Piriformis Syndrome

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Case Description

A 45-year-old man presents to pain clinic with 6 month history of right-sided buttock pain. He describes that pain as sharp and stabbing present deep in the buttock, radiating down the leg into the heel. The pain increases after 30 min of sitting, prompting him to stand up or shift his weight to the left buttock, making hard to work during the day. Walking and lying down is least uncomfortable while bending and lifting things makes the pain worse. The patient describes a tingling sensation that occurs, whether seated or standing. He has been to physical therapy, which has not been of benefit, and he has tried rest, NSAIDs and OTC topical analgesic medications without benefit. On examination, his gait is antalgic. He has distinct tenderness to deep palpation over the gluteal muscles. Internal and external rotation of his hip is painful. Lumbar spine range of motion is within normal limits. Neuro examination is intact, and he has a negative straight leg raise test on examination.

What Is Your Preliminary Diagnosis?

The patient has a vague radicular pain with no sign of nerve compression. His neuro examination is unremarkable. There is no finding on lumbar spine physical examination. There is pain on rotation of the hip joint which requires close scrutiny of hip joint as a cause of his pain. The list of potential structures as source of pain for such kind of presentation would include sacroiliac joint, ischial or greater trochanter bursas, lumbar disc disease, lumbar facet joint, lumbosacral radiculopathies, and piriformis/gluteal muscles [1–5].

Sacroiliac joint tends to cause pain in the low back, rarely radiate below the knee, and tends to ease up with sitting. Lumbar disc pain does get worse with sitting, but pain localization tends to be over the spine, with limitation of lumbar range of motion due to pain. Facet joint pain is usually ipsilateral, gets better with sitting, and tends to occur in older patients unless due to injury. Hip joint pain does not tend to get worse with sitting, though pain with hip joint rotation can point toward the hip joint as a source of problem [6]. Considering the overall clinical features of the pain symptom constellation, it looks most likely piriformis syndrome.

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How Is the Diagnosis Confirmed?

Piriformis syndrome is a diagnosis of exclusion. The diagnosis is confirmed when a patient demonstrate certain clinical features in the absence of other pathologies as confirmed by chemical testing or imaging. Clinical feature have not been validated; pain relief after piriformis muscle injection is considered as the most diagnostic test.

Patients will describe deep buttock pain that may or may not radiate down the leg, which improves with movement and worsens by sitting/standing for even short periods of time (10–15 min) (Table 37.1). This pain may be associated with difficulty walking due to antalgic gait or foot drop, weakness of the ipsilateral lower extremity, numbness in the ipsilateral foot, or even contralateral SI pain. As these symptoms are similar to a variety of other causes of lower back, hip, and lower extremity pain, physical exam isolating the piriformis muscle is critical in differentiating it as the primary diagnosis.

In a systematic review of more than 50 case studies of piriformis syndrome, the most common presenting symptoms were buttock pain, external tenderness over the greater sciatic notch, and aggravation of the pain through sitting.

On physical exam, a patient will have point tenderness over the piriformis muscle, especially over the attachment points at the greater trochanter (Table 37.2) [5]. There may be tenderness extending to the SI joint as well. In the supine position, a relaxed patient would show ipsilateral foot external rotation, referred to as *a positive piriformis sign*∗. In the setting of chronic piriformis syndrome, surrounding neurovascular and muscular tissues can be impacted, leading to sacral plexus neuropathies as well as ipsilateral weakness of gluteus muscles, adductor magnus, quadratus femoris, and obturator externus muscles, further confounding the diagnosis. Sacral anterior rotation is often seen in piriformis syndrome, which can result in a shortening of the ipsilateral leg as well as compensatory lumbar vertebral counter-rotation, which can cause confounding lower lumbar/thoracic pain and decreased range of motion.

As there is no single test that is specific to piriformis syndrome, a variety tests frequently used to aid in the diagnosis include Freiberg, Pace, FABER, FAIR, and Beatty tests [2–4]. In the *Freiberg* test, the patient is placed in a supine or prone position, the extended hip is passively internally rotated (Table 37.3). A positive test will elicit pain in the sciatic notch. The *Pace* test is performed with the patient in the seated position with patient abducting the legs, leading to a contraction of the piriformis muscle and a resulting deep buttock pain. The *FABER* (flexion, abduction, external rotation of the hip) will result in back and deep buttock pain, as will the *FAIR* (flexion, adduction and internal rotation of the hip), reproducing the patients sciatic pain. In the Beatty test, the patient is placed in the lateral decubitus position with the painful side up. The leg on the painful side is flexed and the knee is placed on the examining table. The patient is asked to lift and hold the knee. A positive finding yields deep buttock pain [7]. Even with multiple positive physical exam tests, it can still be difficult to discern the etiology of pain and isolate the pain to the piriformis muscle. In certain instances, EMG, CT, MR, and ultrasonography have been used to further differentiate the cause. MRI or CT

Table 37.1 Symptoms of piriformis syndrome

1. Point tenderness in buttocks
2. Pain radiating down back of leg
3. Tingling down back of leg
4. Pain improves with ambulation
5. Antalgic gait

Table 37.2 Diagnostic signs of piriformis syndrome (many of below will not be met, mostly diagnosis of exclusion)

1. Pain with palpation over piriformis muscle
2. Positive Freiberg test
3. Positive pace
4. Positive FABER test
5. Positive FAIR test
6. Negative MRI spine

Table 37.3 Freiberg's criteria

Tenderness at the sciatic notch
Positive Lasègue sign
Improvement with nonsurgical treatment

of the piriformis muscle have shown both atrophy and hypertrophy of the muscle, so assessing the size of the muscle on imaging does not refute or confirm anything. The imaging of spine or pelvis is more helpful in ruling out other pathologies. Neurophysiologic testing can distinguish piriformis syndrome from disc herniation based on which muscle groups show abnormalities, with disc herniation causing nerve impingement showing abnormalities in muscles proximal to the piriformis muscle, while in piriformis syndrome the abnormalities would be distal to it [8]. Some people use delay or loss of H-reflex in the peroneal or tibial nerve as a very important diagnostic information. The delay is more pronounced in the peroneal distribution. Increase in the delay of H-reflex or its loss when the test is repeated with leg in FAIR position will confirm the entrapment of the nerve at the piriformis muscle level. CT and MR studies can show hypertrophy of the piriformis muscle or anomalous course of sciatic nerve either above or splitting through the piriformis muscle, which are known risk factors for developing this syndrome [9, 10, 11]. Radiographic studies can additionally be used to rule out alternative causes of pain. Ultrasound can also be used to assess for hypertrophy of piriformis muscle as well as anatomic variations of sciatic nerve course.

What Is the Pathophysiology of This Condition?

In order to understand how the piriformis muscle can result in sciatic nerve irritation and pain, a basic understanding of the neuroanatomy as well as rare variations in the sciatic nerve course is critical. There are two types of piriformis syndrome, primary and secondary. The primary form results from anatomic variations of the piriformis muscle in relation to the sciatic nerve. The piriformis muscle functions to work at the hip joint, acting as an abductor, flexor, and external rotator of the joint. Anatomically, the muscle spans the anterior aspect of the sacrum to the greater trochanter, with the sciatic nerve exiting the greater sciatic foramen deep along the inferior surface of the piriformis muscle (Fig. 37.1). In as much as 22% of the population, the sciatic nerve pierces the piriformis muscle, splits the piriformis muscle, or both, predisposing these individuals to piriformis syndrome [12]. Secondary piriformis syndrome results from macro-/microtrauma resulting in ischemic mass effect or local inflammation of the sciatic nerve. This version of piriformis syndrome is most often caused by macrotrauma or direct trauma to soft tissue and the piriformis muscle, resulting in soft tissue inflammation and muscle spasm. This creates an impingement point as the sciatic nerve courses under the muscle. Microtrauma can be caused by repetitive use of the piriformis muscle, seen in long distance runners, or by direct compression, seen in people

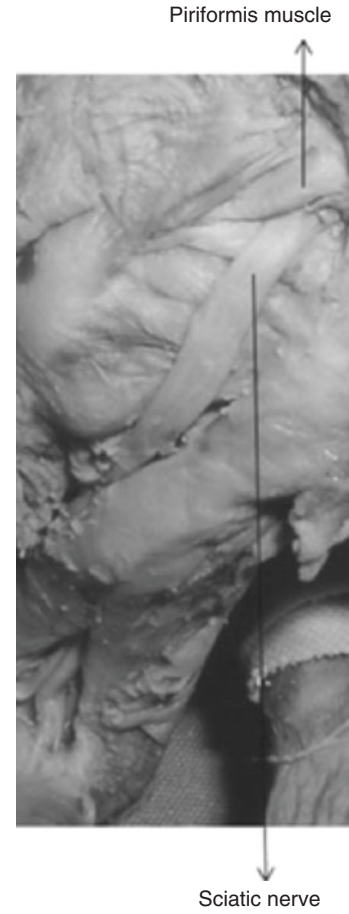

Fig. 37.1 Standard course of sciatic nerve inferior to the body of the piriformis muscle. (From Sulak et al. [22], with permission)

chronically sitting on hard surfaces, like taxi drivers or patients who sit on large wallets [7].

How Is This Problem Managed?

Initial treatment of piriformis syndrome includes rest, analgesics, and physical therapy. Pharmacotherapy includes NSAIDs, acetaminophen, muscle relaxants, gabapentin, and opiates. Initial therapies should be chosen based on symptoms and titrated to effect.

In patients refractory to conservative management, more invasive approaches should be considered. Diagnostic and therapeutic injections have been shown to be effective. While injections under fluoroscopy have been done, ultrasound-guided injections of the piriformis muscle have been shown to be superior in multiple studies.

Local anesthetic and steroid injections were evaluated in an approach correlating anatomical dissection with a combined fluoroscopic-nerve stimulator technique to demonstrate efficacy of therapy [4]. Other authors have studied the use of ultrasound as a combination modality to gauge feasibility and outcome [13, 14, 15]. One group compared the accuracy of fluoroscopically guided versus ultrasound piriformis injections in a cadaveric study, demonstrating a higher success rate in the ultrasound guided approach than in the X-ray guided group.

In addition to technique, the efficacy of different injectate has been debated with local anesthetic versus combination local and steroid preparations [16]. The addition of corticosteroid did not confer additional benefit. The limitations of the study include volume administration (5 mL) and the use of a singular steroid (betamethazone). No comparison was made with a different class of steroid, dexamethasone for example, in this evaluation.

Muscle relaxants are also a possible treatment option for patients dealing with discomfort from piriformis syndrome. Common routes include oral agents and also the use of botulinum toxin injections. A case report highlights the use of botulinum toxin A in a patient with chronic pain due to spasms [17]. Another study, a prospective single site trial, evaluated the effect of botulinum toxin using CT guidance [18]. Physical modalities including TENS, massage, and soft tissue mobilization have also demonstrated efficacy for the treatment [19].

What Is the Prognosis of This Condition?

The prognosis of the condition is unknown as most patients get better by themselves [20, 21]. Only recalcitrant cases come to seek medical help. It's suspected that a piriformis flare-up pain resolves over few weeks in general. It's more important to find the underlying factors that are causing piriformis muscle to act up, i.e., improve sitting posture, gait balance training, and fix leg length discrepancy or any anatomical factors that need surgical correction so that it does not happen again. Proper diagnosis and directed treatment can lead to improvement in symptoms and benefit for patients with piriformis syndrome.

Discussion

Prevalence

The lifetime prevalence of sciatica in the general population has been reported between 12% and 27% with an annual prevalence of between 2.2% and 19.5% [1]. Piriformis syndrome, compression of the sciatic nerve by the piriformis muscle, is a relatively rare cause of sciatica and is estimated to account for 0.6–8% of all cases of sciatica

With annual incidence of 40 million new cases of back pain, the annual incidence of piriformis syndrome would then be around 2 million cases. Prevalence rates variability is most likely due to alterations in diagnostic criteria used to diagnose piriformis syndrome.

Differential Diagnosis

The differential diagnosis of this syndrome includes lumbago, lumbar radiculopathy, ischial bursitis, and cluneal neuralgia. The possibilities

can be eliminated with a thorough history and physical examination of the patient. Lumbago would be limited primarily to axial low back pain, with piriformis presenting as buttock and posterior thigh and leg pain. Piriformis syndrome is often confused with lumbar radiculopathy; however, in the absence of disk pathology on imaging (CT or MRI), piriformis is more likely. Additionally, physical examination findings such as palpation over the piriformis muscle, provocative tests, along with the history of pressure on the piriformis muscle (such as when seated) leading to radicular-type symptoms, lead to piriformis as a diagnosis. Ischial bursitis, while in the general gluteal region, is primarily diagnosed with palpation over the ischio-gluteal bursa. Cluneal neuralgia is the irritation of the cluneal nerves over the buttock and would not be expected to lead to radicular symptoms.

Predictive Value of Different Clinical Features and Lab Testing/Imaging

The predictive values of physical exam tests have not been validated. A FAIR and FABER tests have a reported sensitivity of 0.78 and specificity of 0.80. Pace test (seated stretch test) has sensitivity of 0.53 and a specificity of 0.90, while the Lasegue's test (straight leg raise) has a sensitivity of 0.15 and specificity of 0.95. The combination of the Pace test with other tests that actively stretch piriformis muscles has shown a sensitivity of 0.91 and specificity of 0.80 for the endoscopic finding of sciatic nerve entrapment. Hence, the diagnosis of piriformis syndrome is best achieved with a combination of the history, physical examination, and diagnostic studies.

Laboratory testing including EMG testing may be performed. Usually EMG test is normal in patients with piriformis syndrome. The test is usually done to exclude other conditions. If positive, it reveals slowing of conduction velocity and or amplitude of action potentials. The degree of slowing correlates with the duration of the pathology. Fishman et al. evaluated the H-reflex of 918 patients [8]. The test was done with their leg FAIR position. He found that a delay in the H-reflex greater than 3 SD had a sensitivity of 0.88 and specificity of 0.83 for the diagnosis of piriformis syndrome. Patients who have prolonged H-reflex in the FAIR position tend to improve significantly (improvement >50%) with conservative therapy. Needle EMG of piriformis muscle tends to be normal till the very end; presence of denervation sign may be present but unlikely and a sign of severe compression. MRI of the piriformis muscle may be equivocal, but the absence of degenerative disk disease can exclude discal pathology from the differential diagnosis of piriformis syndrome. Piriformis muscle asymmetry on MRI is looked for but has not diagnostic value. MRI neurography is a little more promising. MRI of the sciatic nerve when visualized using STIR sequence may show signs of nerve irritation or edema. Hyperintensity of sciatic nerve has been seen in 86–94% patients. The MRI finding of piriformis muscle asymmetry and ipsilateral sciatic nerve hyperintensity at the sciatic notch had a specificity of 0.93 and sensitivity of 0.64 for predicting good to excellent outcome from piriformis muscle release surgery. Additionally, Ultrasound imaging of the region may demonstrate nerve entrapment and possibly muscular trauma. Increase in size of the sciatic nerve is a sign of nerve swelling at the piriformis level, but more work is needed to make the test more useful.

Strength of Evidence for Different Treatment Modalities

Rest, analgesics, and stretching exercises all have a role in the treatment of this entity. Around half the patients respond to conservative therapy. Additionally, interventional options may hasten recovery from this syndrome. Landmark technique is not reliable at all. Some sort of imaging to guide injection is almost mandatory for accuracy reason. As described above, fluoroscopic guidance was traditionally used for treatment. With the advent of ultrasound-guided intervention for this entity, a robust and dynamic modality allows for direct visualization of target and treatment delivery. In one study, MRI-guided local anesthetic injection into the piriformis muscle in patients with piriformis syndrome gave

complete relief to 15% of patients with no recurrence of pain, another 8% needed a repeat injection for complete relief, 37% had 2–4 months of relief with a subsequent recurrence despite repeat injection, 24% had less than 2 weeks of relief with subsequent recurrence, and 16% had no relief at all. There is no proof that addition of steroid to local anesthetic adds any benefit. In a double-blind, randomized, placebo-controlled trial, botulinum toxin was found to be superior to a combination of lidocaine and steroid as well as normal saline placebo for pain relief in patients with piriformis syndrome. When combined with physical therapy, injection of botulinum has shown to improve symptoms in resistant cases. When looking at more invasive treatments, including nerve blocks, steroid injections, and botox injections, there is a paucity of randomized controlled trials on injectate type for this pain condition. Surgical intervention often involves tenotomy of the piriformis muscle tendon and sciatic nerve decompression.

Future Directions or Clinical Trials in Progress

Greater use of ultrasound guidance for diagnosis and tracking of therapy is a direction of interest for pain providers. Identifying the etiology of piriformis syndrome, whether an isolated phenomenon, or the fruition of concomitant lumbago, or lumbar radiculopathy would allow providers to identify, stratify and intervene with therapy early on in the development of this entity. Early diagnosis is crucial in treating this process as intervening sooner with treatment modalities would improve treatment outcomes with less potential nerve damage.

Conclusion/Summary

Piriformis syndrome is a clinical entity that requires a pain provider to synthesize history, exam, and diagnostic tools to give a diagnosis. While a clinical diagnosis leads to treatment, precision can lead to improved outcomes. The use of combination of diagnostic tests, instead of relying on one, on physical exam allows practitioners to exclude confounding entities. The advent of ultrasound guidance for injections as well as for diagnostic purposes has led to an improvement in outcomes. Debate continues to exist as to the most effective mixture for injection to calm the musculature and underlying sciatic nerve. Future controlled trials should evaluate choice of injectate and speed and persistence of clinical result. What is not debatable, however, is the requirement for a multi-disciplinary approach, including pharmacology, interventional treatments, and physical modalities, to improve pain and numbness related to the piriformis syndrome.

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