■ SYSTEMATIC REVIEW

Deep gluteal syndrome as a cause of posterior hip pain and sciatica-like pain

J. W. Park,
Y-K. Lee,
Y. J. Lee,
S. Shin,
Y. Kang,
K-H. Koo

From Seoul National University Bundang Hospital, Seongnam, South Korea

Deep gluteal syndrome is an increasingly recognized disease entity, caused by compression of the sciatic or pudendal nerve due to non-discogenic pelvic lesions. It includes the piriformis syndrome, the gemelli-obturator internus syndrome, the ischiofemoral impingement syndrome, and the proximal hamstring syndrome. The concept of the deep gluteal syndrome extends our understanding of posterior hip pain due to nerve entrapment beyond the traditional model of the piriformis syndrome. Nevertheless, there has been terminological confusion and the deep gluteal syndrome has often been undiagnosed or mistaken for other conditions. Careful history-taking, a physical examination including provocation tests, an electrodiagnostic study, and imaging are necessary for an accurate diagnosis.

After excluding spinal lesions, MRI scans of the pelvis are helpful in diagnosing deep gluteal syndrome and identifying pathological conditions entrapping the nerves. It can be conservatively treated with multidisciplinary treatment including rest, the avoidance of provoking activities, medication, injections, and physiotherapy.

Endoscopic or open surgical decompression is recommended in patients with persistent or recurrent symptoms after conservative treatment or in those who may have masses compressing the sciatic nerve.

Many physicians remain unfamiliar with this syndrome and there is a lack of relevant literature. This comprehensive review aims to provide the latest information about the epidemiology, aetiology, pathology, clinical features, diagnosis, and treatment.

Cite this article: ***Bone Joint J*** **2020;102-B(5):556–567.**

Introduction

Although posterior hip pain may be less common than anterior or lateral hip pain, it has become increasingly recognized because of improvements in our understanding of the anatomy and neural kinematics of hip movement. Lumbar spinal pathology can often present as lateral or posterior hip pain as the capsule of the hip is innervated by sensory nerves from the L2-S1 roots.[1] However, in a series of 158 patients with sciatica and disabling back pain, el Barzouhi et al[2] reported that 50 (32%) did not have corresponding lumbar pathology on MRI. Posterior hip pain or non-discogenic sciatica (sciatica-like pain) can also be caused by any lesion involved the sciatic nerve, originating from L4 to S3.[3] Posterior hip pain is therefore often difficult to distinguish from sciatica.

Approximately 90 years ago, it was reported that sciatica might result from sacroiliitis due to inflammation within the piriformis muscle and consequent irritation of the sciatic nerve.[4,5] A cadaver study subsequently led to the hypothesis that piriformis spasms could lead to irritation of the nerve.[6] Robinson[7] introduced the term "piriformis syndrome" to describe non-discogenic sciatica secondary to an abnormal condition of the piriformis, in 1947. This syndrome has also been referred to as "credit-card-wallet sciatica", "pseudosciatica", or "pelvic outlet syndrome".[3,8,9] However, it has remained controversial as a distinct clinicopathological entity because there is no clear-cut evidence of damage to the sciatic nerve in many patients with non-discogenic buttock pain, and consequent disagreement over whether it is under- or overdiagnosed.[10-12]

In 1999, McCrory and Bell[13] proposed that the term "deep gluteal syndrome" should replace that of the piriformis syndrome, on the basis that posterior hip pain could result from entrapment of the sciatic or other nerves by various structures in the deep gluteal space. Recently, the gemelli-obturator internus syndrome, ischiofemoral impingement syndrome, and proximal hamstring syndrome have been recognized as producing sciatica-like pain and have been included in the concept of the deep gluteal syndrome.[14-16] As described by McCrory and Bell, the piriformis syndrome is only one component of the deep gluteal syndrome, and a broad spectrum of pelvic conditions not associated with piriformis can cause similar symptoms.[17,18]

Correspondence should be sent to Y. J. Lee; email: yn35@snu.ac.kr

doi:10.1302/0301-620X.102B5.BJJ-2019-1212.R1 $2.00

Bone Joint J 2020;102-B(5):556–567.

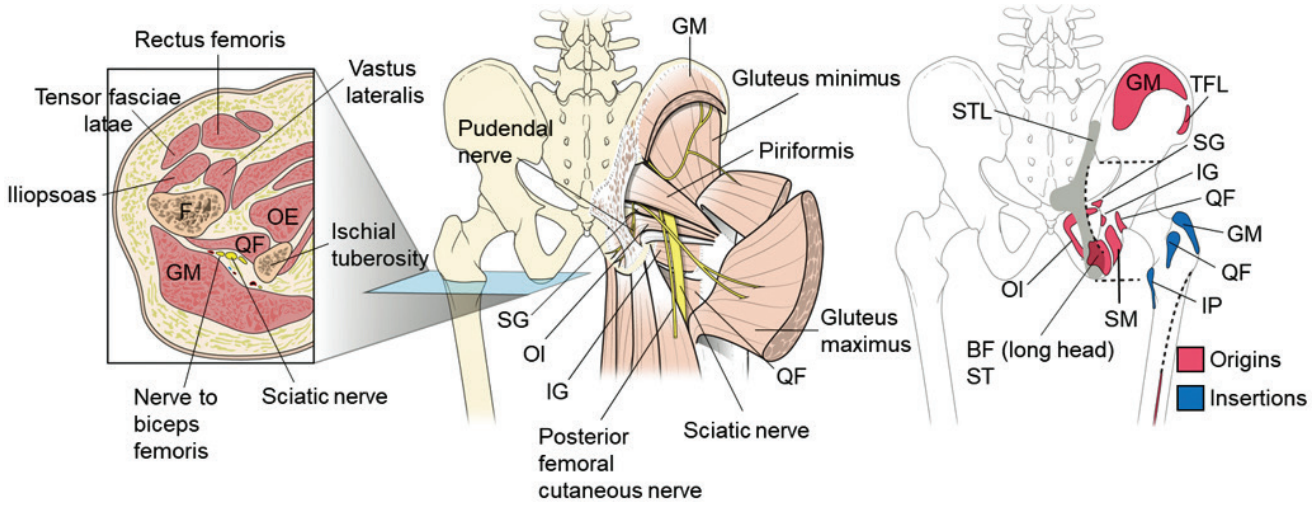

Fig. 1

Schematic illustration of the anatomy of the deep gluteal space. BF, biceps femoris; F, femur; GM, gluteus medius; IG, inferior gemellus; IP, iliopsoas; OE, obturator externus; OI, obturator internus; QF, quadratus femoris; SG, superior gemellus; SM, semimembranosus; ST, semitendinosus; STL, sacrotuberous ligament; TFL, tensor fasciae latae.

Nevertheless, there has been terminological confusion: some authors have used deep gluteal syndrome as a synonym of piriformis syndrome.[8,19]

The concept of the deep gluteal syndrome extends our understanding of posterior hip pain due to nerve entrapment beyond the traditional model of piriformis syndrome. Its clinical presentation includes entrapment of the sciatic nerve producing posterior hip pain, and entrapment of the pudendal nerve causing pain in the perineal, perianal, and genital areas. The deep gluteal syndrome is often undiagnosed or mistaken for other conditions with similar symptoms because it has no definitive diagnostic criteria. In this review, we differentiate the deep gluteal syndrome with sciatic nerve entrapment from the piriformis syndrome, gemelli-obturator internus syndrome, the ischiofemoral impingement, and the proximal hamstring syndrome and review the nature of the condition, the diagnostic approaches, and treatment of each.

Epidemiological considerations. Previously, several reviews described the piriformis syndrome as having an estimated prevalence of 5% to 8%.[20,21] However, the reported prevalence has varied according to the definitions, study designs, and cohorts which were included. In a series of 93 patients with chronic low back pain, piriformis syndrome was prospectively diagnosed in 16 (17.2%).[22] In 143 patients with sciatica without evidence of lumbar pathology, 24 (17.0%) were classified as having piriformis syndrome, and in 783 patients who underwent MRI of the hip, 74 (9.5%) had piriformis syndrome.[23,24]

To date, there has been little epidemiological data regarding other subtypes of deep gluteal syndrome: gemelli-obturator internus syndrome, ischiofemoral impingement, and proximal hamstring syndrome. Filler et al[25] re-evaluated 239 patients with sciatica but no diagnosis using MRI and reported a final diagnosis of piriformis syndrome in 67.8% of patients, proximal hamstring syndrome in 4.7%, pudendal nerve entrapment in 3.0%, a sciatic tumour in 1.7%, and sacroiliitis with fracture in 1.2%. During endoscopic decompression, only three of 35 patients with deep gluteal syndrome presented with involvement of the obturator internus, and two had pathological changes in the hamstring tendon insertion.[26] Furthermore, in a recent systematic review including 481 patients with deep gluteal syndrome undergoing surgical treatment, the diagnosis was piriformis syndrome in 124 (26%), sciatic nerve entrapment by non-piriformis muscles in 67 (14%), endometriosis in 27 (6%), and compression by the inferior gluteal vein in eight (2%).[16] Thus, the piriformis syndrome may be much more prevalent than that of other subtypes of the deep gluteal syndrome. Ischiofemoral impingement, and particularly the proximal hamstring syndrome, are considered uncommon. In a study from a single institution, there were 30 suspected cases of ischiofemoral impingement among more than 1,000 hip operations and this diagnosis was eventually confirmed in 17.[27] The proximal hamstring syndrome mainly develops in patients who undertake competitive sports and has usually been reported in case reports and series. In a retrospective study involving 162 patients, sciatic nerve-related symptoms were noted in 45 patients (28%) with a history of proximal hamstring avulsion.[28] Thus, sciatic nerve entrapment after proximal hamstring injury may be under-recognized.

Anatomical considerations. The deep gluteal space is bordered by: posteriorly, the gluteus maximus; anteriorly, the posterior acetabular column, hip joint capsule and proximal femur; laterally, the lateral lip of the linea aspera and the gluteal tuberosity; medially, the sacrotuberous ligament and falciform fascia; superiorly, the inferior margin of the sciatic notch; and inferiorly, the proximal origin of the hamstrings at the ischial tuberosity.[29] The piriformis, superior gemellus, obturator internus, inferior gemellus, and quadratus femoris are located in this space, from caudal to distal.[14] The superior and inferior gluteal, sciatic, posterior femoral cutaneous, and pudendal nerves traverse the deep gluteal space (Figure 1).[14]

Table I. Clinical tests for the diagnosis of the deep gluteal syndrome.

Clinical tests	Clinical subtype	Mean prevalence, % (95% CI)	Mean sensitivity (95% CI)	Mean specificity (95% CI)	Reference
Lasègue sign	PFS	41 (33 to 50)	N/A	N/A	[73]
	DGS	N/A	0.15 (0.05 to 0.33)	0.95 (0.68 to 1.00)	[74]
Freiberg's sign	PFS	34 (26 to 42)	N/A	N/A	[73]
Pace's sign	PFS	31 (23 to 39)	N/A	N/A	[73]
Beatty sign	PFS	11 (5 to 17)	N/A	N/A	[73]
Flexion, adduction, and internal rotation (FAIR)	PFS	33 (22 to 43)	N/A	N/A	[73]
Active piriformis test	DGS	N/A	0.78 (0.58 to 0.90)	0.80 (0.49 to 0.94)	[74]
Seated piriformis stretching	DGS	N/A	0.52 (0.33 to 0.71)	0.90 (0.60 to 0.98)	[74]
Active piriformis test or seated piriformis stretching	DGS	N/A	0.91 (0.73 to 0.98)	0.80 (0.49 to 0.94)	[74]
Ischiofemoral impingement test	IFI	N/A	0.82 (0.56 to 0.95)	0.85 (0.54 to 0.97)	[64]
Long-stride walking test	IFI	N/A	0.94 (0.69 to 0.99)	0.85 (0.54 to 0.97)	[64]
Ischiofemoral impingement and long-stride walking test	IFI	N/A	0.76 (0.50 to 0.93)	0.85 (0.54 to 0.98)	[64]
Active hamstring test at 30° and 90°	PHS	N/A	0.84 (0.66 to 0.93)	0.97 (0.76 to 0.99)	[75]
Long stride heel strike test	PHS	N/A	0.55 (0.37 to 0.72)	0.73 (0.48 to 0.89)	[75]

CI, confidence interval; DGS, deep gluteal syndrome; IFI, Ischiofemoral impingement; N/A, not available; PFS, piriformis syndrome; PHS, proximal hamstring syndrome.

The kinematic behaviour of the sciatic nerve plays a role in the pathophysiology of the deep gluteal syndrome. The tension in the nerve is affected by the position of the hip and knee joints.[26] It is stretched by an excursion of 28.0 mm during a modified straight leg raise with knee extension.[18] However, the flexed, abducted, and externally rotated position of the hip can limit its excursion.[19] When the hip is flexed to 90° and the knee is fully extended, sciatic strain increases by a mean of 26%, which may result in neural dysfunction.[30] Thus, impaired gliding of the sciatic nerve can disrupt its normal excursion and induce the deep gluteal syndrome.

The piriformis syndrome. The piriformis lies centrally in the buttock and is a key reference for identifying the neurovascular structures in the deep gluteal space. Caudal to it are the superior gluteal artery and nerve, and distal to it are the inferior gluteal artery/nerve, the posterior femoral cutaneous nerve, the sciatic nerve, the obturator internus/gemellus superior nerves, and quadratus femoris/gemellus inferior nerves.[15] A total of 13 anatomical variations in the relationship between the sciatic nerve and piriformis have been described.[31-33] The nerve passes beneath piriformis in 85.2% of individuals.[34] Its peroneal portion pierces the piriformis while the tibial component passes distal to it in 9.8%; this most common variant is more prevalent in the Asian population (17.0%) than in other ethnicities.[34] It has been suggested that anomalous variants may pose a risk for the development of the deep gluteal syndrome but the association remains unproven.[32]

Foster[35] classified the piriformis syndrome into a primary condition, caused by intrinsic problems within the muscle, and a secondary condition, caused by irritation from the sacroiliac joint or an adjacent mass. In two studies involving patients with secondary piriformis syndrome, between 7/24 (29.2%) and 50/62 (80.6%) had benign or malignant space-occupying lesions in the deep gluteal area.[23,36] Intrinsic pathologies of the piriformis which cause the primary syndrome include an anomalous variation of the relationship between the nerve and piriformis, piriformis hypertrophy, tumours or inflammatory and infectious lesions, and myofascial pain syndrome.[8] Piriformis syndrome may occur after overuse or repetitive trauma to the gluteal area, and many patients with post-traumatic entrapment of the sciatic nerve have constricting fibrous or fibrovascular bands from the piriformis over the nerve.[7,15,37] Thus, since the concept of deep gluteal syndrome was introduced, Foster's dichotomic classification has had little clinical relevance.

The gemelli-obturator internus syndrome. After passing the piriformis, the sciatic nerve runs posterior to the obturator/gemelli complex and quadratus femoris muscle.[14] The insertion of piriformis is variable: prior to its insertion onto the greater trochanter the piriformis may join the tendon of the superior gemellus and obturator internus (29.5%) or the tendon of obturator internus and gluteus medius (13.4%).[38] In these variants, impingement of piriformis on the sciatic nerve can cause sciatica-like symptoms, especially during internal rotation of the hip. The nerve is attached to the gemelli-obturator internus complex by connective tissue, which can lead to sciatic entrapment.[39,40] Muscular spasm or myofascial pain syndrome, acute strain, haematoma, abscess/pyomyositis, tendinitis, and bursitis of the obturator internus can cause posterior hip pain.[40–45]

The ischiofemoral impingement syndrome. The sciatic nerve passes between the ischial tuberosity and the lesser trochanter, lying close to the posterior capsule of the hip and enters the posterior thigh at the lower margin of quadratus femoris.[14] The

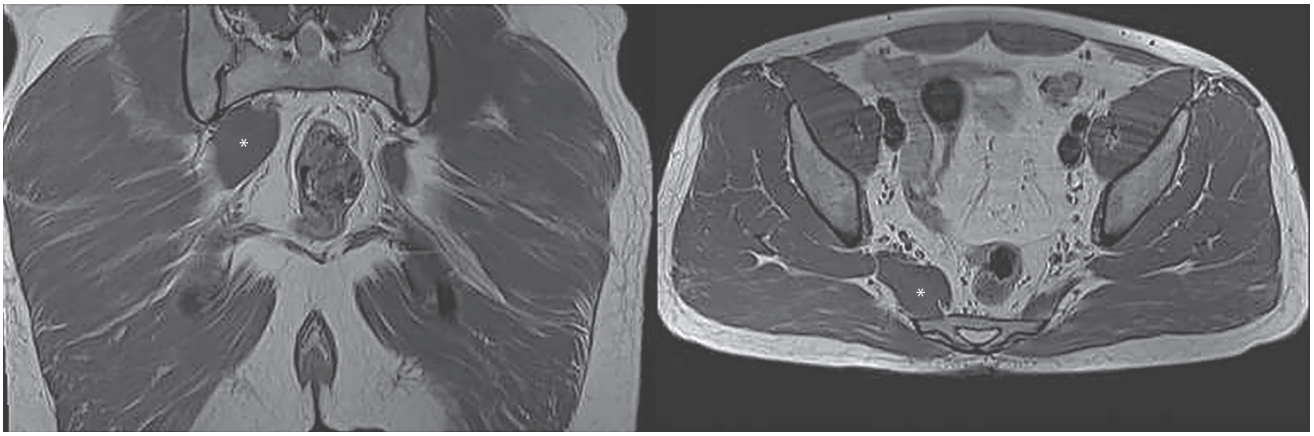

Fig. 2

Piriformis syndrome (37-year-old male). Asymmetrically large right piriformis muscle (white asterisks) was shown on T1-weighted coronal and axial images of MRI.

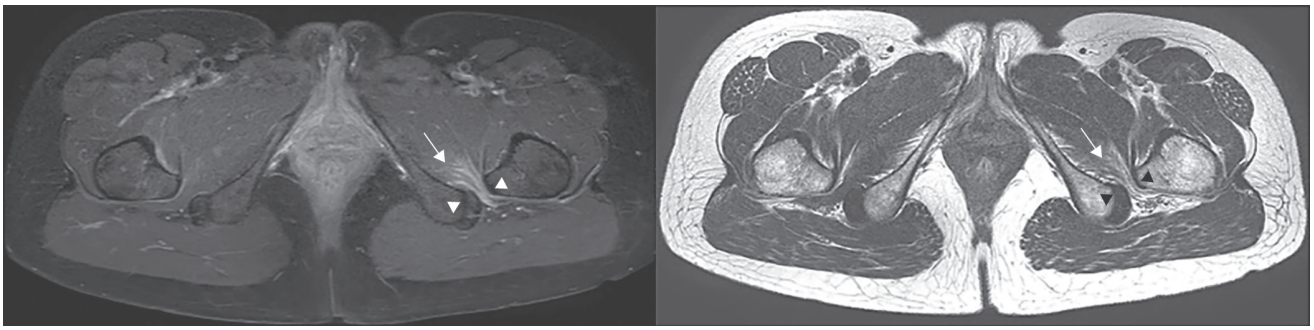

Fig. 3

Ischiofemoral impingement syndrome (46-year-old female). Increased signal intensity and contrast enhancement were noted in quadratus femoris at the left ischiofemoral interval (white arrows) on T2 and T1 weighted axial MRI images. White and black triangles indicate the ischium and lesser trochanter.

ischiofemoral impingement syndrome is related to the proximity of the sciatic nerve to quadratus femoris. It was first noted in patients with persistent pain in the medial thigh and groin following total hip arthroplasty, and was thought to be due to lesser trochanteric impingement.[46] In conditions leading to a narrow ischiofemoral distance, the lesser trochanter may compress the intervening quadratus femoris. This impingement can result in oedema or tearing of quadratus femoris or the hamstring tendon. Using CT, Hujazi et al[47] reported that the mean ischiofemoral distance was smaller in females than males (18.6 mm (SD 8) versus 23.0 mm (SD 7), $p < 0.001$). Won et al[48] confirmed this sex difference with a mean distance in females of 24.3 mm (SD 8.9) compared with 33.2 mm (9.2) in males ($p < 0.001$). Underlying pathologies in the ischiofemoral impingement syndrome include hip abductor insufficiency, high femoral/acetabular version and neck-shaft angle, developmental dysplasia of the hip, valgus intertrochanteric osteotomy, inflammatory myositis, intramuscular lesions of quadratus femoris, ischial tuberosity avulsion fracture, and exostosis.[49–56]

The proximal hamstring syndrome. In the posterior thigh, the sciatic nerve runs superficial to the adductor magnus, but deep to the hamstring muscle complex consisting of biceps femoris, semitendinosus, and semimembranosus. The hamstrings originate from the ischial tuberosity, except for the short head of biceps femoris; the semimembranosus tendon is attached to the superolateral facet of the ischial tuberosity and the conjoint tendon of biceps femoris, and semitendinosus is attached to the inferomedial facet. The sciatic nerve lies approximately 1.2 cm lateral to the ischial tuberosity or the outer border of the semimembranosus tendon.[17,57] Thus, the proximal part of this muscle complex is close to the sciatic nerve.

Proximal hamstring tendon pathologies can cause irritation or entrapment of the sciatic nerve because of the intimate relationship of the nerve and muscle at the level of the ischial tuberosity. Many cases have been associated with repetitive stress on the hamstring tendon and reported in sporting activities involving running, kicking, or jumping.[58] It was reported that, even though high signal intensity of the sciatic nerve was not observed on MRI in two-thirds of patients after hamstring injury, there was significant impairment in sciatic conductivity compared with that in noninjured subjects.[59] Conditions which predispose to the proximal hamstring syndrome include strains (partial tears), avulsions (complete tears), or hamstring tendinopathy and avulsion fracture or apophysitis of the ischial

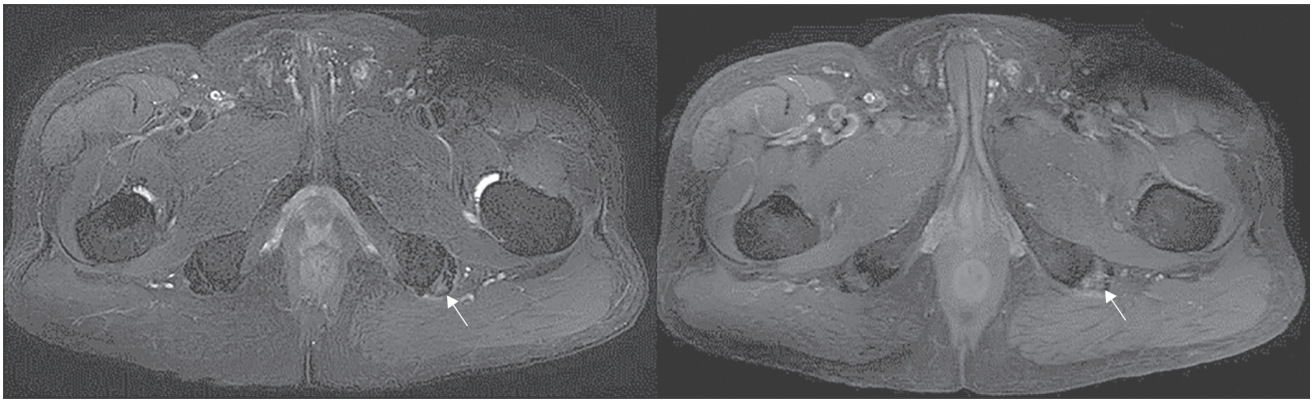

Fig. 4

Proximal hamstring syndrome (57-year-old male). Hamstring tendinopathy was shown with hyperintensity and gadolinium contrast enhancement of the hamstring tendon at the left ischial tuberosity attachment site (white arrows) on T2-weighted axial MRI images.

tuberosity.[60,61] In some patients with a hamstring injury, tight fibrotic bands compressing the sciatic nerve have been found at surgery.[62]

Causes located in the intrapelvic space or at the sacral foramen. As the sciatic nerve passes through the sacral foramen and runs below the sacroiliac joint, a broad spectrum of local pathologies can cause non-discogenic sciatica and the deep gluteal syndrome, including pelvic soft tissue and bone tumours, haematomas, presacral abscesses, aneurysms, infectious and noninfectious sacroiliitis, and gynaecological conditions such as endometriosis and tubo-ovarian abscesses.[63]

Clinical features and physical examination. Most patients complain of intermittent or persistent pain and/or dysaesthesia in the buttock, posterior hip, or thigh rather than low back pain. Significant focal neurological signs including foot drop are not typical presentations of the deep gluteal syndrome. If female patients have cyclical or menstruation-related sciatica, intrapelvic gynaecological conditions should be considered.[19] Pain in the deep gluteal syndrome is commonly increased by activities involving hip flexion on the affected side, such as sitting and walking. Because patients are often uncomfortable when sitting for long periods of time, an antalgic position may be observed. Patients with ischiofemoral impingement usually experience a worsening of pain during running or taking larger steps since the distance between the ischium and lesser trochanter becomes narrower during terminal hip extension (long-stride walking test).[64] Patients with proximal hamstring syndrome, however, may complain of ischial pain during the initial heel strike (long-stride heel strike test).[65]

On inspection, persistent external rotation of the affected leg may be noted when the patient is supine because of tonic contracture of the piriformis ('piriformis sign'). Using the ischial tuberosity as a reference, localization of the tenderness helps to identify which structures are involved. When the affected ischial area is palpated in a seated or contralateral decubitus position with a flexed hip, pain in the lateral ischium or at the ischial tuberosity may suggest ischial impingement or the proximal hamstring syndrome. The piriformis syndrome should be considered in patients with tenderness between the ischium and femoral head, close to the sciatic notch.[19] A palpable sausage-shaped mass over the piriformis muscle area or ipsilateral gluteal atrophy is sometimes seen in the piriformis syndrome, and a thickened hamstring tendon or muscular defect over the ischial tuberosity may be seen in patients with hamstring injuries.[7,66]

The seated piriformis stretch test is positive when pain is induced by adducting an internally rotated hip in the seated position with the hip flexed at 90° and the knee extended. For a positive active piriformis test, pain and tenderness is induced on resisted abduction with external rotation of the hip. If tenderness is localized medial to the ischium, pudendal nerve entrapment should be considered.[19] Different manoeuvres aimed at reproducing the symptoms of the piriformis syndrome have been described, including Lasègue's sign,[7] Freiberg's stretching manoeuvres,[4] Pace's contraction manoeuvre,[67] Beatty's resisted contraction manoeuvre,[68] and the flexion, adduction, and internal rotation test.[69] The ischiofemoral impingement test is intended to induce pain lateral to the ischium when the hip is extended with adduction and to relieve it when the hip is abducted with extension.[64] Provocative tests for hamstring injury include the Puranen-Orava test or the bent-knee stretch test.[62,70] The active hamstring test is based on the assumption that a proximal hamstring injury can result in a selective loss of hamstring strength and activity.[71] In patients with proximal hamstring syndrome, weakness of knee flexion and reproduction of pain on resisted knee flexion may be seen during resisted knee flexion at 30°, but not at 90°, in a seated position. The details of these clinical tests have been described in two excellent reviews.[19,72] Table I summarizes the reported prevalence, sensitivity, and specificity of these tests. Unfortunately, their reliability in the diagnosis of the deep gluteal syndrome has not been thoroughly validated in a large cohort of patients.

Diagnostic investigation. Radiographs of the pelvis and hips can suggest a specific cause such as hip pathology, sacroiliitis, or a calcified shadow in the pelvic cavity. In patients with non-radiological axial spondyloarthritis or acute infectious sacroiliac disease, active sacroiliitis is only visible on MRI.[76] CT can be used to evaluate bony morphology predisposing to ischiofemoral impingement and to guide diagnostic or therapeutic

Table II. A summary of the interventional therapeutic techniques and their outcome in patients with deep gluteal syndrome (DGS), 11 open and seven endoscopic.

Authors	Levels of evidence	Patient number	Diagnosis	Treatment	Reported outcome
Open surgery					
Benson ER and Schutzer SF[37] (1999)	IV (case series)	14	Post-traumatic piriformis syndrome	Piriformis tendon release and sciatic neurolysis	Excellent (n = 11) and good (n = 4) results during the follow-up (24 to 73 months) in 15 interventions
Indrekvam K and Sudmann E[102] (2002)	IV (case series)	19	Piriformis syndrome	Piriformis tenotomy at the insertion on the greater trochanter.	Pain was resolved in eight patients and 13 reported to be better in postoperative patient's assessment after one to 16 years.
Fishman LM et al[70] (2002)	III (retrospective comparative study)	665	Piriformis syndrome	Surgical neurolysis (n = 43)	About 79% of patients reported symptomatic improvement ≥ 50% with piriformis injection and physical therapy after mean 10.2 months. Among 43 patients undergoing surgical therapy, 69% achieved ≥ 50% pain relief.
Meknas K et al[39] (2003)	II (lesser-quality randomized controlled trial)	12	Gemelli-obturator internus syndrome	Sciatic nerve exploration and obturator internus sectioning (n = 6)	The median pain score on VAS was reduced from 8.5 to 3.5 at three-month follow up although the reduction did not reach statistical significance at six-month follow-up. Conservative treatment group showed no significant symptom improvement at any timepoint.
Kim DH et al[103] (2004)	IV (case series)	353	Sciatic nerve injury at the buttock (n = 175) and thigh (n = 178) levels	Sciatic nerve exploration and neurolysis or repair with sutures or grafts	In the buttock-level injuries, patients with positive intraoperative nerve action potentials underwent neurolysis and attained at least grade 3 outcomes in 87% for the tibial division or 71% for the peroneal divisions. For suture repair, recovery to at least grade 3 occurred in 73% for buttock-level tibial division injuries and 30% for peroneal lesions. For graft, good recovery occurred in 62% buttock-level tibial lesions.
Issack PS et al[100] (2007)	IV (case series)	10	Sciatic neuropathy with acetabular fracture	Sciatic nerve decompression	All patients had partial to complete relief of pain and sensory symptoms. But three of seven had no improvement of motor symptoms during a mean 26 months of follow-up.
Young IJ et al[99] (2008)	IV (case series)	44	Proximal hamstring syndrome	Proximal hamstring surgical release	All had failed in conservative treatment preoperatively. After the operation, they reduced average pain intensity on VAS from 6.5 to 2.0 and mean weakness on VAS from 6.6 to 2.8. During a mean 53 months follow-up, 77.3% returned to previous sporting activities.
Topuz K et al[111] (2011)	IV (case series)	42	Sciatic nerve injection injury	Sciatic nerve exploration and decompression or neurolysis (n = 29)	Excellent outcome was achieved in 24.1%, good outcome in 48.2%, fair outcome in 13.8%, and poor outcome in 13.8% during a mean 31 months of follow-up. Fully recovery occurred in 34.6% of subjects with motor deficits or 81.8% of those with sensory deficits.
Bowman KF et al[112] (2013)	IV (case series)	17	Proximal hamstring syndrome	Open debridement and primary hamstring tendon repair	Postoperatively the LEFS score was 73.3 and the mean Marx score was 6.5. Among 14 patients completed subjective surveys, 93% were satisfied with post-operative results and 71% could perform strenuous activities after a mean 32 months.
Salga M et al[87] (2015)	IV (case series)	45	Neurogenic heterotopic ossification (NHO)	Sciatic nerve neurolysis	In total, 55 surgical interventions required sciatic nerve neurolysis in 116 interventions for posterior hip NHO. After sciatic nerve decompression, the proportion of patients able to independently sit increased 84.4% to 93.3% and that of those able to walk increased 15.5% to 42.3%.

Continued

Table II. Continued

Authors	Levels of evidence	Patient number	Diagnosis	Treatment	Reported outcome
Vassalou EE et al[36] (2018)	IV (case series)	74	Primary (n = 12) and secondary (n = 62) piriformis syndrome	Treatment according to underlying pathologies including anticancer surgery, chemotherapy, or radiotherapy	In 59 with available follow-up data, therapeutic outcomes were not associated with imaging findings or the presence of neoplastic disorder. Nonsurgical treatment was effective in 5/8 patients with primary piriformis syndrome and surgical treatments led to partial (n = 6) or complete resolution (n = 11) in 24 with secondary piriformis syndrome.
Endoscopic surgery					
Martin HD et al[26] (2011)	IV (case series)	35	DGS	Arthroscopic sciatic nerve decompression	Pain intensity on VAS was decreased from 6.9 to 2.4 and mHHS was improved from 54.4 to 78.0. About 70% reported excellent to good outcome.
Park MS et al[113] (2016)	IV (case series)	60	DGS, excluding patients undergoing arthroplasty or having acetabular fracture or severe spondylosis.	Endoscopic sciatic nerve neurolysis	Pain intensity on VAS decreased from 7.4 to 2.6 and mHHS was improved from 81.7 to 91.8. Sit pain was reported in 88.3% and 8.3%, before and after the surgery. The proportion of patients with paraesthesia was reduced from 60% to 6.7%. Postoperative satisfaction was excellent or good in 53 (88.3%) patients.
Hernandez A et al[114] (2017)	IV (case series)	2	Ischiofemoral impingement syndrome	Endoscopic total resection of the lesser trochanter	Conservative treatment failed in both patients. The ischio-femoral distance increased from 4 mm to 24 mm and from 6 mm to 22 mm. Postoperative mHHS was improved from 30 to 82 and 40 to 87. But one had mild persistent pain in the right buttock at two-year follow-up.
Possover M[98] (2017)	IV (case series)	46	Intraneural endometriosis of the sciatic nerve	Laparoscopic sciatic nerve resection (more than 30% of the sciatic nerve)	The resected portion was the cranial part (L5, 82.6%), the middle (S1/S2, 13.0%) or the caudal part (4.3%). Temporal neurapraxia of the obturator nerve was presented in 75%, but fully recovered within two weeks. Pain intensity on VAS decreased from 9.33 to 1.25 at a five-year follow-up. Significant functional recovery occurred in most patients after 2.5 to three years and normal gait function was achieved after four to five years.
Ham DH et al[104] (2018)	IV (case series)	24	DGS	Endoscopic sciatic nerve decompression	Pain intensity on VAS was reduced from 7.1 to 2.5 and mHHS was improved from 59.4 to 85.0 at one-year follow-up. Excellent and good outcome was noted in 62.5% and 25.0%, respectively.
Ilizaliturri VM et al[115] (2018)	IV (case series)	15	Piriformis syndrome	Endoscopic release of the piriformis on the greater trochanter and sciatic nerve exploration	Pain intensity on VAS was reduced from 7.4 to 1.86 and mHHS was improved from 46.8 to 84.9 at 24-month follow-up. All patients were able to return to work. Excellent and good outcome was noted in 73.3% and 20.0%, respectively.
Park MS et al[116] (2019)	IV (case series)	25	Post-traumatic sciatic neuropathy with a history of fracture or reconstructive acetabular surgery	Endoscopic sciatic nerve decompression	More favourable outcomes were observed in the idiopathic DGS group (n = 45) than in the post-traumatic group. mHHS was improved from 61.5 to 84.1 in post-traumatic group and from 73.8 to 94.4 in idiopathic group after two year or more. Excellent to good outcome was reported in 56.0% of post-traumatic patients versus 84.4% of those with idiopathic DGS.

DGS, deep gluteal syndrome; LEFS, Lower Extremity Functional Scale;[100] mHHS, modified Harris Hip Score;[101] VAS, visual analogue scale.

injections in patients with deep gluteal syndrome.[47,77] Recently, high-resolution ultrasonography has been proposed as a reliable diagnostic tool and to assist in diagnostic and therapeutic injections.[78,79]

MRI is the most useful imaging tool for the diagnosis of the deep gluteal syndrome. High-resolution pelvic imaging is now available with 3-Tesla MRI, which offers visualization of deep gluteal structures and the sciatic nerve with identification of underlying pathology in the deep gluteal space, such as an entrapped sciatic nerve, compressive fibrous bands, and pathological muscle changes (Figures 2 to 4). MRI evaluation of the peripheral nerves, often referred to as MR neurography, is performed with a fluid-sensitive fat-suppressed T2-weighted sequence and T1-weighted sequence. Due to the abundant fat around the fascicles and the nerve itself, these structures are clearly visible on T1-weighted images. Normal nerves show intermediate-to-minimal hyperintensity on T2-weighted images and have a fascicular pattern, while neuropathic nerves have an abnormally increased T2-signal intensity, similar to that of vessels.[80] The course of the nerve, its calibre, fascicular pattern and size, as well as the presence of space-occupying lesions along the nerve, can also be assessed with T1-weighted images, whereas signal intensity and fascicular shape are assessed on axial fat-suppressed T2-weighted images.[81–83] MR neurography can also detect aberrant anatomy of the sciatic nerve.[15] This is best performed in the sagittal plane, with T1- or intermediate-weighted sequences without fat suppression.[84] Furthermore, denervation-induced muscle signal intensity changes can indicate the presence of neuropathy and aid in determining the chronicity or level of nerve pathology.

Electrodiagnostic testing is another useful tool, especially beneficial in excluding lumbosacral radiculopathy. Although these tests are often normal in the deep gluteal syndrome, a peripheral nerve injury can be diagnosed with localization by either: 1) detecting decreased conduction velocities or amplitude of action potentials in a nerve conduction study, or 2) detecting a denervation pattern with electromyography (EMG).[85,86] In patients with an axonotmetic or neurotmetic lesion, it is difficult to localize the injured site using nerve conduction studies. However, if present, the degree of delayed conduction has been reported to be correlated with the duration of symptoms in patients with piriformis syndrome.[87] In these patients, a significant dynamic delay in the H-wave was seen several seconds after placing the hip in the flexion, adduction, and internal rotation test. However, this finding has not been reproduced by others.[69,88] In order to identify the signs of active denervation, EMG should be performed at least three to four weeks after the onset of symptoms. EMG abnormalities are more commonly seen in muscles innervated by the peroneal nerve.[85] In a retrospective study, there was significant association between the symptoms and EMG findings in patients requiring sciatic nerve neurolysis.[89]

Treatment. Various forms of treatment have been described and proposed, but an optimal treatment algorithm has not been established. In a recent systematic review, the decision to recommend surgery was made according to clinical and investigational findings in 50% of studies involving the deep gluteal syndrome, and surgical release of the sciatic nerve was performed after conservative treatments failed in the other 50%.[16] The indications for each treatment are not well-defined because there is a paucity of controlled trials examining the effectiveness of non-surgical and surgical treatments. Nevertheless, if patients do not have clear-cut indications for surgery, a stepwise approach—escalating from conservative to invasive management—is recommended. In a study of 64 patients suspected of having piriformis syndrome, almost all (41/42, 98%) of those with normal MRI and CT studies had complete resolution spontaneously or with conservative treatment over a maximum of 35 days.[36] Thus, early surgical treatment can be avoided in these patients. Early surgery, however, is required in patients with a space-occupying lesion compressing the sciatic nerve, especially those with potentially malignant lesions or those with chronic neurological impairment.

Non-surgical treatment. The nonsurgical management of patients with the deep gluteal syndrome involves conservative multidisciplinary treatments with rest, oral pharmacological agents, physiotherapy, and injections. Oral non-steroidal anti-inflammatory drugs (NSAIDs), neuropathic agents such as gabapentin and pregabalin, and muscle relaxants may be used. A tailored educational programme can be implemented to avoid provoking postures or movements. For example, these patients should avoid prolonged cross-legged sitting or internal rotation of the hips. Physiotherapy includes heat, muscle stimulation, soft tissue mobilization, stretching and strengthening exercises, and aerobic conditioning.[90] If taut bands are palpated in the gluteal muscles and myofascial pain syndrome is suspected, trigger point injection or massage can be used.[91] In a prospective study of 250 patients with piriformis syndrome, treatment with NSAIDs, muscle relaxants, and physiotherapy led to the resolution of symptoms in 128 (51%) after three months.[92]

Diagnostic and therapeutic local anaesthetic injections can be performed using anatomical landmarks or under ultrasound, CT, or MRI guidance. The accuracy of injections into the piriformis is much higher when using an ultrasound-guided method (95%) than when using anatomical landmarks (30%).[93] Although glucocorticoid intramuscular injections with or without local anaesthetic improved pain in patients with piriformis syndrome, a randomized double-blind study did not find any superior effect of lidocaine and betamethasone compared with lidocaine alone.[94–96] Botulinum toxin injection has been introduced as treatment for the deep gluteal syndrome during the last two decades. In two double-blind placebo-controlled trials, botulinum toxin was superior to a combination of lidocaine and glucocorticoids or a placebo in patients with piriformis syndrome.[97,98] Using a physiotherapy protocol combined with triamcinolone injections, Fishman et al[69] found that 279 (79%) of 353 patients with piriformis syndrome had at least 50% improvement. However, in a study involving 162 patients using MRI-guided local anaesthetic, only 24 (15%) had complete relief without recurrence, 112 (69%) had temporary (< four months) relief with subsequent recurrence and 26 (16%) had no relief.[25] Thus, despite the promising initial outcomes, conservative treatments may not provide long-lasting benefit.

Surgical treatment. In order to review the surgical treatments for patients with the deep gluteal syndrome, a comprehensive

search of English literature was performed using the MEDLINE via PubMed, EMBASE, Cochrane Library, and Scopus databases. The search was carried out in July 2019 and the complete search strings are shown as Supplementary Material. Unpublished data, letters to the editor, case reports, review articles, and instructional courses were excluded. Two of the authors (JWP, YKL) independently screened the titles and abstracts from the results of the search and full texts were reviewed to evaluate whether the papers were appropriate for inclusion (Supplementary Figure a). A total of 18 studies were included. The interventional therapeutic techniques and their outcomes are summarized in Table II. Most studies were case series. Sciatic neurolysis for piriformis syndrome was performed in a Level III retrospective comparative study and sciatic nerve exploration with obturator internus sectioning was performed in a Level II randomized controlled trial.[69,99] The decision to offer surgery was made following failure of conservative treatment and those undergoing surgery were mostly those with more severe or recurring symptoms. In patients in whom lesions were suspected to cause a mass effect including haematoma, lipoma, or heterotopic ossification, surgery was indicated without preoperative conservative treatment.[16] In total, 11 studies used open surgical procedures and seven used endoscopic treatments.

Endoscopic decompression of sciatic nerve entrapment was reported to be an effective and minimally invasive approach for the deep gluteal syndrome in 2003.[102] It has subsequently become a popular procedure for these patients; among nine papers published after 2011, six involved endoscopic treatment (Table II). It has been reported that patients undergoing this form of treatment have fewer complications, including pain than those undergoing open procedures.[16,26] Both open and endoscopic techniques had good postoperative outcomes, although endoscopic treatment requires a high level of skill and a severely adherent sciatic nerve may be difficult to release using this technique.

To date, there is no clear guidance from the literature regarding operating methods for the treatment of the deep gluteal syndrome. When planning surgery, the underlying pathological conditions should be considered. All surgical procedures initially focused on exploration of the sciatic nerve regardless of the aetiology of the deep gluteal syndrome. After exploration of the nerve, specific methods of decompression varied according to the aetiology: piriformis tenotomy or release for piriformis syndrome,[36,69,78,103] obturator internus sectioning for gemelli-obturator internus syndrome,[99] proximal hamstring release or debridement with primary hamstring tendon repair for proximal hamstring syndrome,[104,105] lesser trochanter resection for ischiofemoral impingement,[23] and neurolysis or repair with sutures or grafts for sciatic nerve injuries following trauma or heterotrophic neurogenic ossification.[106–109] In endometriosis involving the sciatic nerve, a transperitoneal laparoscopic approach and complete removal with lesion-free margins can be used.[110] Possover[103] observed gradual recovery of sciatic function over five years after partial (1/3 to 2/3) resection of the nerve due to endometriosis. However, this result conflicts with most of the orthopaedic literature. Nerve resection in limb salvage surgery for sarcoma encasing the sciatic nerve is known to result in significant morbidity and functional deficit.[111] Therefore, epineural dissection can be used in patients with unavoidable resection of the sciatic nerve to minimize postoperative impairment.[112]

In conclusion, the deep gluteal syndrome is a recently classified entity although one of its subtypes, the piriformis syndrome, has long been well known. The deep gluteal syndrome is essentially defined as compression of the sciatic or pudendal nerve by any anatomical structure in the deep gluteal space. As the symptoms of this syndrome are similar to those of lumbar disc herniation, cautious history-taking, physical examination, electrodiagnostic studies, and imaging such as MRI are needed for an accurate diagnosis. After excluding spinal lesions, pelvic MRI can aid in the diagnosis and identifying the underlying pathology. It can initially be treated conservatively with rest, drugs including NSAIDs, injections, and physiotherapy. However, if severe symptoms continue or recur after conservative therapy, or if a mass lesion conspicuously compresses the sciatic nerve, surgery is indicated. Good results have been reported following both open and endoscopic decompression of the sciatic nerve. However, recent literature supports the preferential use of endoscopic neural release because it is less invasive and has fewer complications.

Take home message

- Deep gluteal syndrome results from the compression of the sciatic or pudendal nerve in the deep gluteal space and can lead to pseudosciatica.
- A systematic and rational approach including MRI are needed for an accurate diagnosis.
- It can initially be treated conservatively and surgical decompression is indicated in case of severe symptoms, recurrence after conservative therapy, or a mass lesion compressing the sciatic nerve.

Supplementary material

Flow diagram of the process in screening papers andsearch strings used for the present systematic review.

References

1. **Birnbaum K**, **Prescher A**, **Hessler S**, **Heller KD**. The sensory innervation of the hip joint—an anatomical study. *Surg Radiol Anat*. 1997;19(6):371–375.
2. **el Barzouhi A**, **Vleggeert-Lankamp CL**, **Lycklama à Nijeholt GJ**, **et al**. Influence of low back pain and prognostic value of MRI in sciatica patients in relation to back pain. *PLoS One*. 2014;9(3):e90800.
3. **Ogin GA**. Pseudosciatica. *Anesth Analg*. 1989;69(2):261.
4. **Yeoman W**. The relation of arthritis of the sacro-iliac joint to sciatica, with an analysis of 100 cases. *Lancet*. 1928;2(5492):1119–1122.
5. **Freiburg AH**, **Vinke TA**. Sciatica and the sacroiliac joint. *J Bone Joint Surg Am*. 1934;16-A:126–136.
6. **Beaton LE**, **Anson BJ**. The sciatic nerve and the piriformis muscle. Their interrelation and possible cause of coccygodynia. *J Bone Joint Surg Am*. 1938;20-A:686–688.
7. **Robinson DR**. Pyriformis syndrome in relation to sciatic pain. *Am J Surg*. 1947;73(3):355–358.
8. **Jankovic D**, **Peng P**, **van Zundert A**. Brief review: piriformis syndrome: etiology, diagnosis, and management. *Can J Anaesth*. 2013;60(10):1003–1012.
9. **Lutz EG**. Credit-card-wallet sciatica. *JAMA*. 1978;240(8):738.
10. **Fishman LM**, **Schaefer MP**. The piriformis syndrome is underdiagnosed. *Muscle Nerve*. 2003;28(5):646–649.
11. **Miller TA**, **White KP**, **Ross DC**. The diagnosis and management of Piriformis Syndrome: myths and facts. *Can J Neurol Sci*. 2012;39(5):577–583.

12. **Stewart JD**. The piriformis syndrome is overdiagnosed. *Muscle Nerve*. 2003;28(5):644–646.
13. **McCrory P**, **Bell S**. Nerve entrapment syndromes as a cause of pain in the hip, groin and buttock. *Sports Med*. 1999;27(4):261–274.
14. **Carro LP**, **Hernando MF**, **Cerezal L**, **et al**. Deep gluteal space problems: piriformis syndrome, ischiofemoral impingement and sciatic nerve release. *Muscles Ligaments Tendons J*. 2016;6(3):384–396.
15. **Hernando MF**, **Cerezal L**, **Pérez-Carro L**, **Abascal F**, **Canga A**. Deep gluteal syndrome: anatomy, imaging, and management of sciatic nerve entrapments in the subgluteal space. *Skeletal Radiol*. 2015;44(7):919–934.
16. **Kay J**, **de Sa D**, **Morrison L**, **et al**. Surgical management of deep gluteal syndrome causing sciatic nerve entrapment: a systematic review. *Arthroscopy*. 2017;33(12):2263–2278.
17. **Pombo M**, **Bradley JP**. Proximal hamstring avulsion injuries: a technique note on surgical repairs. *Sports Health*. 2009;1(3):261–264.
18. **Coppieters MW**, **Alshami AM**, **Babri AS**, **et al**. Strain and excursion of the sciatic, tibial, and plantar nerves during a modified straight leg raising test. *J Orthop Res*. 2006;24(9):1883–1889.
19. **Martin HD**, **Reddy M**, **Gómez-Hoyos J**. Deep gluteal syndrome. *J Hip Preserv Surg*. 2015;2(2):99–107.
20. **Hallin RP**. Sciatic pain and the piriformis muscle. *Postgrad Med*. 1983;74(2):69–72.
21. **Papadopoulos EC**, **Khan SN**. Piriformis syndrome and low back pain: a new classification and review of the literature. *Orthop Clin North Am*. 2004;35(1):65–71.
22. **Kean Chen C**, **Nizar AJ**. Prevalence of piriformis syndrome in chronic low back pain patients. A clinical diagnosis with modified FAIR test. *Pain Pract*. 2013;13(4):276–281.
23. **Shah SS**, **Consuegra JM**, **Subhawong TK**, **Urakov TM**, **Manzano GR**. Epidemiology and etiology of secondary piriformis syndrome: A single-institution retrospective study. *J Clin Neurosci*. 2019;59:209–212.
24. **Bartret AL**, **Beaulieu CF**, **Lutz AM**. Is it painful to be different? Sciatic nerve anatomical variants on MRI and their relationship to piriformis syndrome. *Eur Radiol*. 2018;28(11):4681–4686.
25. **Filler AG**, **Haynes J**, **Jordan SE**, **et al**. Sciatica of nondisc origin and piriformis syndrome: diagnosis by magnetic resonance neurography and interventional magnetic resonance imaging with outcome study of resulting treatment. *J Neurosurg Spine*. 2005;2(2):99–115.
26. **Martin HD**, **Shears SA**, **Johnson JC**, **Smathers AM**, **Palmer IJ**. The endoscopic treatment of sciatic nerve entrapment/deep gluteal syndrome. *Arthroscopy*. 2011;27(2):172–181.
27. **Miller GK**. Editorial Commentary: Ischiofemoral Impingement: Is This the "New" Femoroacetabular Impingement? *Arthroscopy*. 2016;32(7):1285.
28. **Wilson TJ**, **Spinner RJ**, **Mohan R**, **Gibbs CM**, **Krych AJ**. Sciatic Nerve Injury After Proximal Hamstring Avulsion and Repair. *Orthop J Sports Med*. 2017;5(7):2325967117713685.
29. **Nakano N**, **Yip G**, **Khanduja V**. Current concepts in the diagnosis and management of extra-articular hip impingement syndromes. *Int Orthop*. 2017;41(7):1321–1328.
30. **Fleming P**, **Lenehan B**, **O'Rourke S**, **et al**. Strain on the human sciatic nerve in vivo during movement of the hip and knee. *J Bone Joint Surg Br*. 2003;85-B(3):363–365.
31. **Beaton LE**, **Anson BJ**. The relation of the sciatic nerve and of its subdivisions to the piriformis muscle. *Anat Rec*. 1937;70(1):1–5.
32. **Smoll NR**. Variations of the piriformis and sciatic nerve with clinical consequence: a review. *Clin Anat*. 2010;23(1):8–17.
33. **Barbosa ABM**, **Santos PVD**, **Targino VA**, **et al**. Sciatic nerve and its variations: is it possible to associate them with piriformis syndrome? *Arq Neuropsiquiatr*. 2019;77(9):646–653.
34. **Tomaszewski KA**, **Graves MJ**, **Henry BM**, **et al**. Surgical anatomy of the sciatic nerve: A meta-analysis. *J Orthop Res*. 2016;34(10):1820–1827.
35. **Foster MR**. Piriformis syndrome. *Orthopedics*. 2002;25(8):821–825.
36. **Vassalou EE**, **Katonis P**, **Karantanas AH**. Piriformis muscle syndrome: A cross-sectional imaging study in 116 patients and evaluation of therapeutic outcome. *Eur Radiol*. 2018;28(2):447–458.
37. **Benson ER**, **Schutzer SF**. Posttraumatic piriformis syndrome: diagnosis and results of operative treatment. *J Bone Joint Surg Am*. 1999;81-A(7):941–949.
38. **Windisch G**, **Braun EM**, **Anderhuber F**. Piriformis muscle: clinical anatomy and consideration of the piriformis syndrome. *Surg Radiol Anat*. 2007;29(1):37–45.
39. **Balius R**, **Susín A**, **Morros C**, **et al**. Gemelli-obturator complex in the deep gluteal space: an anatomic and dynamic study. *Skeletal Radiol*. 2018;47(6):763–770.
40. **Leigh RE**. Obturator internus spasm as a cause of pelvic and sciatic distress. *J Lancet*. 1952;72(6):286–287.
41. **Dalmau-CarolJ**. Myofascial pain syndrome affecting the piriformis and the obturator internus muscle. *Pain Pract*. 2005;5(4):361–363.
42. **Khodaee M**, **Jones D**, **Spittler J**. Obturator Internus and Obturator Externus Strain in a High School Quarterback. *Asian J Sports Med*. 2015;6(3):e23481.
43. **Aouba A**, **Breton S**, **Harroche A**, **et al**. Spontaneous obturator internus muscle haematoma: a new unpublished cause of ilioipelvic pain in haemophilia. *Haemophilia*. 2013;19(1):157–160.
44. **Bertrand SL**, **Lincoln ED**, **Prohaska MG**. Primary pyomyositis of the pelvis in children: a retrospective review of 8 cases. *Orthopedics*. 2011;34(12):e832–e840.
45. **Chen B**, **Rispoli L**, **Stitik T**, **Leong M**. Successful Treatment of Gluteal Pain from Obturator Internus Tendinitis and Bursitis with Ultrasound-Guided Injection. *Am J Phys Med Rehabil*. 2017;96(10):e181–e184.
46. **Johnson KA**. Impingement of the lesser trochanter on the ischial ramus after total hip arthroplasty. Report of three cases. *J Bone Joint Surg Am*. 1977;59-A(2):268–269.
47. **Hujazi I**, **Jones T**, **Johal S**, **et al**. The normal ischiofemoral distance and its variations. *J Hip Preserv Surg*. 2016;3(3):197–202.
48. **Won H**, **Lee YK**, **Lee BS**, **et al**. Normal Ischiofemoral Distance and Its Associated Factors: Computed Tomography-Based Study. *Arthroscopy*. 2020;36(1):150–155.
49. **DiSciullo AA**, **Stelzer JW**, **Martin SD**. Dynamic Ischiofemoral Impingement: Case-Based Evidence of Progressive Pathophysiology from Hip Abductor Insufficiency: A Report of Two Cases. *JBJS Case Connect*. 2018;8(4):e107.
50. **Morris WZ**, **Fowers CA**, **Weinberg DS**, **et al**. Hip morphology predicts posterior hip impingement in a cadaveric model. *Hip Int*. 2019;29(3):322–327.
51. **Ohnishi Y**, **Suzuki H**, **Nakashima H**, **et al**. Radiologic Correlation Between the Ischiofemoral Space and Morphologic Characteristics of the Hip in Hips With Symptoms of Dysplasia. *AJR Am J Roentgenol*. 2018;210(3):608–614.
52. **de Carvalho AD**, **Garcia FL**, **Nogueira-Barbosa MH**. Ischiofemoral impingement secondary to valgus intertrochanteric osteotomy: a case report. *Radiol Bras*. 2017;50(5):335–337.
53. **Kwee RM**, **Ahlawat S**, **Fritz J**. Magnetic Resonance Imaging Findings of Ischiofemoral Impingement in Patients With Inflammatory Myositis: An Observational Study. *J Comput Assist Tomogr*. 2017;41(2):212–216.
54. **Zibis AH**, **Fyllos AH**, **Karantanas AH**, **et al**. Quadratus femoris tear as an unusual cause of hip pain: a case report. *Hip Int*. 2016;26(1):e7–e9.
55. **Spencer-Gardner L**, **Bedi A**, **Stuart MJ**, **et al**. Ischiofemoral impingement and hamstring dysfunction as a potential pain generator after ischial tuberosity apophyseal fracture non-union/malunion. *Knee Surg Sports Traumatol Arthrosc*. 2017;25(1):55–61.
56. **Viala P**, **Vanel D**, **Larbi A**, **Cyteval C**, **Laredo J-D**. Bilateral ischiofemoral impingement in a patient with hereditary multiple exostoses. *Skeletal Radiol*. 2012;41(12):1637–1640.
57. **Miller SL**, **Webb GR**. The proximal origin of the hamstrings and surrounding anatomy encountered during repair. Surgical technique. *J Bone Joint Surg Am*. 2008;90-A(Suppl 2 Pt 1):108–116.
58. **Lempainen L**, **Johansson K**, **Banke IJ**, **et al**. Expert opinion: diagnosis and treatment of proximal hamstring tendinopathy. *Muscles Ligaments Tendons J*. 2015;5(1):23–28.
59. **Kouzaki K**, **Nakazato K**, **Mizuno M**, **et al**. Sciatic Nerve Conductivity is Impaired by Hamstring Strain Injuries. *Int J Sports Med*. 2017;38(11):803–808.
60. **Bucknor MD**, **Steinbach LS**, **Saloner D**, **Chin CT**. Magnetic resonance neurography evaluation of chronic extraspinal sciatica after remote proximal hamstring injury: a preliminary retrospective analysis. *J Neurosurg*. 2014;121(2):408–414.
61. **Kujala UM**, **Orava S**, **Karpakka J**, **Leppävuori J**, **Mattila K**. Ischial tuberosity apophysitis and avulsion among athletes. *Int J Sports Med*. 1997;18(2):149–155.
62. **Puranen J**, **Orava S**. The hamstring syndrome. A new diagnosis of gluteal sciatic pain. *Am J Sports Med*. 1988;16(5):517–521.
63. **Kulcu DG**, **Naderi S**. Differential diagnosis of intraspinal and extraspinal non-discogenic sciatica. *J Clin Neurosci*. 2008;15(11):1246–1252.
64. **Gómez-Hoyos J**, **Martin RL**, **Schröder R**, **Palmer IJ**, **Martin HD**. Accuracy of 2 Clinical Tests for Ischiofemoral Impingement in Patients With Posterior Hip Pain and Endoscopically Confirmed Diagnosis. *Arthroscopy*. 2016;32(7):1279–1284.
65. **Martin HD**, **Khoury A**, **Schröder R**, **Palmer IJ**. Ischiofemoral impingement and hamstring syndrome as causes of posterior hip pain: where do we go next? *Clin Sports Med*. 2016;35(3):469–486.
66. **Sarimo J**, **Lempainen L**, **Mattila K**, **Orava S**. Complete proximal hamstring avulsions: a series of 41 patients with operative treatment. *Am J Sports Med*. 2008;36(6):1110–1115.
67. **Pace JB**, **Nagle D**. Piriform syndrome. *West J Med*. 1976;124(6):435–439.

68. **Beatty RA**. The piriformis muscle syndrome: a simple diagnostic maneuver. *Neurosurgery*. 1994;34(3):512–514.
69. **Fishman LM**, **Dombi GW**, **Michaelsen C**, **et al**. Piriformis syndrome: diagnosis, treatment, and outcome—a 10-year study. *Arch Phys Med Rehabil*. 2002;83(3):295–301.
70. **Fredericson M**, **Moore W**, **Guillet M**, **Beaulieu C**. High hamstring tendinopathy in runners: meeting the challenges of diagnosis, treatment, and rehabilitation. *Phys Sportsmed*. 2005;33(5):32–43.
71. **Sole G**, **Milosavljevic S**, **Nicholson HD**, **Sullivan SJ**. Selective strength loss and decreased muscle activity in hamstring injury. *J Orthop Sports Phys Ther*. 2011;41(5):354–363.
72. **Kirschner JS**, **Foye PM**, **Cole JL**. Piriformis syndrome, diagnosis and treatment. *Muscle Nerve*. 2009;40(1):10–18.
73. **Hopayian K**, **Danielyan A**. Four symptoms define the piriformis syndrome: an updated systematic review of its clinical features. *Eur J Orthop Surg Traumatol*. 2018;28(2):155–164.
74. **Martin HD**, **Kivlan BR**, **Palmer IJ**, **Martin RL**. Diagnostic accuracy of clinical tests for sciatic nerve entrapment in the gluteal region. *Knee Surg Sports Traumatol Arthrosc*. 2014;22(4):882–888.
75. **Martin RL**, **Schröder RG Gomez-HoyosJ**, **Gomez-Hoyos J**, **et al**. Accuracy of 3 Clinical Tests to Diagnose Proximal Hamstrings Tears With and Without Sciatic Nerve Involvement in Patients With Posterior Hip Pain. *Arthroscopy*. 2018;34(1):114–121.
76. **Rudwaleit M**, **Landewé R**, **van der Heijde D**, **et al**. The development of Assessment of SpondyloArthritis international Society classification criteria for axial spondyloarthritis (part I): classification of paper patients by expert opinion including uncertainty appraisal. *Ann Rheum Dis*. 2009;68(6):770–776.
77. **Ozisik PA**, **Toru M**, **Denk CC**, **Taskiran OO**, **Gundogmus B**. CT-guided piriformis muscle injection for the treatment of piriformis syndrome. *Turk Neurosurg*. 2014;24(4):471–477.
78. **Rosales J**, **García N**, **Rafols C**, **Pérez M**, **Verdugo MA**. Perisciatic Ultrasound-Guided Infiltration for Treatment of Deep Gluteal Syndrome: Description of Technique and Preliminary Results. *J Ultrasound Med*. 2015;34(11):2093–2097.
79. **Chang KV**, **Wu W-T**, **Lew HL**, **Özçakar L**. Ultrasound imaging and guided injection for the lateral and posterior hip. *Am J Phys Med Rehabil*. 2018;97(4):285–291.
80. **Petchprapa CN**, **Rosenberg ZS**, **Sconfienza LM**, **et al**. MR imaging of entrapment neuropathies of the lower extremity. Part 1. The pelvis and hip. *Radiographics*. 2010;30(4):983–1000.
81. **Chhabra A**, **Andreisek G**, **Soldatos T**, **et al**. MR neurography: past, present, and future. *AJR Am J Roentgenol*. 2011;197(3):583–591.
82. **Chhabra A**, **Williams EH**, **Wang KC**, **Dellon AL**, **Carrino JA**. MR neurography of neuromas related to nerve injury and entrapment with surgical correlation. *AJNR Am J Neuroradiol*. 2010;31(8):1363–1368.
83. **Chhabra A**, **Chalian M**, **Soldatos T**, **et al**. 3-T high-resolution MR neurography of sciatic neuropathy. *AJR Am J Roentgenol*. 2012;198(4):W357–364.
84. **Varenika V**, **Lutz AM**, **Beaulieu CF**, **Bucknor MD**. Detection and prevalence of variant sciatic nerve anatomy in relation to the piriformis muscle on MRI. *Skeletal Radiol*. 2017;46(6):751–757.
85. **Yuen EC**, **So YT**, **Olney RK**. The electrophysiologic features of sciatic neuropathy in 100 patients. *Muscle Nerve*. 1995;18(4):414–420.
86. **Robinson LR**. Traumatic injury to peripheral nerves. *Muscle Nerve*. 2000;23(6):863–873.
87. **Chang CW**, **Shieh SF**, **Li C-M**, **Wu W-T**, **Chang K-F**. Measurement of motor nerve conduction velocity of the sciatic nerve in patients with piriformis syndrome: a magnetic stimulation study. *Arch Phys Med Rehabil*. 2006;87(10):1371–1375.
88. **Robinson LR**. Is the FAIR-test a fair method of detecting piriformis syndrome? *Muscle Nerve*. 2019;60(3):E20.
89. **Salga M**, **Jourdan C**, **Durand MC**, **et al**. Sciatic nerve compression by neurogenic heterotopic ossification: use of CT to determine surgical indications. *Skeletal Radiol*. 2015;44(2):233–240.
90. **Gómez-Hoyos J**, **Martin RL**, **Martin HD**. Current Concepts Review: Evaluation and Management of Posterior Hip Pain. *J Am Acad Orthop Surg*. 2018;26(17):597–609.
91. **Probst D**, **Stout A**, **Hunt D**. Piriformis Syndrome: A Narrative Review of the Anatomy, Diagnosis, and Treatment. *PM R*. 2019;11(Suppl 1):S54–S63.
92. **Michel F**, **Decavel P**, **Toussirot E**, **et al**. Piriformis muscle syndrome: diagnostic criteria and treatment of a monocentric series of 250 patients. *Ann Phys Rehabil Med*. 2013;56(5):371–383.
93. **Finnoff JT**, **Hurdle MF**, **Smith J**. Accuracy of ultrasound-guided versus fluoroscopically guided contrast-controlled piriformis injections: a cadaveric study. *J Ultrasound Med*. 2008;27(8):1157–1163.
94. **Masala S**, **Crusco S**, **Meschini A**, **et al**. Piriformis syndrome: long-term follow-up in patients treated with percutaneous injection of anesthetic and corticosteroid under CT guidance. *Cardiovasc Intervent Radiol*. 2012;35(2):375–382.
95. **Albayrak A**, **Ozcafer R**, **Balioglu MB**, **et al**. Piriformis syndrome: treatment of a rare cause of posterior hip pain with fluoroscopic-guided injection. *Hip Int*. 2015;25(2):172–175.
96. **Misirlioglu TO**, **Akgun K**, **Palamar D**, **Erden MG**, **Erbilir T**. Piriformis syndrome: comparison of the effectiveness of local anesthetic and corticosteroid injections: a double-blinded, randomized controlled study. *Pain Physician*. 2015;18(2):163–171.
97. **Childers MK**, **Wilson DJ**, **Gnatz SM**, **Conway RR**, **Sherman AK**. Botulinum toxin type A use in piriformis muscle syndrome: a pilot study. *Am J Phys Med Rehabil*. 2002;81(10):751–759.
98. **Fishman LM**, **Wilkins AN**, **Rosner B**. Electrophysiologically identified piriformis syndrome is successfully treated with incobotulinum toxin a and physical therapy. *Muscle Nerve*. 2017;56(2):258–263.
99. **Meknas K**, **Christensen A**, **Johansen O**. The internal obturator muscle may cause sciatic pain. *Pain*. 2003;104(1):375–380.
100. **Bradley JP**, **Cohen SB**, **Bradley JP**. Operative management of partial-thickness tears of the proximal hamstring muscles in athletes. *Am J Sports Med*. 2013;41(6):1363–1371.
101. **Byrd JW**, **Jones KS**. Prospective analysis of hip arthroscopy with 2-year follow-up. *Arthroscopy*. 2000;16(6):578–587.
102. **Dezawa A**, **Kusano S**, **Miki H**. Arthroscopic release of the piriformis muscle under local anesthesia for piriformis syndrome. *Arthroscopy*. 2003;19(5):554–557.
103. **Possover M**. Five-Year Follow-Up After Laparoscopic Large Nerve Resection for Deep Infiltrating Sciatic Nerve Endometriosis. *J Minim Invasive Gynecol*. 2017;24(5):822–826.
104. **Young IJ**, **van Riet RP**, **Bell SN**. Surgical release for proximal hamstring syndrome. *Am J Sports Med*. 2008;36(12):2372–2378.
105. **Issack PS**, **Toro JB**, **Buly RL**, **Helfet DL**. Sciatic nerve release following fracture or reconstructive surgery of the acetabulum. *J Bone Joint Surg Am*. 2007;89-A(7):1432–1437.
106. **Nazlıkul H**, **Ural FG**, **Öztürk GT**, **Öztürk ADT**. Evaluation of neural therapy effect in patients with piriformis syndrome. *J Back Musculoskelet Rehabil*. 2018;31(6):1105–1110.
107. **Indrekvam K**, **Sudmann E**. Piriformis muscle syndrome in 19 patients treated by tenotomy—a 1- to 16-year follow-up study. *Int Orthop*. 2002;26(2):101–103.
108. **Kim DH**, **Murovic JA**, **Tiel R**, **Kline DG**. Management and outcomes in 353 surgically treated sciatic nerve lesions. *J Neurosurg*. 2004;101(1):8–17.
109. **Ham DH**, **Chung WC**, **Jung DU**. Effectiveness of Endoscopic Sciatic Nerve Decompression for the Treatment of Deep Gluteal Syndrome. *Hip Pelvis*. 2018;30(1):29–36.
110. **Lomoro P**, **Simonetti I**, **Nanni A**, **et al**. Extrapelvic Sciatic Nerve Endometriosis, the Role of Magnetic Resonance Imaging: Case Report and Systematic Review. *J Comput Assist Tomogr*. 2019;43(6):976–980.
111. **Sweiti H**, **Tamimi N**, **Bormann F**, **et al**. Limb-Salvage Surgery of Soft Tissue Sarcoma with Sciatic Nerve Involvement. *Sarcoma*. 2018;2018(6):6483579.
112. **Clarkson PW**, **Griffin AM**, **Catton CN**, **et al**. Epineural dissection is a safe technique that facilitates limb salvage surgery. *Clin Orthop Relat Res*. 2005;438(438):92–96.
113. **Topuz K**, **Kutlay M**, **Simşek H**, **et al**. Early surgical treatment protocol for sciatic nerve injury due to injection—a retrospective study. *Br J Neurosurg*. 2011;25(4):509–515.
114. **Park MS**, **Yoon SJ**, **Jung SY**, **Kim SH**. Clinical results of endoscopic sciatic nerve decompression for deep gluteal syndrome: mean 2-year follow-up. *BMC Musculoskelet Disord*. 2016;17(1):218.
115. **Hernandez A**, **Haddad S**, **Nuñez JH**, **et al**. Ischiofemoral impingement syndrome: outcomes of endoscopic resection of the lesser trochanter. *Clin Orthop Surg*. 2017;9(4):529–533.
116. **Ilizaliturri VM**, **Arriaga R**, **Villalobos FE**, **Suarez-Ahedo C**. Endoscopic release of the piriformis tendon and sciatic nerve exploration. *J Hip Preserv Surg*. 2018;5(3):301–306.
117. **Park MS**, **Jeong SY**, **Yoon SJ**. Endoscopic Sciatic Nerve Decompression After Fracture or Reconstructive Surgery of the Acetabulum in Comparison With Endoscopic Treatments in Idiopathic Deep Gluteal Syndrome. *Clin J Sport Med*. 2019;29(3):203–208.

Author information:
J. W. Park, MD, Clinical and Research Fellow
Y-K. Lee, MD, PhD, Associate Professor
Department of Orthopedic Surgery, Seoul National University Bundang HospitalSeongnam, South Korea.

Y. J. Lee, MD, PhD, Professor, Department of Internal Medicine, Seoul National University Bundang Hospital, Seongnam, South Korea; Department of Internal Medicine, Seoul National University College of Medicine, Seoul, South KoreaSeongnam, South Korea.

S. Shin, MD, Clinical Fellow, Department of Internal Medicine, Seoul National University Bundang HospitalSeongnam, South Korea.

Y. Kang, MD, PhD, Assistant Professor, Department of Radiology, Seoul National University Bundang HospitalSeongnam, South Korea.

K-H. Koo, MD, PhD, Professor, Department of Orthopedic Surgery, Seoul National University Bundang Hospital, Department of Orthopedic Surgery, Seoul National University College of Medicine Seongnam, South Korea.

Author contributions:
J. W. Park: Reviewed the literature, Wrote the manuscript.
Y-K.Lee: Conceived the study, Reviewed the literature review, Edited the manuscript.
Y. J. Lee: Supervised the study, Wrote and edited the manuscript.
S. Shin: Designed the figures, Wrote and Edited the manuscript.
Y. Kang: Conceived the study, Edited the manuscript.
K-H.Koo: Conceived the study, Edited the manuscript.

Funding statement:
No external funding was received for this work. No benefits in any form have been received or will be received from a commercial party related directly or indirectly to the subject of this article.

This article was primary edited by J. Scott.