

Deep Gluteal Pain in Orthopaedics: A Challenging Diagnosis

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ABSTRACT

Identifying the specific source of gluteal pain can elude the most seasoned orthopaedic diagnosticians. Patients will often present with a protracted course of symptoms, and failure to successfully identify and treat the underlying etiology leads to frustration for both patient and clinician. Pain deep in the buttocks can arise from compression, inflammation, or injury of one or more of the structures in this anatomically dense area. Although sacroiliitis, hip arthritis, and trochanteric bursitis may also masquerade as gluteal pain, sciatic nerve irritation in its various presentations causes a substantial percentage of cases. Deep gluteal syndrome, hamstring syndrome, and ischiofemoral impingement can have overlapping presentations but can be differentiated by clinical examination and judiciously placed diagnostic corticosteroid injections. Although nonsurgical management, including physical therapy, relative rest, and injections represent the mainstay of treatment, open and endoscopic surgical approaches have yielded encouraging success rates in refractory cases.

Gluteal pain presents both a diagnostic and therapeutic challenge for orthopaedic surgeons because of the ambiguous literature on the subject and complex regional anatomy. Furthermore, gluteal pain nomenclature and disease descriptors are often vague. The term piriformis syndrome was used in the past to describe nondiscogenic sciatica-like pain due to entrapment of the sciatic nerve (SN) by the pear-shaped piriformis muscle.¹ Since then, fibrous bands and other muscles adjacent to the SN, including the proximal hamstring, coccygeus, levator ani, obturator internus, and gemelli muscles, have been reported as potential sources of SN entrapment.² Contact exists between the SN and its adjacent musculature in every human being; however, mere anatomic proximity cannot satisfactorily explain symptomatology. Furthermore, in pure gluteal pain presentations without a traumatic origin or motor/sensory radicular deficits, no study has shown definitive electromyographic corroboration of SN dysfunction. McCrory and Bell suggested that structures other than the SN might give rise to pain in this area and proposed the more encompassing term deep gluteal syndrome (DGS) to

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describe deep buttock pain.² Recently, Kizaki et al³ defined DGS more generally as a nondiscogenic SN disorder with entrapment in the deep gluteal space. Hamstring syndrome (HS), related to chronic or repetitive tears of the proximal hamstring and ischiofemoral impingement (IFI) resulting from conflict between the ischium and the lesser trochanter (LT) of the femur, produces similar deep buttock pain with or without radiation leading to an overlap of symptoms and diagnostic ambiguity. This article reviews causes of deep gluteal pain with emphasis on anatomic relationships and diagnostic pearls to facilitate evaluation and management of this challenging entity.

Anatomy

The deep gluteal space is anatomically demarcated by (1) anteriorly, the posterior acetabular column and hip joint capsule; (2) posteriorly, the gluteus maximus muscle; (3) laterally, the gluteal tuberosity, (4) medially, the sacrotuberous ligament; (5) superiorly, the inferior margin of the sciatic notch; and (6) inferiorly, the proximal origin of the hamstrings at ischial tuberosity.¹ (Figure 1).

The Sciatic Nerve

The SN may be compressed at several points as it passes through the deep gluteal space. The SN coalesces from the L4-S3 ventral nerve roots into a single trunk with both tibial and common peroneal fascicular groups surrounded by a common epineurial sheath. Diving through the greater sciatic foramen, where the bony contour may

act as a potential tether, it emerges from deep to superficial at the inferior border of the piriformis muscle belly, where entrapment may also occur.⁴ It then crosses the obturator-gemelli complex, another potential site of impingement, and continues about 1 cm lateral and deep to the proximal hamstring tendon origin, where adjoining fibrous bands can develop. At this level, it also lies posterior to the quadratus femoris muscle, which separates it from the LT.⁵ Chronic stress on the nerve at any of these levels may result in dysfunction. Rat models, for example, have indicated that even at only 12% strain, SN nerve conduction is blocked and may not recover when this is maintained past 1 hour.⁶

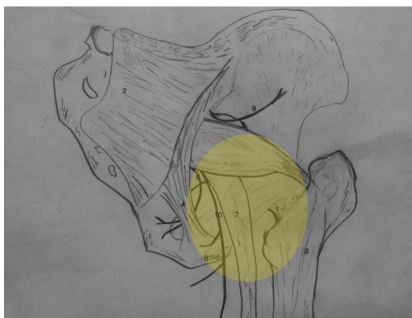
Some anatomic variation has been identified as the nerve enters the deep gluteal space. A recent systematic review reporting on the prevalence of the six major variants of the original 1937 Beaton and Anson classification found that type A (SN trunk emerging inferior to the piriformis) occurred 90% of the time, whereas type B (common peroneal emerging through the piriformis muscle belly and tibial emerging inferior to piriformis) was seen in 8% of cases. Other, less prevalent variants and some sex and ethnic differences seem to exist.⁴ Despite hypotheses suggesting a link, no study has found a correlation between anatomic variants and gluteal pain symptoms. Furthermore, no increased incidence of anatomic variants has been detected at the time of surgery compared with the general cohort.⁷

Other Nerves

The posterior cutaneous nerve (PFCN) of the thigh originates from the sacral plexus (S1 and S2 rami) and emerges through the greater sciatic foramen inferior to the piriformis muscle belly along with the SN, the inferior gluteal nerve and artery, the internal pudendal artery, and the pudendal nerve.⁸ It lies medial to and then diverges from the SN in the deep gluteal area. It then rises posteriorly toward the fascia lata of the thigh, where it remains deep to the fascia but superficial to the biceps femoris muscle belly. Cutaneous branches of the PFCN, including cluneal and perineal branches, innervate the gluteal region, the perineum, and the posterior thigh and leg.⁸ The PFCN is vulnerable to traumatic or iatrogenic injury.

The pudendal nerve, arising from lumbosacral plexus contributions of S2-S4, provides motor, sensory, and autonomic functions by branching into the dorsal nerve of the penis/clitoris, the perineal nerve, and the inferior anal nerve. Once it exits the greater sciatic foramen, the pudendal nerve passes deep to the piriformis muscle and emerges inferiorly to it. It then weaves between the

Figure 1



The boundaries of the deep gluteal space include (1) anteriorly, the posterior acetabular column and hip joint capsule; (2) posteriorly, the gluteus maximus muscle; (3) laterally, the gluteal tuberosity, (4) medially, the sacrotuberous ligament; (5) superiorly, the inferior margin of the sciatic notch, and (6) inferiorly, the proximal origin of the hamstrings at ischial tuberosity. In addition, the sciatic nerve (7), pudendal nerve (8), superior gluteal nerve, (9) and posterior cutaneous nerve of the thigh (10) are depicted.

sacrospinous and sacrotuberous ligaments, which may act to entrap the nerve in a lobster claw fashion, about 2 to 3 cm from the superior and medial aspects of the proximal hamstring origin on the ischial tuberosity.⁹ Damage to the pudendal nerve most often results in anal and penile/clitoral pain that can radiate into the buttock or medial hamstring, is made worse by sitting, and can lead to sexual dysfunction.

The superior (SGN) and inferior gluteal nerves exit the greater sciatic foramen superior and inferior to the piriformis, respectively. Both of them are exclusively motor nerves. The SGN innervates the gluteus medius and minimus as well as the tensor fascia lata, and the inferior gluteal nerve innervates the gluteus maximus. Nerve damage is primarily traumatic or iatrogenic, reported with the lateral (Hardinge) and anterolateral (Watson-Jones) approaches. Injury or entrapment results in muscle dysfunction and atrophy but occasionally can present with buttock pain.¹⁰

The superior (SCN) and medial cluneal nerves (MCN) are pure sensory nerves that represent terminal ends of the T12-L3 spinal nerve posterior rami lateral branches.¹¹ They traverse the posterior iliac crest penetrating the thoracolumbar fascia. Part of the SCN passes through an osteofibrous tunnel formed by the thoracolumbar fascia and the iliac crest. The SCN, innervating the ipsilateral superficial buttock and low back, is most often involved in a compression syndrome.¹¹ The MCN may also lead to buttock and posterior thigh symptoms. In cases of suspected cluneal nerve entrapment, Tinel sign along the posterior iliac crest at the site of nerve compression usually reveals the pathology.¹¹

Vascular Structures

The inferior gluteal nerve and artery and the internal pudendal artery emerge from the greater sciatic foramen inferior to the piriformis muscle belly, whereas the superior gluteal exits superior to it. Rarely, these vessels are associated with arteriovenous malformations or aneurysms. In most cases, aneurysms are posttraumatic, but true isolated aneurysms leading to deep buttock pain have been reported. More commonly, several reports have documented venous varicosities in the deep gluteal area entrapping the SN and leading to radiating gluteal pain that is successfully treated with varix resection.¹²

Muscular and Tendinous Structures

The piriformis muscle is centrally located within the buttock and provides an anatomic reference for structures leaving the pelvis and entering the deep gluteal space above and below it. Its muscle belly originates from

the ventrolateral surface of the S2-S4 sacral vertebrae, gluteal surface of the ileum, and sacroiliac joint capsule. It crosses the greater sciatic foramen, and its tendon inserts on the piriformis fossa at the medial aspect of the greater trochanter. Within the deep gluteal space, the piriformis and adjacent fibrous bands have been most commonly associated with SN compression.

The internal obturator-gemelli complex and the quadratus femoris muscle, both distal to the piriformis, have also been implicated in cases of persistent gluteal pain.^{13,14} Case reports have described associated bursae, iatrogenic injury, and space-occupying lesions within these muscles such as hematomas, myositis ossificans, arteriovascular malformations, and abscesses.^{13,15}

The proximal hamstring is itself an important pain generator in this area. Puranen and Orava¹⁶ coined the term HS to describe gluteal pain secondary to tendon pathology such as tendinitis and tears or SN-irritating fibrotic bands. At its origin on the ischial tuberosity, the semitendinosus and biceps femoris conjoined tendon footprint extends about 2.7×1.8 cm.¹⁷ Adjacent to it and inferolaterally on the tuberosity, the semimembranosus attaches in a 3.1×1.1 cm crescent. The proximal hamstring footprint lies 1 cm from the SN and only 2 to 3 cm from the pudendal nerve.¹⁸ In addition, the sacrotuberous ligament shares a fascial attachment with the proximal hamstring conjoined tendon on the ischium and can be simultaneously injured. The surrounding ischial and biceps femoris bursa can also develop symptomatic inflammation.

The gluteus medius and minimus attach on the greater trochanter and can develop pathologies leading to gluteal pain. Although trochanteric bursitis or gluteal tendinitis and tears will most often present with lateral pain, a subset of patients will report posterior pain.¹⁹

History

Patients with deep gluteal pain will often present with long-standing symptoms with or without a traumatic event. Onset can be insidious or more acute and involve isolated deep buttock pain or associated sciatica-like radiation. A standard thorough examination of history should be performed. Table 1 summarizes history pearls for the major deep gluteal pain entities. The acuity of the onset and mechanism of symptoms can hint at the underlying issue. Proximal hamstring strains or tears will often present after running or explosive activities involving a quick acceleration or deceleration. Acute

Table 1. History Pearls for Deep Gluteal Pain

Diagnosis	Onset	Sitting	Walking	Radiation
Deep gluteal syndrome	Traumatic or insidious	Pain after 20-30 min	No pain	Yes. Pain or dysesthesias
Hamstring syndrome	Often acceleration/ deceleration event May be insidious	Pain after 20-30 min	Pain on heel strike of the front leg during long strides	Yes. Pain or dysesthesias
Ischiofemoral impingement	Usually insidious	No pain	Pain on extension of the back leg during long strides	Yes. Pain or dysesthesias
Pudendal nerve entrapment	Usually insidious	Pain relieved by standing or sitting on the toilet seat.	No pain	Yes. Saddle distribution or medial buttock. Burning, electrical or foreign body sensation
Posterior trochanteric bursitis	Usually insidious	Pain when getting up from a seated position	Pain	Yes. Pain or dysesthesias
Femoroacetabular impingement	Usually insidious but may be acute	Pain	Pain	Not typically
Sacroiliac disorders	Traumatic or insidious	No pain	Pain on long strides	Possibly
Lumbosacral radiculopathy	Traumatic or insidious	Pain if discogenic Less pain if spinal stenosis	Pain if spinal stenosis Less pain if discogenic	Pain or dysesthesias

lumbosacral discogenic entrapment needs to always be ruled out, but a new onset of radiating pain after heavy lifting increases diagnostic suspicion. Stress fracture symptoms may typically develop subacutely, often in the setting of increased high-impact activity, for example, an increase in running volume. Patients who place their hips at the extremes of motion for occupational or recreational reasons may also predispose the deep gluteal structures to impingement or irritation. Yoga practitioners, for example, may report pain in extremes of external rotation and hip flexion, indicating hip intra-articular or extra-articular pathology including posterior femoroacetabular impingement, hip arthritis, or posterior greater trochanteric pain syndrome. We have found that a subset of patients who spend substantial time in the cross legged position may develop painful contact between the posterior trochanter and ischium.

Painful sitting in patients who drive or are excessively sedentary at work suggests ischial bursitis, HS, or DGS. Pain during sitting may potentially help differentiate SN entrapment disorders such as DGS and HS from IFI. Patients with DGS and HS will often report an inability to sit for extended periods with concomitant radiating pain or dysesthesias. In their systematic review, Kizaki et al³ found that, in general, posterior hip pain, radicular pain, and pain aggravation when sitting for more than 20 to

30 minutes were the most commonly reported symptoms in DGS. Conversely, patients with IFI tend to be more comfortable sitting. IFI patients will instead complain of symptom exacerbation with walking, especially in terminal extension when the back leg approximates the femur to the ischium.^{20,21} Critically, however, DGS, HS, and IFI have all been associated with radiating pain and dysesthesias. Furthermore, in our experience, these entities can coexist and are not mutually exclusive of each other.

Pain medial to the ischium or in a saddle distribution along with burning, electrical or foreign body sensation exacerbated by sitting but improved by standing or sitting on a toilet seat denotes pudendal nerve entrapment.²² Corroboration of the diagnosis usually involves a pelvic floor manual test performed by an experienced pelvic floor physical therapist.

Physical Examination

After a thorough history creates a short list within the gluteal pain differential, a focused systematic physical examination is essential in setting the stage for accurate diagnosis. A complete hip examination including gait analysis, palpation, active and passive range of motion,

provocative tests, and neurovascular assessment should be performed. Many of these tests can be performed either seated or side lying.

The examination begins with an observation of gait, using a hallway or corridor if possible. Patients with greater trochanteric conditions (ie, bursitis, gluteus medius and minimum tendinitis, or tears) may exhibit a Trendelenburg gait. Internal foot progression can hint at increased femoral anteversion, which has been shown by Gomez-Hoyos et al²³ to be found more commonly in patients with IFI. During the gait examination, the long stride test helps differentiate between IFI and HS (Figure 2). HS symptoms will be exacerbated at heel strike, when the hamstrings contract eccentrically. Conversely, pain recreation in terminal extension of the back leg that is subsequently relieved by taking shorter strides suggests IFI.⁶ Specifically for IFI, Gomez-Hoyos et al²⁴ demonstrated a high test sensitivity and specificity of 0.94 and 0.85, respectively, for the long stride test. Hip abductor performance can then be further assessed with the patient standing. McGovern et al²⁵ have shown that the single leg squat test and the step-down test offer good reliability and validity in assessing coronal plane control of the gluteal muscle group. In the sagittal plane, abnormal anterior or posterior pelvic tilt has been shown to reflect dysfunction of the pelvic stabilizers and may be a risk factor for IFI and DGS.²⁶

The patient is next asked to lay supine. At this point, passive hip range of motion can be assessed. Asymmetries, especially decreased internal or external rotation with the hip flexed to 90°, suggest femoroacetabular impingement (FAI) or hip arthritis. Reproduced pain with the flexion/adduction/internal rotation (FADDIR) or flexion/abduction/external rotation (FABER) maneuver can further elucidate anterior

Figure 2



The long stride test can help differentiate between ischiofemoral impingement (IFI) and hamstring syndrome (HS). In IFI, the buttock area is painful during extension of the back leg as the ischiofemoral interval is narrowed. In hamstring syndrome, pain is felt in the ischial area during heel strike of the front leg.

or posterior FAI. A straight leg raise, looking for Lasegue sign can be performed to identify a discogenic cause of pain. The sacroiliac joint can be assessed by the FABER maneuver and sacroiliac palpation.

Finally, the prone examination can reveal areas of tenderness. Ischial tuberosity tenderness suggests HS. Tenderness just lateral to the ischial tuberosity may indicate sciatic neuritis from entrapment secondary to DGS and IFI. Tenderness medial to the ischial tuberosity strongly suggests pudendal nerve entrapment.

Special Tests

Deep Gluteal Syndrome

The rationale for most tests seeking to uncover DGS involves either placing the piriformis and external rotators on stretch, thereby exacerbating SN compression or resisting active external rotation. The FAIR test, for example, elicits pain when the hip and knee are flexed to 90°, and the hip is internally rotated and adducted across the patient's other leg, effectively placing the piriformis on stretch.²⁰

In the study by Martin et al.,²⁰ a straight leg raise had only 0.15 sensitivity but 0.95 specificity when looking at SN entrapment. In their study, they also assessed two other tests, the active piriformis (Figure 3A) and piriformis stretch tests (Figure 3B). Martin et al concluded that the combination of these two tests gives the greatest diagnostic accuracy (sensitivity 0.91 and specificity 0.80) for identifying SN entrapment.^{6,20}

To elicit the Pace sign (Figure 3C), the patient is seated with the legs abducted and dangling on either side of the table. A positive test will recreate pain with resistance to abduction and external rotation. Kizaki et al³ found that tenderness in the deep gluteal space, a positive seated piriformis test, and a positive Pace sign were the most commonly observed tests in DGS.

Figure 3



A, In the active piriformis test, the patient is placed in lateral decubitus and abducts and externally rotates the leg against resistance. Pain in the deep gluteal area suggests piriformis or external rotator entrapment of the sciatic nerve. **B**, In the piriformis stretch test, with the examiner's hand palpating the greater sciatic foramen, pain is reproduced by bringing the affected leg into knee extension with hip flexion, adduction, and internal rotation while the patient is seated. **C**, The Pace sign is positive when gluteal pain is recreated by the seated patient resisting abduction.

Hamstring Syndrome

The physical examination for HS focuses on reproducing pain in the ischial tuberosity area with either hamstring stretch or resistance to knee flexion. Tenderness over the ischial tuberosity suggests proximal hamstring pathology. A positive Puranen-Orava test (Figure 4A) reproduces pain when the patient stands and places the affected leg on a support with the knee straight and the hip at 90°.16 The modified bent knee test involves having the patient lie supine (Figure 4, B and C). The knee and hip are maximally flexed, and the examiner subsequently straightens the knee quickly.27 Cacchio et al28 found good reliability with a sensitivity and specificity of the Puranen-Orava test of 0.76 and 0.82, respectively, and of the modified bent knee test of 0.89 and 0.91, respectively. In addition, with the patient prone, pain or weakness with hamstring contraction against resistance with the knee at 30° flexion can help identify HS.

Ischiofemoral Impingement

The IFI test (Figure 5) involves having the examiner extend the affected hip while maintaining neutral positioning or adduction. Extension reproduces the pain that gets relieved with the same maneuver performed in abduction. Gomez-Hoyos showed a sensitivity and specificity of 0.82 and 0.85, respectively, for the IFI test, which in conjunction with the long stride test suggests IFI.24

Greater Trochanteric Pain Syndrome

Sciatica has been reported in patients with trochanteric bursitis, with nearly 11% of patients with sciatica in one prospective study ultimately receiving a trochanteric bursitis diagnosis.29 Greater trochanteric pain syndrome is reliably denoted by tenderness with palpation over the greater trochanter with the patient in lateral decubitus and can include bursitis and abductor tendon inflam-

Figure 4



A, The Puranen-Orava test recreates pain when the standing patient, flexes the affected hip with the knee straight and attempts to grab their foot. **B**, The modified bent knee test recreates pain in the supine patient with the affected hip and knee maximally flexed and then **(C)** rapidly extending the knee.

Figure 5



The ischiofemoral impingement test recreates pain with the affected hip brought into extension and neutral or adduction. This pain is relieved when the maneuver is performed with the hip in abduction.

mation or tearing. In this position, abduction strength is tested as well as an Ober test to assess for iliotibial band tightness. Flexion, abduction, and external rotation, the FABER test, brings the posterior greater trochanter in contact with the ischial tuberosity and can elicit pain. The iliac crest can also be palpated as some proximal gluteal muscle strains can also refer posteriorly.

Differential

Gynecological and intra-abdominal disorders can masquerade as gluteal pain and sciatica. Cysts, tumors, masses, endometriosis, or dysmenorrhea can cause intrapelvic sciatic or pudendal nerve entrapment.21 Pain related to menses, associated bowel or bladder symptoms, or an absence of any buttock tenderness to palpation warrants further imaging workup and referral when necessary.

Several common orthopaedic entities can exhibit deep gluteal pain and need to be ruled out. Stress fractures and hip arthritis can be confirmed with radiographs and magnetic resonance imaging (MRI). FAI may present with atypical lateral and posterior pain in up to 15% to 30% of patients.30 Lumbosacral disorders, including herniated discs, spinal stenosis, and spondylolysis, often manifest a gluteal pain component.

Imaging

Standard anteroposterior pelvis, Dunn lateral, false profile, and frog leg lateral images can reveal hip arthritis or FAI. Predisposing factors, for example, increased hip head-neck angle in cases of IFI, can also be ascertained.

MRI assists diagnosis in several ways. Importantly, the patient's legs should be secured in neutral rotation to

replicate normal stance and not allowed to drift into external rotation. First, pelvic axial cuts can reveal sources of intrapelvic SN entrapment. Second, the ischiofemoral interval can be evaluated on axial cuts. In a retrospective study looking at axial T1WI sequences, Xing et al³¹ found a correlation between symptomatic IFI and decreased IFI interval and quadratus femoris edema (Figure 6). Furthermore, they corroborated that an increased neck shaft angle and femoral anteversion correlate with a smaller interval, increasing the risk of IFI.³¹ Third, hamstrings can be evaluated for partial or full tearing or tendinosis. The sickle sign has been described as a high-intensity undersurface crescent-like signal on T2-weighted images denoting partial hamstring tears.³² Fourth, MRI, specifically magnetic resonance neurography, has been used to identify sciatic neuritis in the setting of DGS. Bilateral imaging is advocated to detect differences in adhesions, associated dilated vessels, volume loss of the subgluteal space, and perineural fat plane effacement.³³ Filler et al¹⁴ reported a 93% sensitivity and 64% specificity for MRI neurography in distinguishing piriformis syndrome (DGS) from other conditions with similar symptoms. Fifth, other nerve entrapments involving the pudendal nerve and SGN have been identified on MRI with focal scarring and nerve enlargement.³³ Finally, MRI is useful to rule out other extragluteal space sources of buttock pain, including acetabular labrum tears, sacroiliitis, stress fractures of the sacrum, femoral neck, head, and pubic rami, as well as gluteal tendon tears. A lumbosacral spine MRI can be obtained if a discogenic source of sciatica is suspected.

Ultrasonography has some limitations in the diagnosis of deep gluteal pain secondary to beam attenuation but is useful in dynamic imaging of various forms of hip

impingement. Ultrasonography can evaluate gluteal snapping, for example, involving the hamstring complex, the posterior trochanter, or adhesions. More importantly, ultrasonography-guided injections can shed light on symptomatic structures in the deep gluteal space. We have found guided injections of local anesthetic with or without corticosteroids within or around the quadratus femoris, piriformis, hamstring, and pudendal nerve to be indispensable in diagnostic decision making.

Although electrodiagnostic testing has been described extensively in the setting of deep gluteal pain, no study has shown a definitive use as a diagnostic aid.⁶ It is most useful for posttraumatic and iatrogenic etiologies of SN, SGL, and PN damage with motor and sensory deficits.

Nonsurgical Treatment

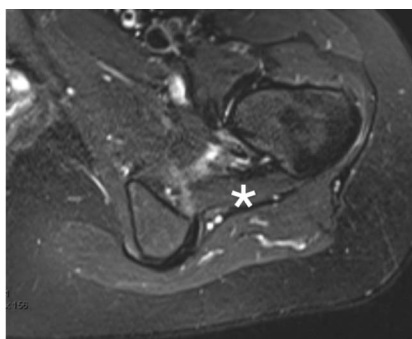
Nonsurgical treatment relies primarily on a regimen of physical therapy, home exercises, neuropsychological assessment nonsteroidal anti-inflammatories, and nonsteroidal intramuscular injections.²¹ Although several case series abound, no long-term (>2 year) studies have been conducted evaluating the efficacy of botulinum toxin in the treatment of deep gluteal pain syndromes. However, botulinum toxin injections seem to offer short- to medium-term relief in cases of piriformis syndrome or IFI and may be effective as a diagnostic tool.³⁴ Fishman et al⁷ reported at least 50% symptomatic improvement at a mean follow-up of 10.2 months in 79% of patients treated with a combined corticosteroid and lidocaine injection in addition to physical therapy. Extracorporeal shockwave therapy has been described in hamstring tendinopathy with reductions in pain reported in short- and medium-term studies.³⁵

Surgical Treatment

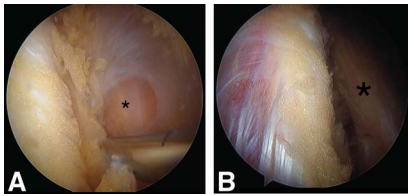
Deep Gluteal Syndrome

Both open and endoscopic procedures have been described for DGS. Historically, good results have been obtained with open SN decompression.²² Han et al³⁶ found satisfactory results in 83% of patients 1 year after open piriformis release. In their cohort, buttock pain improved more than sciatica after surgery, and patients with a traumatic etiology did not improve as much as those with an idiopathic onset. Endoscopic techniques of SN decompression have recently gained more attention (Figure 7). Recently, Park et al³⁷ reported an

Figure 6



Axial T2-weighted MRI cuts of bilateral hips in a 30-year-old woman with symptoms of left ischiofemoral impingement showing fatty atrophy and increased quadratus femoris edema (asterisk).

Figure 7

A, Sciatic nerve (asterisk) surrounded by fibrous bands. **B**, Sciatic nerve (asterisk) after endoscopic release, adjacent to proximal hamstring tendon.

improvement in VAS scores of 7.4 to 2.6 2 years after endoscopic SN decompression in patients with DGS.

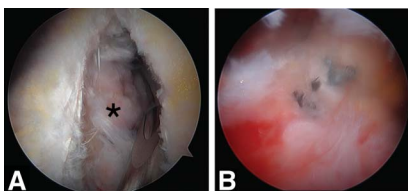
Hamstring Syndrome

Good results have been described for both open and endoscopic management of HS. In cases of HS without major tearing, Puranen outlined a surgical strategy consisting of one or more of the following¹: excision of adherent bursa,² division of semimembranosus or biceps femoris bands,³ liberation of the SN,⁴ excision of scar or fibrotic fibers, or⁵ decompressing the nerve.¹⁶ Benazzo et al³⁸ reported 88% excellent results at a mean of 71 months in partial release of the biceps femoris, semimembranosus, or semitendinosus tendon in athletes with chronic hamstring tendinitis. A recent multicenter study with minimum 2-year follow-up found high PROs and patient satisfaction after either open or endoscopic proximal hamstring repair.³⁹ (Figure 8).

Ischiofemoral Impingement

Surgical treatment for IFI can involve resecting the LT, ischial exostoses, or space occupying lesions in the quadratus femoris.⁴⁰ The medial circumflex artery lies only 18 mm from the LT and should be avoided at all costs.

Hatem et al⁴⁰ reported an increase in modified Harris Hip Scores of 51.3 to 94.2 2 years after partial endoscopic resection of the LT in patients with IFI. In a

Figure 8

Partial proximal hamstring tear. **A**, Endoscopic visualization of ischial tuberosity (asterisk) through rent created in the intact bursal side of the proximal hamstring tendon. **B**, After repair.

cohort of 16 hips, endoscopic LT resection leading to successful relief of pain and increased modified Harris Hip Scores while maintaining no residual iliopsoas weakness was described by Aguilera-Bohorquez et al.⁴¹

Summary

Gluteal pain presents both a diagnostic and therapeutic challenge for orthopaedic surgeons because of the region's complex, layered anatomy. Understanding the anatomic basis of the various overlapping disorders in the deep gluteal region and reinforcing the clinician's diagnostic armamentarium with specific physical examination tests can aid in clarifying the diagnosis. Imaging and diagnostic injections further narrow the differential. Promising nonsurgical and surgical approaches bode well for improved resolution of deep gluteal pain as clinicians become more familiar with this pernicious entity.

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