 disorders - Sacroiliac dysfunction is a separate entity most commonly seen in patients with joint laxity (pregnancy, connective tissue disorder)
Vascular claudication/ Peripheral artery disease	- Pain exacerbated activity and relieved by rest - Depending on the location of the obstruction, pain may manifest in different portions of the lower extremity. Lesions at the aortoiliac bifurcation may produce buttock pain.	- Lower limb dopplers with ankle-brachial index is recommended initial screening test	

Lumbosacral Radiculopathy

Epidemiology

The prevalence of lumbosacral radiculopathy (LSR) is about 3-5%. The two most common causes of LSR are lumbar disc herniations and lumbar spondylosis (e.g., facet hypertrophy, ligamentum flavum thickening) resulting in spinal stenosis. Lumbar disc herniations most commonly occur in patients under the age of 50, while LSR due to lumbar spondylosis is more typical in older patients.

Certain athletic populations are more prone to the development of lumbar disc herniations. Patients who play sports with extreme torsional movements (e.g., golf and baseball) and activities that place high stress on the lower lumbar spine (e.g., rowing, deadlifting) may be more prone to LSR.

Pathophysiology

LSR can be defined as "objective loss of sensory and motor function with or without accompanied spinal and/or referred leg pain following a mechanical or bio-chemical dysfunction of lumbar and sacral nerve roots and their associated dorsal root ganglions."[1] Potential causes of lumbosacral radiculopathy include lumbar intervertebral disc herniations, lumbar spondylosis resulting in spinal stenosis, lumbar spondylolisthesis causing narrowing the neural foramen, or mass occupying lesions (e.g. neoplasm, epidural abscess, or epidural hematoma).

Disc herniations involving herniation of disc inner nucleus pulposus beyond the bounds of the outer annulus fibrosus. Activities that increase pressure on the disc (such as lumbar flexion, lumbar torsion, or lifting) may cause the nucleus pulposus to herniate through tears of the annulus fibrosus and into the spinal canal or neural foramen. These herniations most commonly occur in the posterolateral portion of the disc due to the tapered anatomy of the posterior longitudinal ligament. The herniated disc may cause mechanical compression and/or chemical irritation of an adjacent nerve root.

Lumbar spondylosis describes degenerative changes in the spine including facet arthropathy, disc bulges, and ligamentum flavum thickening. These changes can cause narrowing of the spinal canal and neural foramen.

Clinical Findings

LSR classically presents as low back pain radiating down the lower limb in a dermatomal pattern. However, a lower lumbar or S1 radiculopathy may atypically present as pain radiating to the buttock.

Examination findings that may suggest a patient's buttock pain is from an LSR includes pain worse with lumbar motion, pain with valsalva maneuvers (in the setting of a disc herniation), pain worse with walking that relieves with rest (in the setting of lumbar spinal stenosis with neurogenic claudication), and the description of burning or electric pain.

On examination, pain may be provoked with lumbar range of motion and with dural tension tests (such as the slump test and straight leg raise). In the straight leg test, the patient is in supine and the hip is passively flexed while maintaining knee extension (positive predictive value of 0.16-0.31).[2] In the slump test, the patient is seated with full neck flexion while the knee is passively extended (positive predictive value of 0.18-0.31).[2] In addition, a neurologic exam may find depressed or absent reflexes, sensory changes in a dermatomal pattern, and/or weakness in a myotomal pattern.

L5 radiculopathy is the most common LSR to present as isolated buttock pain. Hip abductor strength should be tested, as this may be asymmetric.

Diagnostic Testing

LSR is usually diagnosed clinically and can be initially managed conservatively without further testing. Indications for imaging include neurologic abnormalities, red flag signs/symptoms (saddle anesthesia, bowel/bladder changes, concern for infectious or neoplastic etiology, progressive neurologic deficits), or persistent pain that does not respond to conservative management.

Lumbar spine radiographs may show disc space narrowing (suggestive of a disc herniation), spondylolisthesis, and/or spondylosis. However, radiographs alone may not be sufficient to confirm the diagnosis of lumbar radiculopathy and are not recommended when disc herniation is suspected.[3]

Lumbar spine magnetic resonance imaging (MRI) is the highest yield diagnostic test for patients with a suspected LSR. MRI best visualizes the lumbar intervertebral discs, spinal canal, and neural foramina, allowing assessment of stenosis that may be causing radiculopathy.[4] Clinicians should be aware, however, that degenerative changes in the lumbar spine are common and may be incidental.[5]

In cases where MRI is contraindicated, computed tomography (CT) may be used, although this imaging modality is not as sensitive for assessment of lumbar stenosis or disc pathology. CT myelogram is superior to traditional CT in the assessment of lumbar stenosis in patients in whom MRI is contraindicated.[4]

Electrodiagnostic testing (EDX) can be useful in suspected LSR when more data is needed to support the diagnosis. It may also be helpful to differentiate LSR from other peripheral nerve pathologies. Six-muscle (including paraspinal) needle electromyography (EMG) testing is highly specific (87-100%) though not as sensitive (49-86%) for LSR.[6] EDX is a reasonable adjunct to office evaluation and imaging when confirmation of a suspected LSR is warranted.

Treatment

The mainstay of treatment in LSR is conservative. In evaluating patients with a diagnosis of LSR, 67-90% see significant recovery with conservative therapy only.[7] Pharmacologic approaches include NSAIDS and acetaminophen. Systemic steroids, specifically a prednisone taper, have resulted in improvements in functional outcomes but not pain.[8] Physical therapy has some of the stronger evidence for functional improvement is LSR.[3] Some physical therapists use the McKenzie or Mechanical Diagnosis and Therapy (MDT) programs for these patients. MDT involves determining the directional movements which worsen and relieve the patient's pain. Exercises done in the relieving direction should hopefully facilitate centralization of the radicular pain. For example, patients with LSR due to a lumbar disc herniation frequently have pain with lumbar flexion movements. In these cases, a lumbar extension-biased therapy program may be more beneficial.

Epidural steroid injections (ESI) are a common non-surgical intervention for LSR.[9] The aim of this procedure is to reduce inflammation at the implicated nerve root. Studies have found this procedure can reduce pain and disability, though the effect may be slight and temporary.[9]

Surgical consideration, such as discectomy, should be reserved for patients with treatment refractory disease.[3] In these cases, shared decision-making including weighing the risks and benefits with a surgical provider is recommended. In more emergent situations, such as patients presenting with "red flag symptoms", surgical evaluation is warranted. In cases where a disc herniation is confirmed as the cause of the LSR, there is evidence to suggest improvement in outcomes for patients who underwent surgery vs those treated non-operatively.[10]

Gluteus Medius Tendinopathy

Epidemiology

Gluteus medius tendinopathy typically presents in the middle-aged population. It has a higher prevalence in women compared to men. Other risk factors include increased body mass index and knee osteoarthritis.[11,12]

Certain athletic endeavors may place individuals at higher risk for gluteus medius tendinopathy, in particular sports with repetitive hip adduction such as dancing and running with a crossover-style gait.

Pathophysiology

Gluteus medius tendinopathy falls under the umbrella of "greater trochanteric pain syndrome". This syndrome has been attributed to greater trochanteric bursitis. However, radiologic studies have found that only a minority of these patients have a true bursitis.[13] Far more common is tendinopathic changes of the gluteal musculature, in particular the tendinous attachment to the gluteus medius.[13]

Like many other tendon disorders, gluteus medius tendinopathy is a degenerative cascade in which the tendon poorly adapts to loading. This results in abnormal tenocyte behavior, matrix disorganization, hypercellularity, and neovascularization that results in a positive feedback cycle, where these cellular changes lead to further tendon injury.[14]

Gluteus medius tendinopathy often occurs secondary to other pathologies that alter the mechanics of the hip, in particular hip osteoarthritis and lower lumbar radiculopathy. However, tendinopathy may also be a primary source of pain when anatomic variations or a patient's activities alter tension and compression forces on the tendon, such as patients with coxa vara (reduced femoral-neck shaft angle), patients who are in prolonged periods of hip adduction, patients who do repetitive hip adduction activities, and patients with weak hip abductors. Patients can overuse their tensor fascia lata (TFL) as hip abductors, resulting in preferential anterior deviation of the hip during hip abduction. As the TFL is confluent with the iliotibial (IT) band, this may explain why patients with gluteus medius tendinopathy may also have lateral thigh pain. Gluteus medius tendinopathy may also occur due to trauma to the lateral hip.

Clinical Findings

Gluteus medius tendinopathy classically presents with posterolateral buttock pain that may radiate down the lateral thigh. The pain is aggravated by activities that require gluteus medius activation. Pain with walking on uneven surfaces (such as on a beach) is relatively specific, as this action requires frequent leveling of the pelvis by the gluteus medius. Other exacerbating activities include stairs, running, and laying on the painful side.

Listed below are exam maneuvers that may assist with diagnosing gluteus medius tendinopathy:

1. Palpation of the greater trochanter in the side-lying position but this is non-specific (sensitivity = 80%, specificity = 47%).[15] The examiner should not palpate aggressively as most people will have pain at the bony prominence with strong enough palpation.
2. Resisted hip abduction in the side-lying position. The examiner should ensure the patient keeps the hip in mild extension (sensitivity = 59%, specificity = 93%).[16]
3. FABER – With the patient supine, the leg is placed in hip flexion, abduction, and external rotation with the ankle placed on the contralateral patella (sensitivity = 42%, specificity = 80%).[15]
4. Resisted hip external rotation – With the patient supine, the hip is flexed to 90 degrees. The examiner then resists the patient from actively externally rotating the hip. Reproduction of pain was found to be both sensitive and specific for gluteus medius tendinopathy (sensitivity = 88%, specificity = 97.3%).[17]
5. Single leg stance held for 30 seconds. Visible dropping of the pelvis on the affected side was found to be specific but not sensitive for gluteus medius tendinopathy.[5] However, reproduction of pain during this test has been found to be sensitive and specific for gluteus medius tendinopathy (sensitivity = 100%, specificity = 97.3%).[17]

Diagnostic Testing

Most patients with gluteus medius tendinopathy can be diagnosed clinically without any further testing. However, in scenarios where testing is needed to confirm the diagnosis, MRI is the test of choice, though gluteus medius tendinopathy can be incidentally present in the asymptomatic population.[18]

Ultrasonography may also be helpful in the diagnosis of gluteus medius tendinopathy. On ultrasound, the tendon may be thickened, hypoechoic, or have heterogeneous echotexture. There may be fluid present in the trochanteric bursa.[19]

Treatment

The mainstay of treatment for gluteus medius tendinopathy is conservative. Early activity modification is recommended such as avoiding laying on the affected side, avoiding sitting with legs crossed and refraining from sports or activities that aggravate the pain.[20] Physical therapy programs should focus on stretching the iliotibial band, tensor fascia lata and other tight structures; strengthening hip abductors (mainly the gluteus medius), external rotators and extensors; strengthening core musculature; and improving hip range of motion.[20] Gait training should be incorporated, as gait abnormalities are common with this pathology.[20] Non-steroidal anti-inflammatory drugs can also be considered in the acute phase however systemic effects and contraindications must be considered.

Interventional approaches may be necessary as well. Corticosteroid injections have been shown to provide only short-term relief. One study found that corticosteroid injections have a 75% success rate at one month but this fell to 48% at 15 months, compared to an 80% success rate with a home exercise program at the same 15-month follow-up.[21] A separate study found that performing no intervention at all provided better intermediate and long-term benefit than a corticosteroid injection.[22] Low energy extracorporeal shockwave therapy may be a useful intervention for treatment of gluteus medius tendinopathy. In a randomized control trial, shock wave treatment

was found to have a success rate of 68% at 4 months and 74% at 15 months.[21] However, this study did not show significant benefit early in the disease course (1-3 months).[21] Biologics are also an emerging field in treating tendinopathies. Further research may be necessary to better determine which patients may be most appropriate for these treatments.

In cases refractory to the above measures, surgery may be considered. A case series showed resolution of pain in 95% of patients at 6 and 12 months after operative gluteal tendon repair.[23] Unfortunately, there is no literature comparing operative and non-operative interventions for gluteus medius tendinopathy.

Proximal Hamstring Tendinopathy

Epidemiology

Proximal hamstring tendinopathy is one of the more common athletic tendon injuries. [24] It is most frequently seen in sagittal-plane dominant sports (e.g., distance running, sprinting, and hurdling) and sports with rapid changes in direction (e.g., soccer, football, and hockey).[25]

Proximal hamstring tendinopathy can also present in non-athletes. These are commonly middle-aged patients performing repetitive squatting or sitting for prolonged periods.[24] In this population, the bilateral hamstrings may be implicated. Risk factors for proximal hamstring tendinopathy include increased exercise volume, hamstring/quadriceps imbalance, increased BMI, excessive hamstring static stretching, older age, perimenopause, metabolic abnormalities (e.g., diabetes mellitus and hyperlipidemia), and fluoroquinolone usage.[26]

Pathophysiology

Proximal hamstring tendinopathy encompasses a range of injuries, from self-limiting strains to chronic degenerative injuries to tendon rupture. Among runners, hamstring injuries typically occur during eccentric contraction in terminal swing as the muscle decelerates the leg.[27] This force may be accentuated with excessive forward trunk lean while running, longer stride length, and running uphill. The proximal hamstring may also be injured from compressive forces. Since the tendon wraps around the ischial tuberosity, it is susceptible to compressive loads.[25] This load is particularly an issue in the end range of hip flexion and truncal flexion.

Clinical Findings

Patients with proximal hamstring tendinopathy present with pain in the inferior buttock region that may radiate down the posterior thigh. In situations where the tendinopathy is not an acute strain or tendon rupture, this pain is gradual and insidious. Early in the course of this disorder, athletes may have pain when they begin exercising, and then the pain then resolves after the warm-up. As the condition progresses, pain may persist beyond warming-up. Proximal hamstring tendinopathy pain may also be exacerbated by activities that place tension on the hamstrings (such as squats, lunges, and hamstring stretching) and activities that put compressive forces on the tendon origin (such as prolonged sitting).[26]

As both lower LSR and proximal hamstring tendinopathies can present with buttock pain that radiates down the posterior thigh, it can be difficult to differentiate the two on history alone. Tendinopathy should not cause any neurologic abnormalities. The presence of neurologic changes may point to radiculopathy as the primary diagnosis.

Acute exam findings include ecchymosis at the lower buttock or posterior thigh. There is often tenderness at the ischial tuberosity. There are two passive stretch tests that may provoke pain in patients with proximal hamstring tendinopathy:

1. **Puranen-Orava Test** – While standing, the patient rests their heel on a table or raised bed while keeping the hip flexed to 90 degrees and the knee fully extended. The patient then leans forward to stretch the hamstring (sensitivity 0.76, specificity 0.82).[28]
2. **Modified Bent Knee Test** – With the patient supine, the examiner passively flexes the hip and knee to end range of motion then rapidly fully extends the knee (sensitivity 0.89, specificity 0.91).[28]

Examiners can test if actions that place the hamstring under load provoke their pain. One option for this is a single leg, bent knee bridge. The patient lays supine, lifts one leg in the air, then bridges by actively extending the other hip while keeping the knee bent and the foot on the surface.

Diagnostic Testing

When the diagnosis of proximal hamstring tendinopathy is not obvious based on clinical findings or if there is poor response to conservative management, imaging may be warranted.

Radiographs may be considered if there is concern for an ischial tuberosity avulsion, particularly for younger athletes. The ischial apophysis may not finish fusing until age 25.[28] MRI has the highest sensitivity in assessing for hamstring tendinopathy. MRI can show increased signal in the tendon on T1 and T2 sequences, tendon thickening, peritendinous edema, ischial bone marrow edema, and ischial tuberosity avulsions.[28] Ultrasound may be helpful for point of care assessment and sonopalpation. On ultrasound, the tendon can be thickened, hypoechoic, or have heterogeneous echotexture. There may also be calcifications within the tendon or cortical irregularity at the ischial tuberosity origin.[29]

Treatment

Most patients with proximal hamstring tendinopathy can be treated conservatively with physical therapy (PT) and oral analgesics. In the acute period after a hamstring injury, patients should follow the RICE protocol (rest, ice, compression, elevation). NSAIDs can be used to manage pain and reduce acute inflammation.

The first phase of PT involves low intensity range of motion exercises and isometric hamstring loading exercise (such as isometric leg curls and bridges). Patients transition to the second phase of PT once they can tolerate hamstring loading during hip flexion with mild pain. The second phase puts the patient through eccentric and concentric hamstring strengthening exercises to improve collagen turnover and re-organization. The focus of this phase is performing resistance exercises at a slow, controlled pace to fatigue. Exercises may include single-leg bridges, prone leg curls, and prone hip extensions. Phase three further strengthens the hip at higher ranges of hip flexion, such as Romanian deadlifts, walking lunges, and hip thrusts. The final phase of PT involves sports specific exercises, particularly for athletes who need to perform high levels of energy loading, such as running and multidirectional movement.[25]

Corticosteroid injections may improve symptoms in the short term. A 2010 retrospective study found 50% of patients who received an ultrasound-guided peritendinous corticosteroid injection got >1 month relief, though only 23.7% got >6 months relief.[30] There have been mixed results for the efficacy of platelet rich plasma injections for proximal hamstring tendinopathy. Further research may be needed to determine which types of tendinopathies may respond better to this treatment.[28]

Surgery is indicated for complete proximal avulsions, partial avulsions with at least 2 tendons injured, >2cm of tendon retraction in active, younger patients, and partial avulsion injuries involving one tendon that have failed conservative treatment.[24,28]

Case Vignette Outcome

Our patient was found to have 4+/5 right extensor hallucis longus weakness, 4/5 right hip abductor weakness, and reduced sensation at the right dorsal first webspace. In addition, her right buttock pain was reproduced with lumbar flexion and dural tension testing. The hip exam was unremarkable. A lumbar spine radiograph showed L4-5 disc space narrowing. These findings raised concern for an L4-5 disc herniation causing a right lower lumbar radiculopathy.

Lumbar MRI without contrast was ordered because her pain was present for 8 weeks without relief despite physical therapy and oral medications. She was started on gabapentin 300mg TID and celecoxib 100mg q12h PRN. The MRI showed a right paracentral L4-5 disc protrusion with right subarticular stenosis compressing the right L5 nerve root. The patient had persistent pain despite the change in medications. A right L5 transforaminal epidural steroid injection was then performed, which provided significant relief. She resumed physical therapy that started with basic core stability exercises and eventually progressed to sports-specific exercises. She was back to playing golf by the end of her PT regimen.

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