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Idiopathic Pelvic Girdle Pain as it Relates to the Sacroiliac Joint

Piriformis Syndrome: A Narrative Review of the Anatomy, Diagnosis, and Treatment

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Abstract

Piriformis syndrome is a form of sciatica caused by compression of the sciatic nerve by the piriformis muscle. It is a relatively uncommon, but not insignificant, cause of sciatica. The diagnosis of piriformis syndrome is complicated by the large differential diagnosis of low back and buttock pain with many diagnoses having overlapping symptoms. This narrative review highlights the relevant anatomy, history, physical exam maneuvers, electrodiagnostic findings, and imaging findings that are used to diagnose piriformis syndrome. Also discussed are posterior gluteal myofascial pain syndromes that mimic piriformis syndrome. The review then outlines the different treatment options for piriformis syndrome including conservative treatment, injections, and surgical treatment. In addition, it provides the reader with a clinical framework to better understand and treat the complex, and often misunderstood, diagnosis of piriformis syndrome.

Introduction

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 6% to 8% of all cases of sciatica.[2] The entity was first described in 1928[3] and the term "piriformis syndrome" was first introduced in 1947.[4] Although piriformis syndrome has been described for almost a century, controversy and confusion remain regarding this diagnosis. Furthermore, piriformis muscle pain and dysfunction and piriformis syndrome are key parts of the differential diagnosis in people presenting with posterior pelvic girdle pain. Recent literature suggests that the term "deep gluteal syndrome" may better describe this constellation of muscle- and nerve-mediated pain.[5] This narrative review focuses on primary piriformis syndrome and posterior gluteal myofascial pain syndromes that mimic piriformis syndrome. Primary piriformis syndrome is the term used for sciatica caused by intrinsic pathology of the piriformis muscle. These pathologies include, but are not limited to, anatomical variations of the course of the sciatic nerve or the anatomy of the piriformis muscle,[6-10] damage to the piriformis muscle from trauma,[4,11-13] piriformis muscle hypertrophy,[9,14-17] and piriformis muscle spasm.[18,19] The purpose of this narrative review is to provide the reader with a clinical framework to work through the complex and sometimes nebulous diagnosis of piriformis syndrome.

Methods

In this narrative review, a literature search was performed using PubMed, CINAHL, and Embase to identify studies reporting on the anatomy, diagnosis, and treatment of piriformis syndrome. Various combinations of the following search terms were used: piriformis syndrome, posterior pelvic pain, chronic pelvic pain, and buttock pain. Additional studies were located through review of the reference lists of the above articles and through personal searches. We found just over 8000 studies and chose to include 71 in this review. The articles were chosen because of their relevance to the purpose of the review based on the authors' clinical and research experience in pelvic girdle, hip, and spine disorders.

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Diagnosis

The differential diagnosis of piriformis syndrome encompasses many other causes of low back pain, buttock pain, and sciatica including radiculopathy, lumbar spinal stenosis, sacroiliac (SI) joint-mediated pain, hip joint-mediated pain, facet joint-mediated pain, greater trochanteric pain syndrome, and pain intrinsic to the buttock musculature and associated tendons and fascia. Hopayian et al[20] note that arguments have been made for the overdiagnosis[21] and underdiagnosis[22,23] of piriformis syndrome. Given the lack of well-validated diagnostic criteria, piriformis syndrome is often a diagnosis of exclusion, as many of these more common pathologies must be ruled out. Below we describe the relevant anatomy, history, and physical exam findings as well as the use of electromyography (EMG) and imaging techniques in the diagnosis of piriformis syndrome.

Anatomy

It has been hypothesized that anatomical variations in the sciatic nerve and piriformis muscle could predispose to the development of piriformis syndrome.[4,7] The piriformis muscle originates on the anterior surface of the sacrum and the sacrotuberous ligament, runs laterally through the greater sciatic foramen, and inserts on the inner surface of the superior greater trochanter (Figure 1).[24] The piriformis muscle functions as a hip external rotator when the hip is extended and as a hip abductor when the hip is flexed.[25] The piriformis muscle is innervated by a nerve composed of branches of the posterior division of the ventral rami of S1 and S2. Classically, the superior gluteal artery and superior gluteal nerve emerge above the muscle and the inferior gluteal artery and inferior gluteal nerve emerge below the muscle..

In a classic study of 240 cadaver dissections, Beaton and Anson describe six different anatomical variations in the relationship between the sciatic nerve and the piriformis muscle (Figure 2).[6] In the study, 90% of cadavers had traditional anatomy with an undivided sciatic nerve emerging below the piriformis muscle. Other variations observed included a divided sciatic nerve passing through and below the piriformis muscle, a divided sciatic nerve passing above and below the piriformis muscle, and an undivided nerve passing through the piriformis muscle. Two additional hypothetical variations were described, but not observed, including a divided sciatic nerve passing through and above the piriformis muscle and an undivided nerve emerging above the piriformis muscle.[6] Additional anatomical variations have also been reported in the literature.[10] In a systematic review and meta-analysis of 18 studies with 6602 cadavers, an abnormal relationship between the sciatic nerve and piriformis muscle was observed in 16.9% of cadavers.[26] Similarly, in a review of 783 hip magnetic resonance imaging (MRI) studies, 19.2% of studies revealed an abnormal

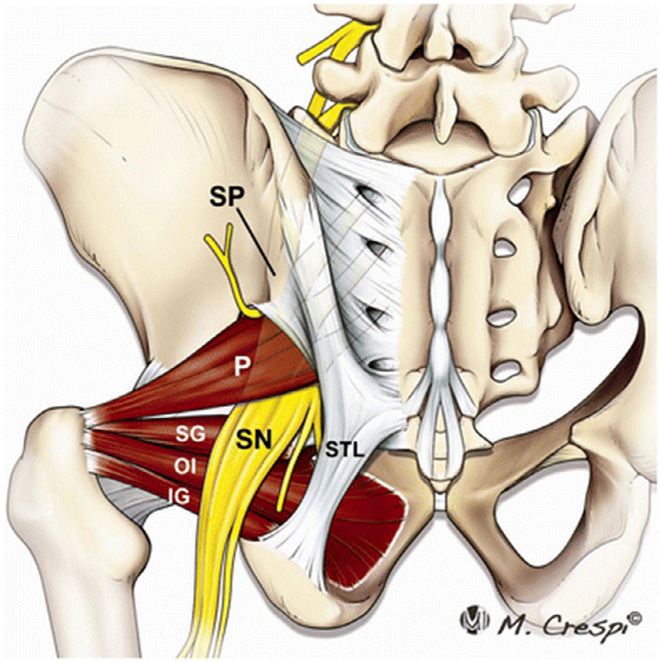

Figure 1. Normal anatomy of the subgluteal space from a posterior view. SP, sacral plexus; SN sciatic nerve; STL, sacrotuberous ligament; P, piriformis muscle; SG, superior gemellus muscle; OI, obturator internus muscle; IG, inferior gemellus muscle [reprinted with permission].[24]

relationship between the sciatic nerve and piriformis muscle.[27] Despite the assumption that an abnormal relationship between the sciatic nerve and piriformis muscles predisposes a patient to piriformis syndrome, in both of these studies, there was no significant difference in the incidence of piriformis syndrome between patients with traditional anatomy compared to those with anatomical variations.[26,27]

In addition to anatomical variations in the relationship between the sciatic nerve and piriformis muscle, variations in the piriformis muscle body and tendon have also been observed. In a study of 112 cadaveric specimens, the diameter of the piriformis tendon varied between 3 and 9 mm. The insertion of the piriformis muscle was found to be variable, with only 53.6% of tendons exhibiting traditional anatomy and inserting on the upper border of the greater trochanter via a rounded tendon (Figure 3). In 29.5% of specimens, the piriformis tendon joined with the superior gemellus tendon and then fused with the obturator internus tendon prior to inserting on the medial surface of the greater trochanter. In 13.4% of specimens, the piriformis tendon fused with the obturator internus and gluteus medius muscles prior to inserting on the medial greater trochanter. Finally, in 3.6% of specimens, the piriformis tendon fused with the fibers of the gluteus medius and inserted on the upper surface of the greater trochanter.[28] Because 43% of the above cases demonstrated fusion of the piriformis tendon

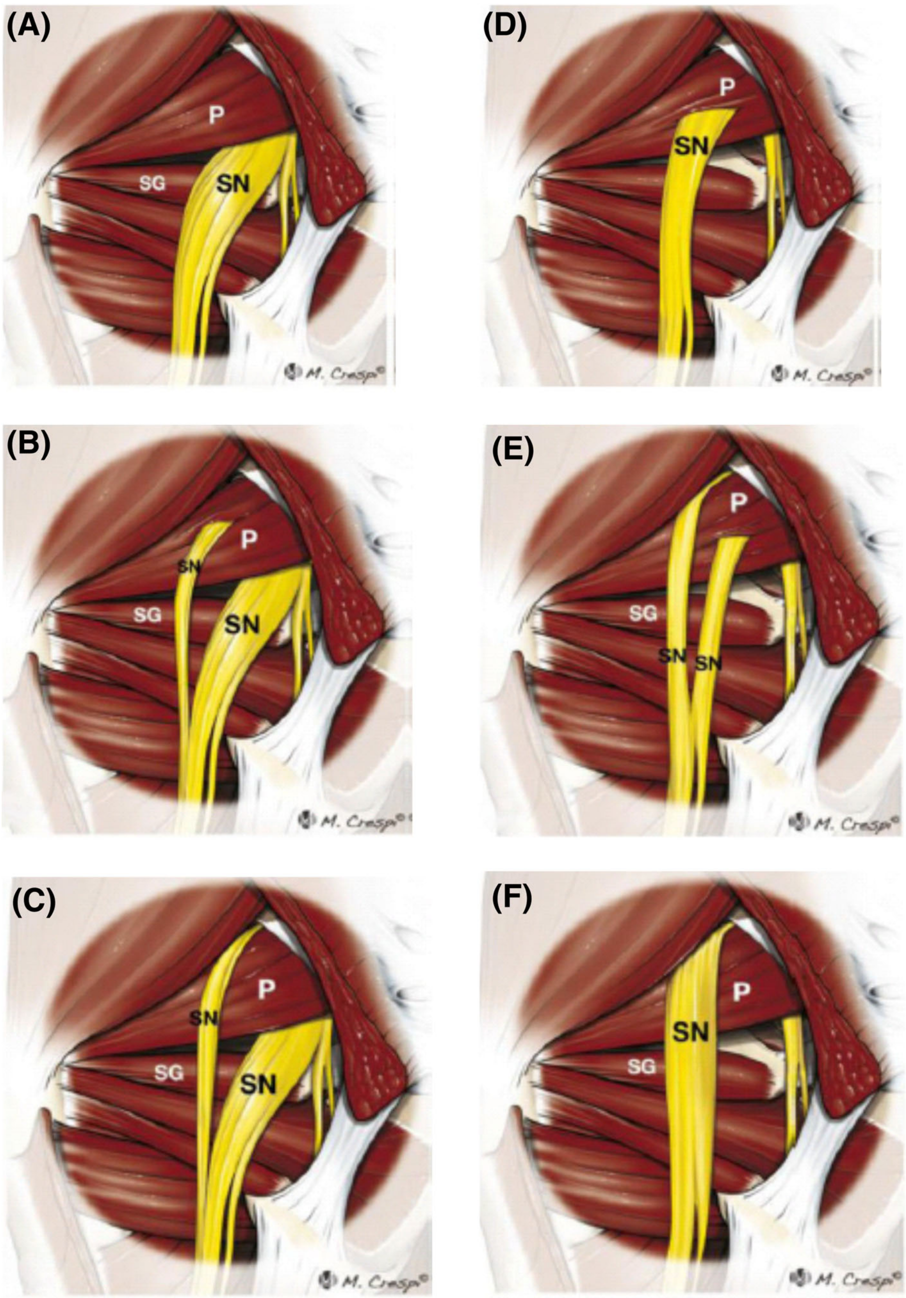

Figure 2. Anatomical variations in the relationship of the piriformis muscle and sciatic nerve as described by Beaton and Anson. (A) Traditional anatomy: an undivided nerve emerges below the piriformis muscle. (B) A divided nerve passes through and below the piriformis muscle. (C) A divided nerve passes above and below the piriformis muscle. (D) An undivided nerve passes through the piriformis muscle. (E) A divided nerve passes through and above the piriformis muscle. (F) An undivided nerve emerges above the piriformis muscle. SN, sciatic nerve; P, piriformis muscle; SG, superior gemellus muscle [reprinted with permission].[24]

with the obturator internus tendon prior to insertion, passive and active internal rotation of the hip in these patients would cause stretching of both the piriformis and obturator internus. Thus hip internal rotation could cause sciatica via the piriformis muscle impinging on the sciatic nerve or cause buttock pain via the stretching of a thickened or stiff obturator internus.[28] Anatomical variations confound the use of physical exam maneuvers, which often rely on passive hip internal rotation to diagnose piriformis syndrome.

Further complicating the diagnostic evaluation, when the Lasègue sign (straight leg raise with patient supine) was performed intraoperatively in six patients with suspected piriformis syndrome without fusion of the piriformis and obturator internus tendon, the obturator internus muscle, and not the piriformis muscle, impinged the sciatic nerve early in hip flexion.[29] In addition, there was successful decompression of the sciatic nerve and pain reduction after sectioning of the obturator internus tendon at its insertion on the greater trochanter.[29]

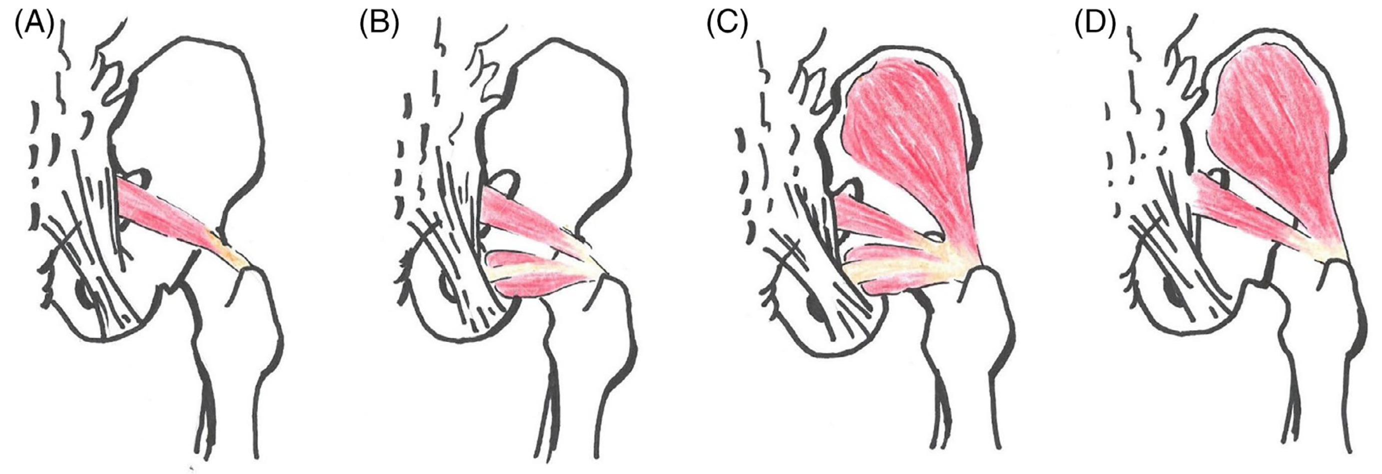

Figure 3. Anatomic variations of the piriformis tendon insertions [printed with permission]. Original drawing. (A) Piriformis traditional anatomy 53.6%. (B) Piriformis fused with the superior gemellus and obturator internus 29.5%. (C) Piriformis fused with obturator internus and gluteus medius 13.4%. (D) Piriformis fused with gluteus medius 3.6%.

Furthermore, Dalmau-Carola describes a patient with symptoms of piriformis syndrome who had only 50% improvement with a piriformis muscle injection. However, the patient had lasting, 100% relief after an injection was performed in the obturator internus muscle, suggesting that the obturator internus muscle contributed to the patient's pain.[30]

Given the common anatomical variations and multiple potential anatomical areas of sciatic nerve compression, recent literature has suggested the use of the term "deep gluteal syndrome" as a possible alternative to piriformis syndrome.[5] Regardless of the terminology, understanding the traditional anatomy and common anatomical variations allows for a better understanding of the pathogenesis of piriformis syndrome. In addition, variations in the tendinous insertion of the piriformis muscle and its relationship to the external rotators of the hip and gluteus medius muscle reveal that myofascial pain derived from the muscles and tendons of the external rotators of the hip, specifically the obturator internus, could be an important mimic of piriformis syndrome.

History

When Robinson first introduced the term "piriformis syndrome" in 1947, he described six cardinal features:

- History of trauma to the sacroiliac and gluteal regions
- Pain in the region of the SI joint, greater sciatic notch, and piriformis muscle that can extend down the limb and cause difficulty with walking
- Acute worsening of the pain caused by stooping or lifting, which can be relieved by traction on the affected leg
- Painful sausage-shaped mass detected on palpation of the piriformis muscle
- Positive Lasègue sign
- Gluteal atrophy depending on the duration of the condition.[4]

Freiberg has also described three criteria to identify piriformis-induced sciatica including:

- Tenderness at the sciatic notch
- Positive Lasègue sign
- Improvement with nonsurgical treatment.[31]

It has been argued that Freiberg's criteria are too broad and that although Robinson's criteria accurately identify piriformis syndrome, the criteria are overly specific and thus identify only a fraction of all piriformis syndromes.[32] Multiple authors have sought to further characterize the symptoms unique to piriformis syndrome. Patients with piriformis syndrome often present with hip pain, buttock pain, dyspareunia (in female patients), and sciatica.[33] The pain is often aggravated by prolonged sitting, such as driving, or by rising from a seated position.[28] 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.[20] These findings remained consistent in a recent, updated systematic review.[34] A retrospective review of 26 cases noted that buttock pain and sciatica were seen in 100% of cases.[22] In addition, 92% of patients had a history of trauma to the piriformis muscle ranging from falls to abnormal stretching of the muscle during athletic events.[22] Although the symptoms described earlier are consistent with piriformis syndrome, they remain nonspecific, and similar symptoms can be observed in many other conditions.

Physical Examination

Although no physical examination maneuver is diagnostic of piriformis syndrome, physical examination can help to support a diagnosis of piriformis syndrome and can be useful for eliminating competing diagnoses from the differential diagnosis. When piriformis syndrome is suspected, a complete examination of the low back, pelvis, buttock, and lower extremities encompassing inspection, range of motion, palpation, strength, sensation, and special tests, which can vary depending on the patient's presenting symptoms, should be completed.

Palpation is thought to play a significant role in the diagnosis of piriformis syndrome. Tenderness to deep palpation of the piriformis muscle was present in 92% of cases reviewed by Durrani.[22] In addition, in a review of more than 50 case studies on piriformis syndrome, external tenderness to palpation over the greater sciatic notch was one of the four most common presenting features of piriformis syndrome.[20,34] Furthermore, a palpable, tender, sausage-like mass over the piriformis muscle was one of Robinson's original six criteria to diagnose piriformis syndrome.[4] Furthermore, fibrous bands in the area of the piriformis and other posterior hip girdle muscles have been described in piriformis syndrome.[35] However, given the relatively small size and deep depth of the piriformis muscle within the buttock, the accuracy of piriformis muscle palpation has been called into question.[28] Sonopalpation with dynamic ultrasound can be utilized for more accurate assessment of the painful muscle and at the same time qualify any anatomical variations or pathological change to the muscle or tendon structure. Often, sonopalpation reveals that the piriformis muscle is not the sole pain generator and the external rotators or gluteal muscles are also involved.[36] Internal palpation of the piriformis muscle to assess for tenderness can be performed via vaginal or rectal exam. In a review of 26 cases of piriformis syndrome, all 26 cases had reproduction of pain upon rectal or vaginal palpation of the piriformis muscle.[22]

Multiple physical examination maneuvers have been identified to help diagnose piriformis syndrome (Figure 4). Several tests are thought to reproduce sciatica in piriformis syndrome via passively stretching the piriformis muscle causing subsequent compression of the sciatic nerve. The Freiberg maneuver involves forcefully internally rotating the affected leg while the patient is supine.[11] In the FAIR test, the patient's hip is placed in *f*lexion, *a*dduction, and *i*nternal *r*otation.[37] This test can be performed in either the side-lying or supine position. In these tests, a reproduction of the patient's typical pain in the posterior pelvis or paresthesia represents a positive finding.

Other maneuvers are thought to induce sciatica by causing active piriformis muscle contraction and subsequent compression of the sciatic nerve. In the active piriformis test, the patient is placed in the lateral side-lying position and the patient actively abducts and externally rotates the hip while the examiner resists these movements.[38] A positive test reproduces the patient's typical pain and has a reported sensitivity of 0.78 and specificity of 0.80.[38] In the similar Beatty test, a side-lying patient holds their flexed hip in abduction against gravity.[13] In the Pace test, the practitioner resists hip abduction with the patient in a seated position.[39] A positive Pace or Beatty test is indicated by reproduction of the patient's typical buttock pain, weakness, or paresthesia on the affected side compared to the unaffected side.

Additional maneuvers are thought to create tension along the irritated sciatic nerve and recreate the patient's symptoms. In the seated piriformis stretch test the patient is seated on the edge of the exam table with the hip flexed to 90° and the knee extended. The examiner palpates the sciatic notch and provides hip adduction and internal rotation.[38] The straight leg raise with the patient supine (also known as Lasègue sign) is performed with the knee in full extension. The examiner raises the leg up to the endpoint of hip flexion or until the patient complains of radiating pain or paresthesia. For both maneuvers, a positive result is indicated by the reproduction of the patient's typical gluteal pain or paresthesia. The seated piriformis stretch test has a reported sensitivity of 0.53 and a specificity of 0.90, while the straight leg raise has a sensitivity of 0.15 and specificity of 0.95.[38] The combination of the seated piriformis stretch test with the active piriformis test has shown a sensitivity of 0.91 and specificity of 0.80 for the endoscopic finding of sciatic nerve entrapment.[5] These physical examination nerve stretch tests overlap with signs of radiculopathy or any cause of neural tension and must be used in conjunction with other physical examination maneuvers and diagnostic testing.

Electrodiagnostic Testing

Electrodiagnostic testing in the setting of piriformis syndrome is often normal and is most useful to exclude other conditions such as lumbosacral radiculopathy. In piriformis syndrome, nerve conduction studies are often normal, but can show conduction slowing or decreased amplitude of sensory nerve action potentials and compound motor action potentials.[40] The degree of slowing has been shown to correlate with the duration of piriformis syndrome symptoms.[40] Fishman et al assessed the H-reflex of 918 patients (1014 legs) while their leg was in hip flexion, adduction, and internal rotation (FAIR position) and 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 according to the following piriformis syndrome diagnostic criteria. At least two out of three positive of the following: (1) pain at the intersection of the sciatic nerve and the piriformis muscle on FAIR test; (2) tenderness to palpation at the intersection of the piriformis muscle and the sciatic nerve; (3) positive Lasègue sign. Of the patients with a significantly prolonged H-reflex in the FAIR position, 81% experienced greater than 50% improvement with conservative therapy.[32] Needle EMG results in piriformis syndrome are often normal; however, with longstanding

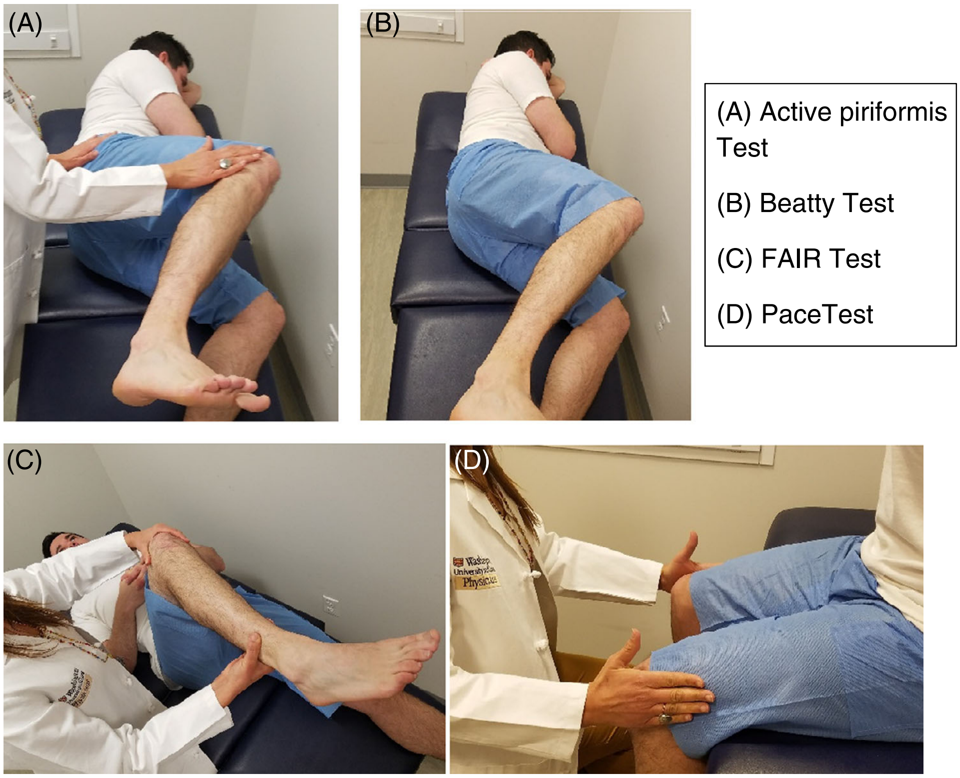

Figure 4. Physical examination special tests for piriformis syndrome. (A) Active piriformis test. (B) Beatty test. (C) FAIR test. (D) Pace test.

nerve compression, denervation may be seen in sciatic nerve innervated muscles.[41] Nevertheless, EMG remains useful to exclude competing diagnoses, with normal paraspinal muscles potentially differentiating peripheral sciatic nerve lesions from lumbosacral radiculopathy.

Imaging

Like electrodiagnostic testing, diagnostic imaging is instrumental to exclude other sources of lower extremity nerve pain. Computed tomography (CT) scan can be used to exclude structural entities causing compression of the sciatic nerve including tumor, hematoma, or abscess, but MRI is the preferred imaging tool to assess patients thought to have piriformis syndrome.[42] MRI of the spine is critical for the assessment of the spinal canal and nerve roots when excluding radiculopathy or spinal stenosis as a cause of sciatica or buttock pain. MRI of the pelvis can also be useful to diagnose piriformis syndrome. MRI can identify anatomical abnormalities that may predispose a patient to piriformis syndrome[43] and can also identify side-to-side differences such as an enlarged piriformis muscle, which has been observed in patients with piriformis syndrome.[14,44] However, this finding is not pathognomonic as many patients with piriformis syndrome do not exhibit piriformis muscle enlargement on MRI[18,43] and piriformis muscle enlargement on MRI has been reported in 19% of asymptomatic patients.[45]

MR neurography has allowed for improved visualization of peripheral nerves by suppressing the signal from surrounding tissue.[46] MR neurography was performed on 14 patients with unexplained sciatica and unremarkable MRI of the lumbar spine. Twelve patients (86%) had increased Short T1 Inversion Recovery (STIR) sequence signal in the ipsilateral sciatic nerve. In eight of those patients, the abnormal signal was seen at or just inferior to the level of the sciatic notch and piriformis muscle.[46] In another study, 239 consecutive patients with pain in the distribution of the sciatic nerve and in whom a diagnosis could not be established or who failed lumbar spine surgery, underwent MR neurography of the sciatic nerve. Edema or hyperintensity in the ipsilateral sciatic nerve relative to the contralateral side was observed in 94% of patients, and of these patients, 88% had reproduction of their symptoms with the FAIR position.[18] The finding of piriformis muscle asymmetry and unilateral 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.[18]

Ultrasound has been established as a reliable tool to diagnostically visualize myofascial trigger points, which may be seen in a piriformis muscle with increased resting tone.[47-49] In addition, high-resolution ultrasound has been used to diagnose nerve entrapment in the upper extremities, as nerve compression is correlated with increased nerve cross-sectional area as seen on ultrasound.[50] Ultrasound has been shown to be a reliable tool in the assessment of the sciatic nerve cross-sectional area in healthy controls,[51] but additional research is needed regarding the ultrasonographic assessment of the sciatic nerve in patients with suspected piriformis syndrome.

Treatment

Multidisciplinary Care

Once a diagnosis of piriformis syndrome appears likely, the initial management focuses on nonsurgical multidisciplinary care. Medications, including nonsteroidal antiinflammatory drugs (NSAIDs), muscle relaxants, and neuropathic agents such as gabapentin and pregabalin, and physical therapy remain mainstays for the treatment of piriformis syndrome.[42] It is important to guide treatment based on the practitioner's physical examination and specifically the assessment of the length and tension of the piriformis muscle. The most common pattern is a piriformis muscle that is shortened and assessed to have increased resting tone with or without the presence of taut bands or myofascial trigger points. This pattern can secondarily cause external rotation of the hip and, over time, shortening of the hip lateral rotators. However, some patients may present with an internally rotated hip and a secondarily overstretched piriformis with or without taut bands and myofascial trigger points. Taut painful bands noted on palpation of the piriformis can be treated manually in a number of ways including: trigger point injection of anesthetic, dry needling, acupuncture, manual overpressure, and massage. Adjunct modalities to any of these can include heat and muscle stimulation. The initial goal of physical therapy is to restore proper length to the muscle and release myofascial trigger points that may be present, hypothetically reducing the compressive force on the sciatic nerve.[32,52] Therapy should focus on lumbosacral stabilization, hip strengthening, and correction of biomechanical errors across the hip, pelvis, and spine that could predispose to gluteal pain and if not corrected contribute to recurrence.[53] In patients found to have short deep lateral rotators, care should be taken to assess not only muscle length but also the joint position of the femoral head in relation to the acetabulum. Often a femoral head that moves repetitively in an anterior gliding position relative to the acetabulum can result in a stiff posterior hip capsule that, when mobilized, helps to improve muscle length and neuromotor control re-education. In patients found to have reproduction of pain distally in the leg with provocative maneuvers that stretch the sciatic nerve, neuroglides as described by Butler can be very helpful in reducing the lower extremity pain and can be used as a monitor of progress during the course of treatment.[54] In 250 patients diagnosed with piriformis syndrome, treatment with medications (NSAIDs and muscle relaxants) and physical therapy had a resolution of symptoms rate of 51.2%.[55] Fishman et al describe a physical therapy protocol which, when used in combination with triamcinolone acetate injection into the piriformis muscle, led to at least 50% relief in 79% of their 665 patients who had prolonged H-reflex on nerve conduction studies while in the FAIR position.[32] However, the results of this study need to be interpreted with caution as there was no control group and, as described below, intramuscular glucocorticoid injections have fallen out of favor.

Injection

Injections into the body of the piriformis muscle in patients with piriformis syndrome can be both diagnostic and therapeutic. These injections have been performed blindly or with EMG, fluoroscopic, ultrasound, MRI, or CT guidance.[33] Some guidance methods are likely superior to others, as a cadaveric study demonstrated only 30% accuracy for anatomic landmark and fluoroscopic-guided injections into the piriformis muscle, while ultrasound guided injections had an accuracy of 95%.[56] In 162 patients diagnosed with piriformis syndrome who underwent MRI-guided local anesthetic injection into the piriformis muscle, 15% had complete relief with no recurrence of pain, 8% had 2-4 months of relief with lasting relief after a repeat injection, 37% had 2-4 months of relief with a subsequent recurrence, 24% had less than 2 weeks of relief with subsequent recurrence, and 16% had no relief.[18] Corticosteroid intra-muscular injections in isolation, and in combination with local anesthetic, have also been demonstrated to help improve pain with piriformis syndrome.[32,57-60] In a randomized, double-blind study, 57 patients with unilateral hip pain with a positive FAIR test and tenderness at the piriformis muscle underwent ultrasound-guided piriformis muscle injection with either lidocaine or lidocaine plus betamethasone. Both groups experienced significant reduction in their pain, but there were no statistical differences between the two groups.[61] As there was equal improvement in pain, the authors concluded that it is reasonable to perform the injections with anesthetic alone. Given these results combined with the muscular atrophy and other side effects associated with intramuscular glucocorticoid injection, intramuscular glucocorticoid injection should be considered in only very limited cases.

In recent years, botulinum toxin injections have shown patient-reported improvement in pain when included for the treatment of piriformis syndrome.[62-64] MRI evidence has demonstrated that botulinum toxin injections into the piriformis muscle led to a decrease in the thickness and volume of the muscle.[65] This finding likely explains the effectiveness of botulinum toxin in treating piriformis syndrome.[65] 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.[19] Furthermore, botulinum toxin injection into the piriformis muscle combined with physical therapy provided greater than 50% relief in 24 of 27 patients with piriformis syndrome and led to a decrease in the mean Visual Analog Scale from 6.7 to 2.3.[66] In a more treatment-resistant population, 122 patients with piriformis syndrome who failed conservative management with medications and physical therapy, botulinum toxin injection led to "good to very good" pain relief in 77% of patients.[55] Despite the effectiveness of botulinum toxin, its cost often prevents it from being a first-line treatment for piriformis syndrome.[67]

Surgical Release

Surgical intervention often involves tenotomy of the piriformis muscle tendon and sciatic nerve decompression.[68] Careful patient selection and exclusion of competing diagnoses prior to the operation is imperative. Some studies have reported positive outcomes with surgical intervention in piriformis syndrome,[18,32,69-71] but to our knowledge, no large, prospective, randomized, controlled trials have been performed. Given the relative success of the more conservative treatments described above, surgical release should be considered only in cases that are refractory to other treatments.

Conclusion

Sciatica is relatively common in the general population and piriformis syndrome is thought to represent 6%-8% of all cases of sciatica. The diagnosis of piriformis syndrome is complicated by the large differential diagnosis, with many diagnoses having overlapping symptoms. As there is currently no definitive diagnostic test, piriformis syndrome remains a diagnosis of exclusion. Nevertheless, history, physical examination, electrodiagnostic testing, and clinical imaging can narrow the differential diagnosis and lead to a more likely diagnosis of piriformis syndrome. The advent of MR neurography has provided additional diagnostic evidence when attempting to establish a diagnosis of piriformis syndrome. Additional compressive etiologies other than the piriformis muscle have led to the consideration of the alternative term, "deep gluteal syndrome." Regardless, nonsurgical treatment remains the mainstay of piriformis syndrome treatment with a focus of addressing biomechanical faults. Injections with local anesthetic, corticosteroid, and botulinum toxin have shown varying degrees of effectiveness in the treatment of piriformis syndrome, but the risks of intramuscular steroid injection seem to outweigh the benefits of that particular treatment. Although surgical release may be an option in a small number of recalcitrant cases, there is a lack of large, prospective, randomized, controlled trials comparing surgical and nonsurgical treatment.

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