Piriformis Syndrome: Epidemiology, Clinical features, Diagnosis, and Treatment

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Abstract Piriformis syndrome (PS) is an example of extra-spinal sciatica that is often confused with spinal sciatica (prolapsed lumbar intervertebral disc), which makes diagnosis and treatment delay. The condition is not as rare as we believe; its prevalence is reported to vary between 0.3% and 36% among patients complaining of radiating low back pain. The female has a higher disease predilection; however, men also get the disease significantly. The most common complaint is a deep-seated gluteal pain that gets worse when sitting for a long time; walking usually intensifies the pain, but in chronic PS cases, ambulation may lessen pain. Moreover, Pace sign, FAIR (Flexion- Adduction-Internal Rotation of hip) test, Freiberg test, and Beatty tests are positive. Pain responds partially with analgesics and therapeutic exercise (stretching of piriformis muscle, PM), some may require ultrasound or fluoroscopy-guided steroid and botulinum toxin injections in PM. PS refractory to the above interventions may require surgery. PS is considered a chronic benign condition; however, deep-seated gluteal pain with raised ESR (Erythrocyte Sedimentation Rate), and CRP (C-Reactive Protein) because of piriformis pyomyositis as seen following vaginal delivery is an emergency and should be treated with judicial antibiotics and surgical drainage, where appropriate.

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Key Messages

- Piriformis syndrome is an example of extra-spinal sciatica
- Piriformis syndrome is a disorder of exclusion of its mimics
- The simultaneous presence of Spinal sciatica (prolapsed lumbar intervertebral disc) and extra-spinal sciatica, for example PS, could result in further diagnostic delay
- Piriformis syndrome is generally benign and more often seen in women
- Piriformis pyomyositis or PS due to cervical cancer may mistakenly be diagnosed and treated as piriformis syndrome.

1 Introduction

Piriformis Syndrome (PS) is an example of extra-spinal sciatica and has also been described as 'deep gluteal syndrome', 'pelvic outlet syndrome' or 'pseudo-sciatica'. PS is characterized by localized gluteal and radiating low back pain (LBP) caused by a spasmodic piriformis muscle (PM) and compressed, irritated and stretched sciatic nerve (SN) behind the PM (Siddiq et al. 2017). Physicians may consider it an orthopedic condition, some categorize it as a neurological disorder, whereas others classify it as a soft-tissue rheumatism of the PM (Siddiq et al. 2020). The Greek doctor Hippocrates (460–370 BC) already used the *term* 'sciatica' or ischias (Greek ἰσχιάς)) for pain irradiating from the os ischii down the leg (Stafford et al. 2007). In the nineteenth century, Lasègue described a provocative test for sciatica pain of disc disorders (Stafford et al. 2007). Later, Mixter and Barr described how intervertebral disc prolapse compressing adjacent SN roots generate sciatica-like pain (Mixter and Barr 1934). Pain in lumbago sciatica is the result of an inflammatory response of the SN to prolapsed disc materials, whereas nerve root compression contributes to functional impairment. In some cases, PS may mimic prolapsed lumbar intervertebral disc (PLID), hence being misdiagnosed and wrongly treated without significant relief. The simultaneous presence of lumbago sciatica and extra-spinal sciatica, for example PS, could result in further diagnostic delay (Siddiq et al. 2020). In this chapter, we focus on anatomy of piriformis muscle and nerve, epidemiology, clinical features, differential diagnosis and treatment of PS.

2 Anatomy of Piriformis Muscle and Nerve

The Musculus (M) piriformis is a flat, pear-shaped muscle located in the gluteal region and proximal thigh (Fig. 1). It courses underneath the gluteus maximus, parallel to the posterior margin of the gluteus medius. The PM originates mainly from the anterior surface of the lateral process of the sacrum, the spinal region of the gluteal muscles, and the gluteal surface of the ilium close to the greater sciatic notch, the capsule of sacroiliac joint, and the sacrotuberous ligament (Chang et al. 2022). The muscle passes through the greater sciatic notch dividing it into superior and inferior compartments and inserts on the medial aspect of the superior greater trochanter of the femur. Before inserting into the femur, the PM tendon forms a conjoint tendon with other hip abductors. The sciatic nerve, leaves the pelvis through the greater sciatic notch remaining behind the PM; however, clinically important congenital anomalies of PM have been described in relation to PS (Chang et al. 2022; Boyajian-O'Neil et al. 2008) (Fig. 1a, b). The PM is an external rotator of the hip during the hip extension and abducts the femur during flexion of the hip. The hip abduction while walking shifts body weight and protect us from falling. The PM receives innervation from L5, S1, and S2 nerves and important in keeping PM functional.

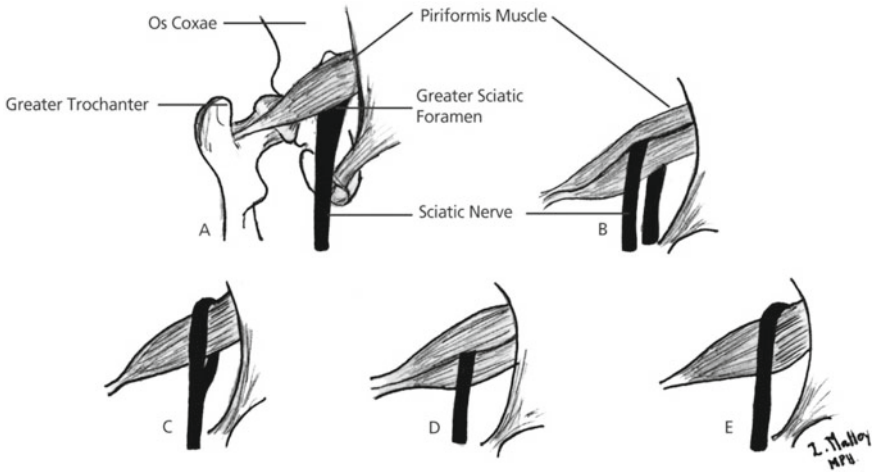

Fig. 1 a Anatomy of hip rotators including piriformis muscle and sciatic nerve in relation to piriformis syndrome (Boyajian-O'Neil et al. 2008; Hopayian et al. 2010); b, variations in relationship of the sciatic nerve to the piriformis muscle (all deep gluteal muscles have been removed, PM has been shown from back): (A) the sciatic nerve exiting the greater sciatic foramen along the inferior surface of the piriformis muscle; the sciatic nerve splitting as it passes through the piriformis muscle with the tibial branch passing (B) Anatomy of hip rotators including piriformis muscle and sciatic nerve in relation to piriformis syndrome

3 Pathophysiology of Piriformis Syndrome

When the PM is overused and/or inflamed, it irritates the adjacent SN with clinical features characteristic of lumbago sciatica. Isolated PM spasm due to chronic poor body posture or some acute injury resulting in a sudden and strong internal rotation of the hip may lead to PS (Hicks et al. 2022). After a fall or direct gluteal blow, a localized hematoma may heal with scar formation in the area between the sciatic nerve and hip extensor, including PM. Sometimes, altered biomechanics of spine and gluteal structures in patients with leg length discrepancy (LLD) may lead to ipsilateral or bilateral PS development (Gerwin 2001).

Extreme use of the PM during long-distance walking, running, repeated squatting, rowing/sculling, kneeling, bicycling, etc. also may result in PS. In some patients PS may be due to myofascial pain syndrome (MPS) involving the PM with taut bands and painful, tender trigger points (TrPs) (Gerwin 2001). Although MPS is a localized painful muscle condition, sometimes it may be widespread due to spreading TrPs from PM to adjacent axial muscle chain of back (Gerwin 2001). Widespread MPS sometimes may get confused with generalized body ache as seen in FMS, an example of central sensitization; sometimes, both conditions may co-exist in the same patient and share a common pathophysiology. MPS patients may have one or more episodes a profound form before they develop FMS (Gerwin 2001). In MPS-PM, an increase in TrPs acetylcholine release at the neuromuscular junction may lead to sustained depolarization of the post-junctional membrane of the muscle fibre producing sarcomere contraction with increased local energy consumption and compromised circulation with resultant hypoxia to PM (Gerwin 2001). The localized muscle ischemia leads to release of substance P that sensitizes PM and afferent nerve nociceptors in the dorsal horn and spinal neurons with pain referral beyond the initial nociceptive region (Siddiq et al. 2017). Thus MPS in PM may contribute to developing widespread body ache (Siddiq et al. 2017; Siddiq et al. 2020; Gerwin 2001).

PS is an example of pseudo-sciatica, thus, before planning an intervention in a patient with sciatica, it is of paramount importance to differentiate between spinal sciatica and extra-spinal pseudo-sciatica (Siddiq et al. 2020). PS in association with lumbar spinal stenosis has also been described, being explained by the "double crush hypothesis" (axons compressed at one site could also get compressed at another site) as explained by Upton and McComas in 1973 (Siddiq and Rasker 2018). Anatomical variations of PM may contribute to PS manifestations (Boyajian-O'Neil et al. 2008) (Fig. 1b).

4 Epidemiology – prevalence and risk factors (occupations, sex distribution, and seasonal variations)

According to various reports, PS may be responsible for between 0.33 and 36% of all causes of low back pain and or sciatica. (Siddiq et al. 2017). It is common in middle-aged people (mean age 38 years) with a predominance among women; however, men also can get the disorder (Siddiq et al. 2017). A wide Q-angle in women may contribute to PS; if this is the case, PS could be bilateral but till date no medical report described bilateral PS in women. In our previous study different occupations have been found to be associated with PS; however, due to the small sample size we could not analyze whether occupation has any significant causal link with PS or whether they differ depending on the gender (Siddiq et al. 2017). Increased PM thickness in PS as measured by high-frequency musculoskeletal ultrasonography (MSUS) has been reported in a cross-sectional analytic study by Siahaan et al. (1.16 ± 0.13 versus 0.85 ± 0.11 cm, respectively for the PS and healthy side, $p < 0.05$) (Siahaan et al. 2021). However, inconsistent clinical features and clinical mimics of PS from surrounding gluteal structures may vary in different geographical areas, cultures, and socioeconomic conditions. As demonstrated in our recent article, the demographic data of PS patients depicted average age at presentation was 43 years, and all patients were within the 21–60 age group (Siddiq et al. 2017). Women had more PS than men (Siddiq et al. 2017). More PS among homemakers and more patients were from rural areas. However, these demographic data may differ if a large study sample is included.

There are *primary and secondary* forms of PS. In primary PS, there is spasm and hypertrophy of the piriformis muscle (PM) without any recognizable cause, sometimes it may be due to anomalous sciatic nerve and PM (Fig. 1b). Secondary PS may happen in patients with lumbar spinal canal stenosis, MPS of PM, long-distance walking, due to occupations (e.g. dancing), myositis ossificans of PM, leg length discrepancy (LLD), etc. (Siddiq et al. 2017; Siddiq et al. 2017). Sometimes, usage of fatty back wallet (walletosis) in men may induce PS (Siddiq et al. 2017).

PS may be seen in women with fibromyalgia (FMS) (Siddiq et al. 2014). PS has also been described in women with iron-deficiency anemia (IDA) and rheumatoid arthritis (RA), improving with blood transfusion and non-steroidal anti-inflammatory drugs (NSAIDs) respectively (Siddiq et al. 2017).

Many patients suggest that the weather may influence their LBP and some associations have been confirmed in several studies Temperature and humidity influence chronic or recurrent self-reported LBP. In a study in England, chronic widespread pain sufferers had most pain in winter, intermediate in autumn and spring, and lowest in summer. However, further research is needed to explore any association between the incidence of acute LBP with precipitation, humidity, wind speed, gust, and direction, and air pressure (Siddiq et al. 2023). PS is a rare type of LBP. The correlation between PS incidence and season changes has never been studied. In a pilot study, we studied whether the pattern of PS, the frequency of PS were associated with PS manifestations over two different seasons (Siddiq et al. 2023). PS was seen more

during dry winter and pre-monsoon, and less in rainy monsoon; however, further research with large number of patients are needed. (Siddiq et al. 2023).

Walletosis, or fat-wallet syndrome, or credit-card neuritis have synonymously but incorrectly been used for deep gluteal PS (Siddiq et al. 2017; Siddiq 2018). In 1966, the first case of wallet neuritis was published. Sometimes, the long-standing use of fatty wallets loaded with unnecessary scraps (even without credit-card), like papers, visiting cards, etc., could result in features resembling SN neuritis (Siddiq et al. 2017). Later, in 1978, Lutz reported two cases of credit-card-wallet sciatica with even a small-sized wallet (Lutz 1978). In our recent case series, we described wallet neuritis in three different professionals (a doctor, a banker, and a day laborer), improving with mere discontinuation of using a buttock wallet, wallectomy! (Siddiq et al. 2017; Siddiq 2018).

Cervical cancer in women may spread to gluteal muscles, including PM resulting in PS, and also radiotherapy for cervical malignancy may cause atrophy of PM inducing or mimicking PS (Siddiq and Rasker 2018). In our recent systematic review, we depicted how purulent PM myositis may develop in women following vaginal delivery and also after abortion, resulting in a medical emergency; these patients usually report deep-seated persistent gluteal pain, fever, and raised inflammatory markers (Siddiq and Rasker 2019). We describe this condition below in more detail.

5 Diagnosis of Piriformis Syndrome

PS is a disorder of diagnostic exclusion of its mimics. Usually, PS presents with localized buttock and deep-seated gluteal pain with variable irradiation and intensity and typical pain aggravating and relieving factors. The pain aggravates with sitting longer, and walking relieves pain, especially in chronic cases (Siddiq et al. 2017). In acute conditions, differentiating PS from PLID is really tough, a situation when PS could be over-diagnosed or underdiagnosed. Immediate clinical differentiation between them can be difficult and MRI of the lumbosacral spine often needed for diagnosis. A positive SLR raising test favors lumbar nerve root compression in PLID; however, in acute PS, patients may also have difficulty in raising the ipsilateral leg and this finding may cause further diagnostic delay. In our recent study, we described PS features in 31 patients (male: female = 1:2) (Siddiq et al. 2017): buttock pain was seen in all patients; pain was aggravated with long-time sitting and affected side-lying. Pain also aggravated during standing from sitting (77.4%), and forward bending (90.3%). Besides, associated tingling feeling according to sciatic nerve distribution was reported in 90.3% cases, 19.35% reported considerable pain improvement while walking. Gluteal tenderness, positive FAIR test, and the Pace sign were elicited in all patients (Siddiq et al. 2017). Palpable gluteal mass and gluteal atrophy was documented in few of them. Straight leg raising test was positive in acute PS cases. High-frequency diagnostic MSUS depicted increased PM thickness on the affected side (Siddiq et al. 2017). There are no diagnostic criteria set for the disorder. In a recent systematic review, Hopayian et al. described the following common features in PS:

buttock pain with external tenderness over the greater sciatic notch, and gluteal pain aggravated through sitting are common. Maneuvers like flexion-adduction-internal rotation (FAIR) test, the Pace sign, the Freiberg test, and digital rectal examination are useful in defining PS (Hopayian et al. 2010). PS is of primary and secondary. In our recent study, we depicted secondary PS and PS-like features in association with preceding fall, rheumatoid arthritis, overuse of PM, lumbar spinal stenosis, FMS, intra-muscular gluteal injection, blunt buttock trauma, leg length discrepancy (LLD), and use of rear pocket's wallet (Siddiq et al. 2017).

Table 1 lists clinical maneuvers that are useful in PS diagnosis. External gluteal tenderness with 'sausage mass' of PM spasm in an area between the greater sciatic notch and the greater trochanter is common (Boyajian-O'Neil et al. 2008). The FAIR test is the most sensitive test and is used widely to diagnose PS (Boyajian-O'Neil et al. 2008). The test is performed in supine position, keeping affected hip flexed at 60° and knee flexed at 90° followed by internal rotation and adduction of the hip joint (Hopayian et al. 2010). In a modified FAIR test, the FAIR test is done in combination with Lasègue's sign, as described by Chen et al. (Chen and Nizar 2013). The Pace sign (resisted abduction and external rotation of the thigh) is reported to be positive in 30–74% (Hopayian et al. 2010). The Pace manoeuver induces gluteal pain on resisted abduction of the flexed hip while sitting (Siddiq et al. 2017; Hopayian et al. 2010). The Freiberg sign (forceful internal rotation of the extended thigh) and the Pace sign are found to be positive, respectively, in 56.2 and 46.5% of the PS patients (Siddiq et al. 2017). The medial end of the PM can be palpated within the pelvis by rectal or vaginal examination, and this test is positive in almost 100% of the patients (Siddiq et al. 2017). Digital rectal examination is the most commonly used internal pain provocation technique for PS, and the finding was considered to be positive provided that patients did jump or changed facial expression during finger gliding over the lateral pelvic wall (Siddiq et al. 2017; Hopayian et al. 2010). The Beatty test is also useful in PS diagnosis.

Besides clinical features, radio-imaging help PS diagnosis. MSUS-guided lidocaine (2%) injection in PM relieving gluteal pain may be both diagnostic and therapeutic (Siddiq et al. 2017). MSUS (PM increased thickness), MRI (hypertrophy of PM), and NCS (nerve conduction study)/electromyogram (EMG) (suggest delayed response when is hip held in FAIR position) are useful in delineating deep-seated gluteal structures and excluding PS mimics (Siddiq et al. 2017; Siahaan et al. 2021). The electrophysiologic approach has been used to diagnose PS by noting the presence of H waves (Siddiq et al. 2017; Hopayian et al. 2010). MRI neurography may show the presence of irritation of the sciatic nerve just adjacent to the sciatic notch; however, it is not readily available in all clinics (Hopayian et al. 2010; Misirlioglu et al. 2018). Sometimes, surgical exploration of the PM may reveal PM calcification, SN impingement between hip abductors, anomalous PM and SN (Hopayian et al. 2010; Foster 2002; Beauchesne and Schutzer 1997).

Table 1 Clinical maneuvers are useful in piriformis syndrome diagnosis (Siddiq et al. 2017; Hopayian et al. 2010)

Name	Description
Lase'gue sign	Localized pain elicited if pressure is applied directly over the piriformis muscle and its tendon, especially with 90° hip flexion and extended knee
Freiberg test	Gluteal pain is experienced during the passive internal rotation of the extended hip
Pace sign	Resisted hip abduction when patients remained seated with flexed knees
Piriformis sign	Gluteal pain with externally rotated ipsilateral foot, a positive piriformis sign
FAIR	Hip flexion, adduction and internal rotation reproduces gluteal pain
Beatty	Patient lying on the asymptomatic side with flexed hip and knee reproduces pain with thigh abduction against gravity
Palpable gluteal mass	Palpable *'sausage-shaped'* mass in the buttock, an evidence of the piriformis muscle spasm
Per-rectal examination	Positive digital rectal examination signifies patients jump or change facial expression during finger gliding over the lateral pelvic wall on digital rectal examination
Diagnostic block	MSUS-guided diagnostic block of the nerve supplying piriformis muscle can be performed at 1.5 cm lateral and 1.2 cm caudal to the lower 1/3rd of the sacroiliac joint with 5 ml of 1% lignocaine injection. If pain is reduced, then the test is regarded positive

FAIR flexion, abduction and internal rotation of the hip

6 Differential Diagnosis

PS is a disorder of exclusion. Sometimes, PS diagnosis is not straightforward, and it may become a diagnostic difficulty. In a recent scoping review article, we described conditions mimicking PS (Table 2) (Siddiq et al. 2020).

7 Piriformis Syndrome-Like Presentation in Emergency

PS is not always benign. Sometimes, an infected PM leads to pyomyositis and deserves special attention. Skeletal pyomyositis follows three distinct phases—invasive stage (myositis without abscess formation), suppurative stage (myositis with abscess formation), and late stage with sepsis (consistent fever, septicemia, toxic and/or coma) that requires intensive management). Staphylococcus aureus is the most common pathogen; however, Group A, as well as Group β Streptococcus, Salmonella typhi, Proteus mirabilis, Brucella melitensis, and *Escherichia coli*, also contribute to the pathogenesis (Siddiq 2018).

Table 2 Conditions mimicking piriformis syndrome (Siddiq et al. 2017; 2020; Hopayian et al. 2010)

Clinical conditions	Description
Wallet neuritis	Walletosis or Fat-wallet neuritis can mimics PS features. Discontinuation of wallet could improve the condition
Piriformis pyomyositis	Persistent deep-seated gluteal pain with fever, raised inflammatory markers positive PS maneuvers, MRI and MSUS-based space occupying lesion in PM is suggestive of piriformis pyomyositis. Common pathogens are—*Staphylococcus aureus, Group A Streptococci, Escherichia coli, Proteus Mirabilis, and Brucella Melitensis*
Superior cluneal nerve (SCN) disorder	LBP and leg pain are aggravated by standing, walking, bending from the waist, twisting, stairs up-down, and lifting weights. Tenderness over the posterior superior iliac crest reproduces the pain. In older people, SCN may be associated with a dorso-lumbar vertebral fracture
Quadratus lumborum (QL) and Gluteal medius myofascial pain syndrome (MPS)	QL trigger points (TPs) refer to pain in the SI joint, lower buttock and lateral hip regions and have the potential to generate distant TPs in gluteus muscles. Gluteal medius tendinitis (GMT) may also present with gluteal pain with pain referral to the thigh back, limited range-of-motion of the hip joint, fever, and raised inflammatory biomarkers that could mimic PS
Osteitis condensans illi (OCI) and SpA	OCI is seen in young women of childbearing age. LBP with pain referral to the thigh back is the presenting complaint. Pain aggravates with sitting and forward bending; however, lying relieves pain. X-ray sacroiliac joint reveals radio-opaque sclerotic change involving the ilial part of the SI joint, though sparing the SI joint. In SpA, inflammatory spinal pain is the presenting feature. Patients may be positive for HLA-B27 with high inflammatory biomarkers
Catamenial sciatica	According to the sciatic nerve distribution, radiating gluteal pain appears cyclically with menstruation and is sometimes associated with lower limb weakness, muscle wasting, reduced jerks, and impaired sensation. Advanced imaging with MRI and CT scan of the lumbosacral spine/pelvis and ultrasonogram of the whole abdomen help diagnose the condition. NCV/EMG demonstrates nerve damage
Post-injection sciatica	Sciatic nerve (SN) injury with sciatica-like features mimicking PS may develop following IM gluteal injection (more with NSAID, penicillin G, and diazepam). Clinical manifestations include sensory disturbance and foot drop with deformity (equinovarus, calcaneocavus or equinus)

(continued)

Table 2 (continued)

Clinical conditions	Description
Sciatic nerve sheath tumor	SN sheath tumor, for example, schwannoma, may present with sciatica-like features. MRI, US scanning of SN, and histopathology findings may help diagnose the lesion and exclude the differential diagnosis
Compressive neuropathy	The lateral cutaneous nerve of the thigh (LCNT), saphenous, sural, and common peroneal nerve compression could mimic lumbago. Lipoma, schwannoma, and ganglion cysts could compress SN at different levels. MSUS and MRI help diagnose them

FAIR flexion-adduction-internal rotation, *PR* per-rectal, *IL* intra-lesional, *NSAID* non-steroidal anti-inflammatory drug, PM: piriformis muscle, *SN* sciatic nerve, *MRI* magnetic resonance imaging, *MSUS* musculoskeletal ultrasound, *LBP* low back pain, *SI* sacroiliac, *CT* computed tomography, PRP: platelet rich plasma, SpA: spondyloarthropathy, *NCV* nerve conduction velocity, *EMG* electromyogram, *IM* intramuscular

Piriformis pyomyositis can develop in women after abortion and following vaginal, forceps, and ventouse delivery. Patients might get PM infection through transient bacteremia from intravenous cannula-associated cellulitis or seeding of infectious agents into PM might happen through the torn vaginal structures. Patients may have bacterial growth (Group β-streptococcus) in blood culture, some may have high vaginal swab growth of *Escherichia coli*, moderate growth of Group βstreptococcus, and mixed anaerobes. Besides, mixed growth could be found in mid-stream urine. Sometimes, an MRI of the pelvis revealed a high signal intensity signifies edematous and swollen PM compatible with purulent myositis with deep-seated gluteal pain and sciatica-like complaints. Purulent PS in type 2 diabetic women has also been reported (Siddiq 2018).

Piriformis pyomyositis in association with septic arthritis of the sacroiliac joint has been reported in children: patients reported high-grade fever, pin-point gluteal pain, and MRI-depicted space-occupying lesion involving greater sciatic notch and lumbar nerve roots. In adolescents hip pain due to piriformis pyomyositis may be reported at the emergency with positive SLR, fever with rigor, and sweating; later pelvic MRI T1-weighted imaging could reveal abnormally high signal intensity in PM, gadolinium administration could reveal the widespread extension of the lesion to soft tissue. Besides, the agglutination test could yield high titre for Brucella melitensis, and blood culture yields Brucella melitensis growth (Siddiq 2018).

Piriformis pyomyositis in men with gluteal pain and positive blood and or pus culture for Salmonella typhi. Apparently healthy tennis and rugby players could present with gluteal pain and MRI could depict fluid accumulation in the left iliopsoas, sacroiliac joint, and an SOL within the PM, patients had raised inflammatory biomarkers and blood cultures were positive for Staphylococcus aureus. In competitive swimmers, Proteus mirabilis-induced piriformis pyomyositis has also been reported. MRI/CT abdomen and pelvis revealed PM swelling. Patients also had raised ESR, CRP, and leukocytosis. Piriformis pyomyositis complicated with an

extensive epidural abscess had also been reported, and culture from pus could yield methicillin-sensitive Staphylococcus aureus (Siddiq 2018).

Antibiotic treatment is the mainstay of piriformis pyomyositis treatment. Besides, patients may require NSAIDs, analgesics, and sedatives. The duration of antibiotic treatment may vary, and depends on the culprit organism types; however, initial intravenous regimen followed by oral therapy for a total of 3–8 weeks is well practiced. No intra-lesional (IL) corticosteroid or botox should be given. CT-guided aspiration followed by culture and sensitivity (C/S) test helps to confirm the diagnosis. Sometimes, no causative agent could be found. Start treatment with broad-spectrum antibiotic, then change it according to pus and blood C/S report. Empirical oral antibiotic therapy for example, oral Cefdinir (10 mg/kg/day) is found effective in pediatric patients. Empirical intravenous cloxacillin (1000 mg 8-hourly) or augmentin (1.2 g every 8 hours) or cefuroxime (750 mg, 3 times/day) followed by their oral formulations also improved pyomyositis in staphylococcus aureus sensitive cases. IV cloxacillin (2 weeks) and amikacin, oral cloxacillin are effective in methicilin-resistant Staphylococcus aureus. Intravenous vancomycin and meropenem are effective where cloxacillin becomes failed in Staphylococcus aureus-positive cases (Siddiq 2018).

Brucellosis also contributes in purulent piriformis myositis and combined doxycycline (100 mg 2 times/day), rifampin 900 mg (once/day), and ciprofloxacin (500 mg 2 times/day) regime relieves from brucellosis myositis. Intravenous vancomycin—15 mg/kg/12-hourly), followed by oral therapy (160 mg trimethoprim/800 mg sulfamethoxazole every 12 h), benzylpenicillin and clindamycin secures overall improvement in Group β-streptococcus and *E. coli* sensitive pyomyositis. Cefotaxime and tobramycin are found effective in Proteus Mirabilis (Siddiq 2018).

8 Treatment of Piriformis Syndrome

Short-term rest can relieve symptoms. Besides, soft tissue mobilizing can be beneficial. Non-steroidal anti-inflammatory drugs (NSAIDs) such as ibuprofen may alleviate the symptoms and provide short-term pain relief. Neuropathic agents such as gabapentin and pregabalin can be adjuncts to NSAIDs. The role of vitamin B preparations in PS treatment is inconclusive. Traditional stretching of gluteal musculatures and pulsed radiofrequency help some patients. Acupuncture shows some promise in the disorder. In some patients, MSUS-guided dry needling may be useful (Vij et al. 2021; Payne 2016).

MSUS-guided injection of lidocaine (2%) and betamethasone in the PM may produce statistically significant pain relief though for short-term periods, and repeated injection may be required (Payne 2016). There is no statistically significant difference between fluoroscopy and ultrasound-guided PM interventions. Some patients may find SN peri-neural corticosteroid injection effective. Injection of botulinum toxin type A (100–200 unit of BoNT-A) inhibiting the release of substance

P at the NMJ of PM could be useful in PS; however, Botox should be preserved for those who do not improve with other interventions (Vij et al. 2021; Fritz et al. 2014).

Surgical management of PS involves dissection of PM and decompression of the SN and can be done open or endoscopically; however, the endoscopic approach has some superiority in terms of improved visualization of PM muscle, less soft-tissue damage and postoperative pain, and reduced recovery times (Vij et al. 2021; Kay et al. 2017).

In summary, piriformis syndrome is an example of extra-spinal sciatica and a disorder of exclusion of many causes of low back pain. A logical diagnostic approach and imaging may make diagnosis of piriformis syndrome possible and should be followed to exclude PS mimics clinically. Pain killers may cause temporary pain relief; however, some patients need guided intervention, and some may benefit from surgery. Purulent piriformis myositis with PS-like deep-seated gluteal pain develop following vaginal delivery with fever, persistent gluteal pain, and raised inflammatory markers could be misdiagnosed as PS and corticosteroid injection in piriformis muscle may cause harm, while these patients need antibiotic therapy. So, PS is not always benign, sometimes it may even be an emergency.

References

Beauchesne RP, Schutzer SF. Myositis ossificans of the piriformis muscle: an unusual cause of piriformis syndrome. A case report. J Bone Joint Surg. 1997;79:906–910.

Boyajian-O'Neil LA, McClain RL, Coleman MK, Thomas PP. Diagnosis and management of piriformis syndrome: an osteopathic approach. J Am Osteopath Assoc. 2008;108:657–64.

Chang C, Jeno SH, Varacallo M. Anatomy, Bony Pelvis and lower limb: piriformis muscle. Updated 2022 Oct 3. In: StatPearls (Internet). Treasure Island (FL): StatPearls Publishing; 2023. Available from: https://www.ncbi.nlm.nih.gov/books/NBK519497/

Chen CK, Nizar AJ. Prevalence of piriformis syndrome in chronic low back pain patients. A clinical diagnosis with modified FAIR test. Pain Pract. 2013;13:276–281 J 19:2095–2101.

Foster MR. Piriformis syndrome. Orthopedics. 2002;25:821–5.

Fritz J, Chhabra A, Wang KC, Carrino JA. Magnetic resonance neurography-guided nerve blocks for the diagnosis and treatment of chronic pelvic pain syndrome. Neuroimaging Clin N Am. 2014;24(1):211–34. https://doi.org/10.1016/j.nic.2013.03.028.

Gerwin RD. Classification, epidemiology, and natural history of myofascial pain syndrome. Curr Pain Headache Rep. 2001;5:412–20.

Hicks BL, Lam JC, Varacallo M. Piriformis syndrome. Updated 2022 Sep 4. In: StatPearls (Internet). Treasure Island (FL): StatPearls Publishing; 2023. Available from: https://www.ncbi.nlm.nih.gov/books/NBK448172/

Hopayian K, Song F, Riera R, Sambandan S. The clinical features of the piriformis syndrome: a systemic review. Eur Spine J. 2010;19:2095–101.

Kay J, de Sa D, Morrison L, Fejtek E, Simunovic N, Martin HD, et al. Surgical management of deep gluteal syndrome causing sciatic nerve entrapment: a systematic review. Arthroscopy. 2017;33(12):2263–2278 e1. https://doi.org/10.1016/j.arthro.2017.06.041.

Lutz EG. Credit-card-wallet sciatica. JAMA. 1978;240(8):738 PMID: 671698.

Misirlioglu TO, Palamar D, Akgun K. Letter to the editor involving the article 'piriformis muscle syndrome: a cross-sectional imaging study in 116 patients and evaluation of therapeutic outcome.' Eur Radiol. 2018;28(12):5354–5.

Mixter WJ, Barr JS. Rupture of the intervertebral disc with involvement of the spinal canal. N Engl J Med. 1934;211:210–5.
Payne JM. Ultrasound-Guided hip procedures. Phys Med Rehabil Clin N Am. 2016;27(3):607–29. https://doi.org/10.1016/j.pmr.2016.04.004.
Siahaan YMT, Tiffani P, Tanasia A. Ultrasound-Guided measurement of piriformis muscle thickness to diagnose piriformis syndrome. Front Neurol. 2021;7(12):721966. https://doi.org/10.3389/fneur.2021.721966.
Siddiq MAB. Piriformis syndrome and wallet neuritis: are they the same? Cureus. 2018;10(5):e2606. https://doi.org/10.7759/cureus.2606.
Siddiq AB, Rasker JJ. Piriformis syndrome: still unsolved issues. Int J Clin Rheumatol. 2018;13(6):338–40.
Siddiq MAB, Rasker JJ. Piriformis pyomyositis, a cause of piriformis syndrome-a systematic search and review. Clin Rheumatol. 2019;38(7):1811–21. https://doi.org/10.1007/s10067-019-04552-y.(12).
Siddiq MA, Khasru MR, Rasker JJ. Piriformis syndrome in fibromyalgia: clinical diagnosis and successful treatment. Case Rep Rheumatol. 2014;2014:893836. https://doi.org/10.1155/2014/893836.
Siddiq MA, Hossain MS, Uddin MM, Jahan I, Khasru MR, Haider NM, Rasker JJ. Piriformis syndrome: a case series of 31 Bangladeshi people with literature review. Eur J Orthop Surg Traumatol. 2017;27(2):193–203. https://doi.org/10.1007/s00590-016-1853-0.
Siddiq MAB, Clegg D, Hasan SA, Rasker JJ. Extra-spinal sciatica and sciatica mimics: a scoping review. Korean J Pain. 2020;33(4):305–17. https://doi.org/10.3344/kjp.2020.33.4.305.
Siddiq MAB, Hossain MS, Khan AUA, Sayed MA, Rasker JJ. Piriformis syndrome in pre-monsoon, monsoon, and winter: an observational pilot study. Cureus. 2023;15(2):e35296. https://doi.org/10.7759/cureus.35296.
Siddiq AB, Jahan I, Alpha M. Wallet neuritis—an example of peripheral sensitization. Curr Rheum Rev. 2017;13(999). https://doi.org/10.2174/1573397113666170310100851.
Stafford MA, Peng P, Hill DA. Sciatica: a review of history, epidemiology, pathogenesis, and the role of epidural steroid injection in management. Br J Anaesth. 2007;99:461–73.
Vij N, Kiernan H, Bisht R, Singleton I, Cornett EM, Kaye AD, Imani F, Varrassi G, Pourbahri M, Viswanath O, Urits I. Surgical and non-surgical treatment options for piriformis syndrome: a literature review. Anesth Pain Med. 2021;11(1):e112825. https://doi.org/10.5812/aapm.112825.