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Piriformis Syndrome: A Cause of Nondiscogenic Sciatica

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Article Outline

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Abstract

Piriformis syndrome is a nondiscogenic cause of sciatica from compression of the sciatic nerve through or around the piriformis muscle. Patients typically have sciatica, buttocks pain, and worse pain with sitting. They usually have normal neurological examination results and negative straight leg raising test results. Flexion, adduction, and internal rotation of the hip, Freiberg sign, Pace sign, and direct palpation of the piriformis cause pain and may reproduce symptoms. Imaging and neurodiagnostic studies are typically normal and are used to rule out other etiologies for sciatica. Conservative treatment, including medication and physiotherapy, is usually helpful for the majority of patients. For recalcitrant cases, corticosteroid and botulinum toxin injections may be attempted. Ultrasound and other imaging modalities likely improve accuracy of injections. Piriformis tenotomy and decompression of the sciatic nerve can be done for those who do not respond.

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Introduction

Piriformis syndrome (PS) is an elusive and difficult diagnosis to make. Even the very definition of this syndrome is reported variably in the literature ([15]). In its most simplistic form, PS is a nondiscogenic cause of sciatica that is due to impingement of the sciatic nerve through or around the piriformis muscle. The literature variably associates PS with buttocks pain and sometimes with low back pain. It may account for anywhere from 0.3% to 6.0% of all cases of sciatica and low back pain ([2,9]). It has been described as

being both under- and overdiagnosed, with decent arguments by opponents and proponents both ways ([6,10,21]).

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ANATOMY

PS has the shape of a flat pyramid and originates from the anterior surface of the second through the fourth sacral vertebrae, sacrotuberous ligament, and the superior margin of the greater sciatic notch. It exits transversely through the greater sciatic notch and inserts on the superior greater trochanter ([2]). It should be noted that changing the position of the hip changes the function of the piriformis. With the hip in flexion, the piriformis is an abductor; however with the hip in extension, it is an external rotator. The sciatic nerve divides distally to form the tibial and peroneal nerves. Most often, the sciatic nerve leaves the greater sciatic notch and lies inferior to the muscle belly of the piriformis. One possible etiology for PS is anatomical variation in the piriformis and the sciatic nerve ([15]). A dissection study of 294 cadavers by Natsis et al. ([18]) demonstrated that, 94% of the time, the piriformis muscle and sciatic nerve followed a typical anatomical pattern. Roughly 4% of the time, the common peroneal nerve passed through and the tibial nerve below a bifid piriformis muscle.

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ETIOLOGY

Some providers consider any sciatica without a discogenic cause to be PS. This is ill-advised, as any lesion of the proximal sciatic nerve in the area of the sciatic notch could cause symptoms and may not directly relate to the piriformis muscle at all. These lesions may occur due to endometriosis, tumors, hematomas, fibrosis, aneurysms, false aneurysms, or arteriovenous malformations ([21]). These lesions, along with disc disease and PS, make up a differential for sciatica (Table 1) ([19]).

Sacroiliac joint dysfunction
Aneurysm or pseudoaneurysm of the inferior gluteal artery
Thrombosis of iliac vein
Gluteal varicosities (causing vascular compression syndrome)
Herniated intervertebral disc
Piriformis Syndrome
Posterior facet syndrome
Renal stones
Endometriosis
Tumors
Hematomas
Fibrosis
Arteriovenous malformations

Table 1

Image Tools

Most experts agree that previous gluteal trauma and posttraumatic scarring are important causes for PS ([1,11,21]). Benson et al. ([1]) hypothesized that etiology is due to hematoma formation and scarring between the sciatic nerve and external rotators. His case series of posttraumatic PS had electromyographic (EMG) abnormalities in muscles supplied by the sciatic nerve that resolved after piriformis release. All of these patients had falls or direct blows to the gluteal muscle, resulting in intractable sciatic nerve pain.

Fishman and Schaefer propose ([6]) that PS is a functional entrapment of the sciatic nerve. This is arrived at because of EMG studies that demonstrate H-reflexes when positioning the hip in a position of flexion, adduction, and internal rotation (FAIR) ([5]). This will be discussed further under Diagnostic Studies.

It also is proposed that PS could develop as a myofascial pain syndrome from the gluteal muscle and external rotator trigger points ([11]). Underlying piriformis spasm from hypertrophy and overuse also are possible etiologies ([11]).

Actual case studies of PS in sports are reported uncommonly. A review of the literature only found two case studies specifically representing a sports case. One was a posttraumatic case of PS from an ischial tuberosity avulsion fracture and subsequent exostosis in a 19-year-old rugby player ([14]). His pain resolved completely in 5 months with conservative treatment. The other case was PS in a female runner that again resolved with conservative treatment ([12]). Despite the minimal reporting, it is likely that PS occurs more frequently in sports, given the pathomechanics described previously.

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HISTORY AND PRESENTATION

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Patients may present acutely, as in cases of posttraumatic PS or, insidiously, in cases of overuse or myofascial pain from the piriformis ([11]). Patients will have sciatica and frequently have gluteal pain. There also have been reports of pain and/or paresthesias in the back, groin, perineum, buttocks, hip, back of thigh, calf, foot, and rectum ([11]). Dyspareunia has been reported in women. Patients also report intense pain with sitting or squatting. A systematic review by Hopayian et al. ([10]) showed that buttock pain was very common and more common than low back pain, happening up to 95% of the time. Pain aggravated by sitting also was common, occurring up to 97% of the time. They noted the most discrepancy with dyspareunia and found reporting of it to be unreliable ([10]).

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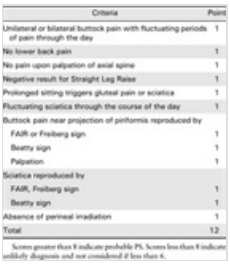
PHYSICAL EXAMINATION

Initial evaluation should start with the lumbar spine, as that would be the most common origin for sciatica. The examination should take into account gait, posture, and alignment, as well as leg length discrepancies ([2]). In addition, examination of the hip, pelvis, and sacroiliac joint should be performed. Sensory, motor, and deep tendon reflexes should be evaluated. In PS, these are typically normal ([2,11]). External tenderness to palpation of the piriformis is present up to 92% of the time and should be differentiated from nonspecific posterior gluteal pain ([2,10]). The piriformis should be palpated just posterior to the hip joint close to the sciatic notch ([2]). There may be reproduction of symptoms as well as tenderness. A positive result for straight leg raise (SLR) is reported but demonstrated variably. In the systematic review by Hopayian et al. ([10]), only 31% of patients had a positive result for SLR. It should be noted that a positive SLR has a pooled sensitivity of 91% but a low specificity of 29% for discogenic sciatica ([3]). Therefore an SLR sign could indicate multiple pathologies. A positive cross straight leg test result adds specificity of about 88% but is not reported in the PS literature ([3]).

There are a variety of specific individual tests that stretch the piriformis and can be used for the diagnosis of PS. The Freiberg sign is performed by putting the hip in extension and internal rotation and having the patient externally rotate against resistance. A positive examination result is reproducing pain around the piriformis or inducing sciatic symptoms. This is positive in up to 63% of patients ([10]). The Pace sign is performed by resisting abduction and external rotation of the hip while the patient is in a seated position. Since the piriformis is an abductor in hip flexion, this also stretches the piriformis. The Pace sign occurs 30% to 74% in PS ([10]). The FAIR test or the piriformis stretch may illicit pain ([2,5]). The Beatty sign also has been described and is accomplished by elevating the flexed leg on the irritated side while the patient lies on the asymptomatic side. Again pain and reproduction of the symptoms are a positive test result ([11]). It should be noted that these tests should be held for 30 s to a full minute, such as when doing the Phalen test for carpal tunnel syndrome, as they serve to irritate the sciatic nerve.

Michel et al. ([16]) proposed a unique scoring system to determine diagnostic criteria in PS. A 12-point clinical scoring system was developed. and a “probable” diagnosis of PS was considered if the score was ≥8 (Table 2). Sensitivity and specificity of the scores were determined to be 96.4% and 100%, respectively, with a positive predictive value of 100% and negative predictive value of 86.9%. Given the lack of invasive studies, this seems to be a very cost-effective and easy criterion to adopt. It should be noted, however, that this scoring criterion has not been validated in further studies.



Criteria	Point
Unilateral or bilateral buttock pain with fluctuating periods of pain through the day	1
No lower back pain	1
No pain upon palpation of axial spine	1
Negative result for Straight Leg Raise	1
Prolonged sitting triggers gluteal pain or sciatica	1
Fluctuating sciatica through the course of the day	1
Buttock pain near projection of piriformis reproduced by	
FAIR or Freiberg sign	1
Beatty sign	1
Palpation	1
Sciatica reproduced by	
FAIR, Freiberg sign	1
Beatty sign	1
Absence of perineal irradiation	1
Total	12

Scores greater than 8 indicate probable PS. Scores less than 8 indicate unlikely diagnosis and not considered if less than 6.

Table 2

Image Tools

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DIAGNOSTIC STUDIES

There are no current gold standard tests to diagnose PS. Standard radiographs of the pelvis and hip should be considered to rule out underlying hip pathology. Magnetic resonance imaging (MRI) of the lumbar spine also would be recommended to evaluate a discogenic cause for the sciatica. It also may be reasonable to get an MRI of the pelvis to rule out an intrapelvic lesion or mass effect in the sciatic notch ([2,19]).

Neurodiagnostic studies also are done commonly to rule out underlying pathology. These studies are often negative in PS. Fishman et al. ([5]) set out to establish objective EMG findings for PS. Hypothesizing that PS is a functional entrapment, they found a delay in the H-reflex on EMG in the FAIR position. Unfortunately the study did not establish accuracy due to a lack of symptomatic controls, and review of their results did not reach statistical significance ([10]).

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Treatment

Treatment for PS often begins conservatively. Various medications can be used, including nonsteroidal anti-inflammatory drugs (NSAID), muscle relaxants, and other medications for neuropathic pain such as gabapentin. Typically physical therapy will be instituted concomitantly. They utilize piriformis stretching and isometric strengthening as well as modalities for pain control ([17]). There are rare controlled studies on treatment outcomes for PS. Often various modalities are combined in the studies. Fishman et al. ([5]) started all of their EMG-positive patients on a trial of physiotherapy with a Botulinum toxin (Botox) injection. They reported that 79% of their patients had at least 50% improvement with this protocol ([5]). In addition to diagnostic criteria, Michel et al. ([16]) also looked into a treatment strategy for PS. They offered all of their patients with PS medications (muscle relaxants and NSAID) as well as 6 wk of physical therapy. Physiotherapy alone offered a 51.2% cure rate. Although there is a relative lack of high-quality prospective studies for noninvasive treatment of PS, therapy still is considered the mainstay and is cited frequently in the literature ([11]).

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INJECTIONS

Piriformis injections are offered commonly and may be both diagnostic and therapeutic. Injections are either done using anatomic landmarks or imaging. As ultrasonography gains popularity, it is becoming the more common modality, but, also, fluoroscopy still is used and reported in the literature. Other imaging techniques to guide injections include CT and MRI using fluoroscopy technology ([7]). Injections are done with local anesthetic, corticosteroids, a mixture of the two, and, more recently, Botox. Injections often are placed in the muscle belly but also have been directed to the piriformis sheath ([20]).

Some experts feel that given the proximity of the piriformis to the sciatic nerve, inferior gluteal artery, and pelvic cavity, landmark-based injection should not be attempted ([11]). Byrd ([2]) describes a landmark-based injection technique, as follows: placing patients on their side in the FAIR position, the injection is directed to the point of maximal tenderness in the area of the piriformis just medial to the posterior hip joint, next to the sciatic notch. If the patient reports radiating leg pain, he recommends withdrawing, as you are likely too close to the sciatic nerve. Fishman ([5]) also describes a landmark-based approach. With the patient in the FAIR position, the injection is located one-third of the distance from the greater trochanter to the point of maximal tenderness in the buttock and oriented toward the navel to a depth of 1 to 2 inches ([5]).

It is difficult to find studies comparing anatomic landmarks versus other imaging modalities. A cadaver study demonstrated only 30% accuracy for anatomic landmark and intermittent fluoroscopy-assisted injection into the piriformis compared with 95% accuracy with ultrasonography ([4]). There are a variety of ultrasound approaches described in the literature ([4,11,20]). A recent study of 28 patients undergoing ultrasonography and fluoroscopy with nerve stimulation

found no statistically significant changes in pain scores, satisfaction, or functional outcomes for the two ([8]). There are not many outcome studies to demonstrate long-term effectiveness of piriformis injections. Fishman et al. ([5]) had one of the largest number of patients included in his study and did demonstrate that 79% of their patients had at least 50% pain reduction with injection of a corticosteroid and physical therapy.

There are promising findings for Botox reported in the literature, especially for recalcitrant cases ([16,22]). The population studied by Michel et al. ([16]) that did not respond to rehabilitation in 6 wk received up to three injections of onabotulinum toxin A. Of this population, 77% reported pain relief in the “very good” to “good” category ([16]). Another small prospective trial demonstrated improved pain and functioning for Botox type A compared with those for corticosteroid at a 12-wk follow-up for patients not responding to conservative treatment after 3 months ([22]). Another case series of 20 patients receiving Botox type B reported decreased pain, and 75% had good-to-excellent improvement in pain at a 16-wk follow-up ([13]). Most studies of corticosteroids and Botox are small in number and need better controls and randomization to determine an appropriate response.

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SURGERY

Surgery for PS includes tenotomy of the piriformis tendon and sciatic nerve decompression. There are no large, prospective, randomized trials that evaluate surgery for PS ([17]). However several case reports with small cohorts report positive results ([1,2,5]). Most case reports show somewhere around 59% to 69% with good-to-excellent results ([2,17]). Unfortunately many case studies have no validated outcome measures included and lack uniform diagnostic criteria.

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CONCLUSIONS

It is very hard to determine the effectiveness of treatment due to lack of consensus criteria for PS. Some studies include people with back pain, other studies include people with positive results for straight leg raising tests. Systematic reviews would indicate that these are not commonly found criteria ([10]). There are also few well-controlled and randomized studies. Many of the studies are of level 3 to 4 evidence, with insufficient numbers to draw adequate conclusions. Further research is needed.

Until diagnostic criteria can be established and used uniformly, diagnostic tests, studies, and treatments will be verified inefficiently. The diagnostic criterion proposed by Michel et al. ([16]) is inexpensive, noninvasive, and straightforward to use. In addition, it is in line with quality systematic reviews ([10]). Despite the difficulty in diagnosing PS, it would appear that a majority of patients will respond to conservative treatment. For those that do not, corticosteroid and Botox injections can be considered. Surgery may be an option for recalcitrant cases after a sufficient trial of conservative treatment is done.

The author declares no conflicts of interest and does not have any financial disclosures.

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