PIRIFORMIS SYNDROME: A CLINICAL REVIEW.

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ABSTRACT: Piriformis Syndrome is a cause for Low back pain which is most of the times misdiagnosed as it may mimic with various other conditions. Abnormal condition of the Piriformis muscle such as hypertrophy, inflammation, or anatomic variations may lead to this condition. Reported incidence rates for Piriformis Syndrome among patients with low back pain vary widely, from 5% to 36%. Etiology of Piriformis Syndrome is also variable. It can be primary due to anatomical problems or secondary due to various other causes like trauma, local ischemia, limb-length discrepancy etc. Diagnosis of Piriformis Syndrome is complex. History with various clinical tests along with MRI, EMG (Electromyography) and Diagnostic blocks may help to diagnose this condition. Here is a review of Piriformis syndrome for better understanding of the problem so that the diagnosis and management are appropriate.

KEY WORDS: Piriformis Syndrome.

INTRODUCTION: Piriformis Syndrome is often a misdiagnosed cause of Low Back Pain and Sciatica secondary to sciatic nerve entrapment in Piriformis muscle at the greater sciatic notch. Abnormal condition of the Piriformis muscle such as hypertrophy, inflammation, or anatomic variations may lead to this condition. Piriformis Syndrome occurs most frequently during the fourth and fifth decades of life and affects individuals of all occupations and activity levels. Reported incidence rates for Piriformis Syndrome among patients with low back pain vary widely, from 5% to 36%. Piriformis Syndrome is more common in women than men, possibly because of biomechanics associated with the wider quadriceps femoris muscle angle (i.e., "Q angle") in the os coxae (pelvis) of women. Barr’s work correlating clinical features with operative and histological findings, the dominant opinion for decades on the cause of sciatica was nerve root compression by a herniated intervertebral disc. An alternative cause, compression of the nerve trunk by the piriformis muscle, was proposed by Freiberg and Vinke and developed by Robinson, who is credited with coining the term Piriformis Syndrome.

ANATOMY OF THE PIRIFORMIS MUSCLE AND THE SCIATIC NERVE: The Piriformis muscle originates from the anterior surface of the S2–S4 sacral vertebrae, the capsule of the sacroiliac joint, and the gluteal surface of the ileum near the posterior surface of the iliac spine. It runs laterally through the greater sciatic foramen, becomes tendinous, and inserts into the piriformis...
ETIOLOGY: There are two types of Piriformis Syndrome—primary and secondary. Primary Piriformis Syndrome has an anatomic cause, such as a split Piriformis muscle, split sciatic nerve, or an anomalous sciatic nerve path. Secondary Piriformis Syndrome occurs as a result of a precipitating cause, including macrotrauma, microtrauma, ischemic mass effect, and local ischemia. Among patients with piriformis syndrome, fewer than 15% of cases have primary causes.¹⁰

A history of trauma is usually elicited in approximately 50% of the cases: The trauma is usually not dramatic and may occur several months before the initial symptoms. It may occur after total hip replacement surgery or laminectomy.⁷

Trauma to the buttock leads to inflammation and spasm of the muscle. Inflammatory substances such as prostaglandin, histamine, bradykinin, and serotonin are released from the inflamed muscle and may irritate the sciatic nerve resulting in pain–spasm–inflammation–irritation cycle. The stretched, spastic, and inflamed piriformis muscle may compress the sciatic nerve between the muscle and the pelvis, with the compression occurring between the tendinous portion of the muscle and the bony pelvis.¹¹⁻¹³

The real cause of this particular syndrome does not only depend on the relation sciatic nerve-piriformis muscle, because the incidence of the anatomical anomalies of these entities is definitely superior to those treated in the reported cases.¹⁴
DIAGNOSIS AND TESTS: Diagnosis of Piriformis Syndrome is by history, which rules out other differential diagnosis of this condition and clinical tests followed by Imaging and diagnostic block. Isolated involvement of Piriformis is uncommon, and there may be concomitant presence of other cause of Low Back Pain and Leg pain.

SYMPTOMS – Following are the possible symptoms of piriformis syndrome.

- Pain with sitting, standing, or lying longer than 15 to 20 minutes.
- Pain and/or paresthesia radiating from sacrum through gluteal area and down posterior aspect of thigh, usually stopping above knee.
- Pain improves with ambulation and worsens with no movement.
- Pain when rising from seated or squatting position.
- Change of position does not relieve pain completely.
- Contralateral sacroiliac pain.
- Difficulty walking (e.g., antalgic gait, foot drop).
- Numbness in foot.
- Weakness in ipsilateral lower extremity.
- Abdominal, pelvic, and inguinal pain.
- Dyspareunia in women.
- Pain with bowel movements.

SIGNS – Following are the possible clinical signs of Piriformis Syndrome.

- Tenderness in region of sacroiliac joint, greater sciatic notch, and Piriformis muscle.
- Tenderness over Piriformis muscle.
- Palpable mass (? Sausage shaped) in ipsilateral buttock.
- Traction of affected limb provides moderate relief of pain.
- Asymmetrical weakness in affected limb.
- Piriformis sign positive.
- Lasègue sign positive.
- Freiberg sign positive.
- Pace sign (flexion, adduction, and internal rotation Test result) positive.
- Beatty test result positive.
- Limited medial rotation of ipsilateral lower extremity.
- Ipsilateral short leg.
- Gluteal atrophy (chronic cases only).
- Persistent sacral rotation toward contralateral side with compensatory lumbar rotation.

Through compensatory or facilitative mechanisms, Piriformis Syndrome may contribute to cervical, thoracic, and lumbosacral pain, as well as to gastrointestinal disorders and headache. According to Parziale et al the following are the six cardinal features of the syndrome:

- History of trauma to the sacroiliac and gluteal region.
- Pain in the region of the sacroiliac joint, greater sciatic notch, and piriformis muscle, extending down the leg and causing difficulty in walking.
- Acute exacerbation of pain by stooping or lifting and moderately relieved by traction.
- Palpable, sausage-shaped mass over the piriformis muscle, which is tender to palpation.
- Positive Lasègue sign.
- Possible gluteal atrophy.
CLINICAL TESTS - The following physical examination signs help in confirming the presence of piriformis syndrome:

- **Pace sign**: pain and weakness on resisted abduction of the hip while the patient is seated, i.e., the hip is flexed.
- **Laseque sign**: pain on voluntary flexion, adduction, and internal rotation of the hip.
- **Freiberg sign**: pain on forced internal rotation of the extended thigh is due to stretching of the Piriformis muscle and pressure on the sciatic nerve at the sacrospinous ligament.

The diagnosis of piriformis syndrome is made mostly on clinical grounds. Recent publications showed the value of electromyography (EMG), computed tomography (CT), and magnetic resonance imaging (MRI). EMG may detect myopathic and neuropathic changes including a delay in the H-reflex with the affected leg in a flexed, adducted, and internally rotated (FAIR) position as compared with the same H-reflex in the normal anatomic position. A three standard deviation prolongation of the H-reflex has recently been recommended as the physiological criterion for piriformis syndrome. This EMG finding suggests entrapment of the nerve by the hip abductor and external rotator, i.e., the Piriformis muscle, under which it passes. MRI confirms the enlarged Piriformis muscle while CT of the soft tissues of the pelvis may show an enlarged piriformis muscle or abnormal uptake by the muscle.

DIFFERENTIAL DIAGNOSIS: The differential diagnoses of piriformis syndrome include the causes of low back pain and sciatica. In contrast to herniated disc or foraminal stenosis, the patient with Piriformis syndrome usually does not have neurologic deficits. Facet syndrome, sacroiliac joint dysfunction, trochanteric bursitis, myofascial pain syndrome, pelvic tumor, endometriosis, and conditions irritating the sciatic nerve should be considered in the differential diagnoses of piriformis syndrome. These conditions can be ruled out by complete medical history, physical examination and imaging.

TREATMENT: The initial treatments of piriformis syndrome include physical therapy combined with the use of anti-inflammatory drugs, analgesics, and muscle relaxants to reduce inflammation, spasm, and pain.

CONSERVE TIVE TREATMENT: Early conservative treatment is the most effective treatment, as noted by Fishman et al, who reported that more than 79% of patients with piriformis syndrome had symptom reduction with use of nonsteroidal anti-inflammatory drugs (NSAIDs), muscle relaxants, ice, and rest.

The mainstay of treatment, however, is piriformis stretching, which aims to correct the underlying pathology by relaxing a tight piriformis, and related muscle stretching to relieve nerve compression. Because the piriformis lies deep in the gluteus maximus, using moist heat or ultrasound prior to stretching is most often suggested. Stretches can be done in both the standing and supine positions, and they involve hip and knee flexion, hip adduction, and internal rotation of the thigh, as in the flexion, adduction, internal rotation position. This may take some time for patients to tolerate, as this is the same position used to provoke piriformis pain. After stretching, lumbosacral stabilization, hip strengthening exercises, and myofascial release are performed.

Strengthening of the hip abductors is added to the regimen when the symptoms improve. Abnormal biomechanics caused by posture, pelvic obliquities, and leg-length inequalities need to be corrected.
Nonsteroidal anti-inflammatory drugs and acetaminophen have been considered the medications of choice in the management of the many conditions that manifest as low back pain, including piriformis syndrome. Patients using NSAIDs, compared with those using placebo, reported global reduction of symptoms after 1 week of treatment. Muscle relaxants are another frequently prescribed medication for patients with piriformis syndrome. Patients using muscle relaxants are nearly five times as likely to report symptom improvement by day 14, compared with patients given placebo. Common adverse effects of muscle relaxants are dryness of mouth, drowsiness, and dizziness.

Few studies have examined the role of narcotic analgesics in managing acute vs. chronic musculoskeletal pain. However, it is clear that some patients with chronic pain do benefit from these medications. Narcotics can be helpful in controlling episodes of severe or debilitating pain, but they should be considered a short-term treatment only. Constipation, gastrointestinal upset, and sedation are common adverse effects of narcotic medications. In addition, the potential for addiction should always be considered when initiating treatment with medications from this drug class.

INTERVENTIONAL MANAGEMENT: Fishman and colleagues reported a 10-year study on the diagnosis, treatment, and outcome of patients with Piriformis syndrome. Each of their patients received an injection of 1.5 ml of 2% lignocaine and 0.5 ml (20 mg) of triamcinolone acetonide using a 3.5-inch 23–25G spinal needle followed by a standardized physical therapy protocol. Other potential treatments for patients with Piriformis syndrome include prolotherapy (i.e., sclerotherapy, ligament reconstructive therapy), Osteopathic manipulative treatments. Participants improved an average of 71.7%, suggesting the efficacy of corticosteroid and lignocaine injection combined with physical therapy in treating piriformis syndrome, however, there was no control group in their study.

Botulinum Toxin is being used increasingly for the treatment of various musculoskeletal pain conditions. Exact mechanisms are not known, but Botulinum Toxin presumably relieves pain by relaxing painfully spastic muscles. Botulinum Toxin A has Patients who do not respond to the above conservative therapy are candidates for local anesthetic and steroid injections. Surgery may be entertained in recalcitrant cases or when there is documented anatomic abnormality of the piriformis muscle. The muscle may be excised, divided, or thinned.

TECHNIQUE OF PIRIFORMIS MUSCLE INJECTION: Initially, piriformis injections were made blindly. Newer techniques involve identification of the piriformis muscle with a muscle EMG or with the use of CT/Ultrasound guidance. In the technique of Fishman et al. fluoroscopy and EMG are utilized to identify the piriformis muscle. The patient is in the prone position and the expected position of the piriformis muscle is identified using the greater trochanter of the femur and lateral border of the sacrum and the sacroiliac joint as landmarks. Correct needle placement is confirmed with muscle EMG and injection of contrast media. The steroid is then injected into the piriformis muscle. Although successful in identifying the piriformis muscle, the technique utilizes a muscle EMG that is not readily available in most pain management centers.

Another technique is the perisciatric injection of Hanania and Kitain. In their technique the patient is in the lateral or semiprone position with the nondependent hip and knee flexed and the dependent extremity straight. The sciatic nerve is located with a nerve stimulator, the needle is withdrawn a few centimeters, and then 40 mg methylprednisolone in 5 to 10 mL dilute local anesthetic is injected. Fluoroscopy was not utilized in their technique. Hanania and Kitain
described 6 patients who were previously unresponsive or partially responsive to blind 
piriformis muscle injections or epidural steroid injections. Their patients had relief of their pain 
for up to 18 months.

A technique was described wherein the lower border of the sacroiliac joint was used as the landmark.

The patient is prone and the lower border of the sacroiliac joint, greater sciatic foramen, 
and the head of the femur are identified by fluoroscopy. The area is prepared and draped, and 
anesthetized with local anesthetic. A 15 cm insulated needle connected to a nerve stimulator is 
inserted at 1.5 ± 0.8 cm (range: 0.5 to 3 cm) lateral and 1.2 ± 0.6 cm (range: 0.5 to 2 cm) caudal 
to the lower border of the sacroiliac joint. The needle is advanced perpendicularly until a motor 
evoked response of the sciatic nerve is obtained at a depth of 9.2 ± 1.5 cm (range: 7.5 to 13 cm). 
The evoked motor response of the foot can be inversion, eversion, dorsiflexion, or plantar 
flexion. The needle is pulled back 0.3 to 0.6 cm, to avoid intraneural injection, and 40 to 60 mg 
methylprednisolone in 5 to 6 mL saline is injected. The needle is pulled back another 0.5 to 0.7 
cm to place the tip of the needle at the belly of the piriformis muscle. Radiopaque dye (2 to 3 
ML) is injected and the muscle is outlined. Methylprednisolone/Triamcinolone (40 to 60 mg) in 
6 to 8 mL local anesthetic is injected into the muscle.

Botulinum toxin may be injected into the muscle if the patient has transient response to 
the steroid and local anesthetic injection. Botulinum toxin blocks the release of acetylcholine at 
the neuromuscular junction resulting in the prolonged relaxation of the muscle.

When examining the piriformis muscle using ultrasound, the patient should first assume the 
prone position. Placing a pillow or towels between the bed and the patient's inguinal area can 
help increase the pelvic tilt. This allows better visualization of the piriformis muscle via 
ultrasound. A curvilinear transducer is placed in a transverse orientation to first identify the 
sacral cornue and is then moved toward the greater trochanter until the lateral edge of the 
sacrum is observed. The transducer is moved further laterally until the greater trochanter and 
ilium are both observed. The piriformis muscle will appear as a hyperechoic band lying between 
the lateral edge of the sacrum and the greater trochanter and deep in the gluteus maximus 
muscle. The sciatic nerve appears as an oval-shaped hypoechoic structure lying deep in the 
piriformis muscle.22

In a recent study of Manuel Reus et al23 all the patients received US-guided perisciatic 
infiltration satisfactorily. The average time of the procedure was 8 min (range: 5–10 min). Few 
patients felt a slight weakness in the lower limb after perisciatic injection, which disappeared 
spontaneously a few hours later. No local or general complications were observed after 
injection.

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