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REVIEW

THE DIAGNOSIS OF DEEP GLUTEAL SYNDROME: A SYSTEMATIC REVIEW OF THE CURRENT LITERATURE

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LEVEL OF EVIDENCE: 4

ABSTRACT

Background: Deep gluteal syndrome (DGS) is an increasingly recognized cause of posterior hip pain, particularly in athletes and physically act give individuals. It results from entrapment of neural structures within the deep gluteal space, frequently associated with musculotendinous imbalance, repetitive hip movements, and sport-specific biomechanical stress.

Purpose: To systematically describe the musculotendinous and neural entrapment mechanisms underlying DGS, with particular focus on clinical presentation, imaging findings, and management strategies in athletes. Furthermore, a secondary aim was to evaluate the frequency and clinical relevance of DGS-related conditions reported in athletes.

Methods: A systematic descriptive review was conducted in accordance with PRISMA guidelines. PubMed, Scopus, PEDro, and the Cochrane Library were searched. Studies addressing anatomical predispositions, clinical features, imaging characteristics, and conservative or surgical treatment were included.

Results: Eighty-four studies met the inclusion criteria. DGS encompasses multiple entrapment patterns involving the sciatic, pudendal, posterior femoral cutaneous and superior gluteal nerves. In athletes, repetitive hip flexion–adduction–internal rotation movements, overuse microtrauma and hypertrophic musculature appear to contribute to neural compression. Clinical diagnosis remains challenging due to symptom overlap with lumbar disorders. MRI represents the primary imaging modality, although findings may be subtle or nonspecific. Initial management is conservative, while endoscopic decompression may be indicated in refractory cases.

Conclusion: There are seven clinical conditions that may cause DGS: ischiofemoral impingement, piriformis syndrome, gemelli-internal obturator syndrome, posterior femoral cutaneous nerve entrapment, superior gluteal nerve entrapment, pudendal neuropathy, and proximal hamstring syndrome. Therefore, DGS is an "umbrella term" that encompasses different pathologies resulting in very similar clinical presentations. In the athletic population, the two most frequent conditions are pudendal neuropathy and proximal hamstring syndrome.

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KEYWORDS
Ischiofemoral impingement, piriformis syndrome, posterior femoral cutaneous nerve entrapment, superior gluteal nerve entrapment, pudendal neuropathy.

INTRODUCTION

Back pain localized in the buttock, hip, or posterior thigh is often caused by extrapelvic compression of the sciatic nerve (SN). Historically, this clinical condition was defined as "piriformis syndrome". Indeed, it was commonly believed that the piriformis muscle was the only anatomical structure that could be responsible for the compression of the SN inside the so-called "deep gluteal space" (DGSp)[1]. In recent years, increasing anatomical knowledge together with advances in imaging techniques, particularly magnetic resonance imaging (MRI), have highlighted several additional causes of sciatic nerve entrapment and soft tissue disorders within the DGSp. Besides the piriformis muscle, irritation or entrapment of the SN may result from bony abnormalities, fibrous or fibrovascular bands, the gemelli-obturator internus muscle complex, hamstring tendons, vascular anomalies, ischiofemoral conflict, and benign or malignant neoplasms[2]. Furthermore, involvement of other neural structures within the DGSp — including the posterior femoral cutaneous nerve[3], pudendal nerve, and superior gluteal nerve — as well as pathological alterations of the surrounding soft tissues, may produce a very similar clinical presentation[4,5]. For this reason, McCrory et al.[6] introduced the term "deep gluteal syndrome" (DGS), referring to a non-discogenic sciatic nerve entrapment syndrome caused by compression or mechanical irritation of neural and soft tissue structures within the DGSP. Current evidence therefore supports the concept of DGS as an "umbrella term" encompassing several distinct clinical entities characterized by overlapping symptoms and frequently challenging differential diagnosis. The aim of this systematic descriptive review was to summarize the current evidence regarding the anatomical, etiopathogenetic, clinical, and imaging aspects of DGS. A secondary objective was to evaluate the frequency and clinical relevance of DGS-related conditions reported in the athletic population.

MATERIALS AND METHODS

Study design

This systematic descriptive review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines[7]. The review protocol was registered in the International Prospective Register of Systematic Reviews (PROSPERO) under registration number 1108092.

Literature search strategy

A systematic literature search was conducted using the following electronic databases: PubMed/MEDLINE, Scopus, Web of Science, PEDro, and the Cochrane Database of Systematic Reviews. The literature search was performed between 1 November 2025 and 20 December 2025. No restrictions regarding publication date or language were applied.

The following keywords were used in different Boolean combinations:

"deep gluteal syndrome", "piriformis syndrome", "sciatic nerve entrapment", "sciatic nerve neuralgia", "pudendal nerve entrapment", "pudendal nerve neuralgia", "posterior femoral cutaneous nerve entrapment", "posterior femoral cutaneous nerve neuralgia", "superior gluteal nerve entrapment", "superior gluteal nerve neuralgia", and "deep gluteal space anatomy".

The principal search strategy was structured as follows:

(deep gluteal syndrome OR piriformis syndrome) AND (sciatic nerve entrapment OR sciatic nerve neuralgia) AND (pudendal nerve entrapment OR pudendal nerve neuralgia) AND (posterior femoral cutaneous nerve entrapment OR posterior femoral cutaneous nerve neuralgia) AND (superior gluteal nerve entrapment OR superior gluteal nerve neuralgia) AND (deep gluteal space anatomy).

Eligibility criteria

Eligibility criteria were defined according to the objectives of this systematic descriptive review, aimed at mapping the anatomical, clinical, imaging, and therapeutic spectrum of DGS and related conditions. Given the heterogeneity and limited availability of high-level evidence in this field, studies were selected to provide a comprehensive synthesis of the current literature, including both primary and secondary sources.

Inclusion criteria

The following types of studies were included:

- Randomized controlled trials.
- Cohort studies (prospective and retrospective).
- Case series and case reports.
- Anatomical and imaging studies.
- Systematic reviews (when providing relevant synthesis of DGS-related conditions).
- Narrative reviews were included only when they provided essential anatomical or clinical synthesis not otherwise available in primary studies.

Exclusion criteria:

- Studies focused on gluteal pain without specific reference to DGS.

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- Studies not specifically addressing the DGSp.
- Articles lacking relevant anatomical, clinical, imaging, or therapeutic information concerning DGS.
- Non-scientific publications and grey literature.

Study selection

Three independent reviewers (GN.B.. A.B., and DP.F.) screened titles and abstracts identified through the database search. Full-text articles considered potentially eligible were subsequently assessed independently by the same reviewers.

Disagreements regarding study inclusion were resolved by discussion and consensus among the reviewers. The study selection process is summarized in the PRISMA flow diagram shown in *Figure 1*.

Data extraction and quality assessment

The following data were extracted from the included studies:

- Study design.
- Level of evidence.
- Anatomical findings.
- Clinical presentation.
- Imaging characteristics.
- Conservative treatment strategies.
- Surgical treatment strategies.
- Clinical outcomes.

Methodological quality and risk of bias were assessed using the Joanna Briggs Institute (JBI) critical appraisal tools according to the specific study design[8].

Studies fulfilling ≥75% of the appraisal criteria were considered at low risk of bias, studies fulfilling 60–74% of the criteria were considered at moderate risk of bias, and studies fulfilling <60% of the criteria were considered at high risk of bias.

The methodological quality assessment results are summarized in *Table 1*.

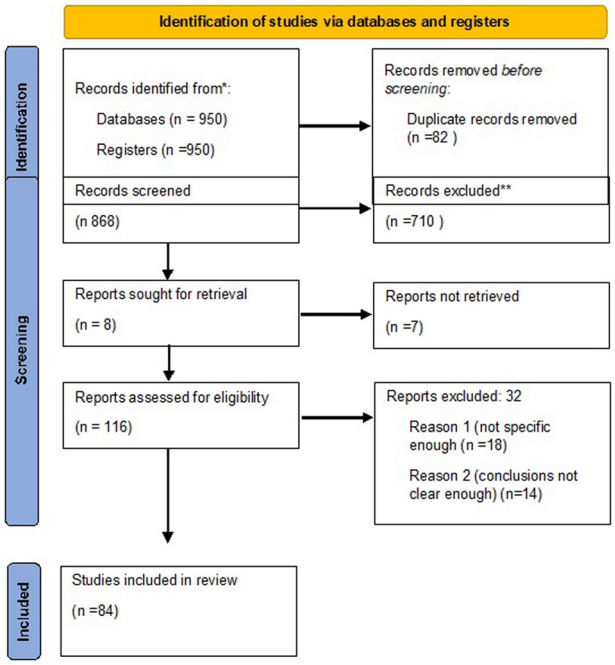


Figure 1. *PRISMA flow diagram summarizing the study identification, screening, eligibility assessment, and inclusion process adopted for the present systematic descriptive review. The figure illustrates the sequential selection procedure, including duplicate removal, full-text assessment, exclusion process, and final study inclusion according to PRISMA reporting standards.*

Data synthesis and statistical analysis

Due to the marked heterogeneity in study designs, populations, diagnostic criteria, and outcome measures, a quantitative synthesis (meta-analysis) was not performed. Instead, a structured narrative synthesis was conducted following established methodological guidance for systematic reviews when statistical pooling is not appropriate[8-9]. Studies were grouped according to pathology, anatomical region, and type of intervention to allow for comparative qualitative analysis.

Results of the systematic descriptive review

After screening 950 articles, 84 studies met the eligibility criteria and were included in this review *(Fig. 1)*. *Table 1* summarizes the study design, level of evidence, JBI score, risk of bias, and main findings of the included studies.

RESULTS

Qualitative synthesis of included studies

A total of 84 studies were included in the systematic descriptive review. The distribution of study designs is as follows: 28 case series (33.3%), 23 narrative reviews (27.4%), 13 systematic reviews (15.5%), 13 case reports (15.5%), and 7 primary clinical studies including randomized controlled trials, retrospective studies, and meta-analyses (8.3%).

Regarding methodological quality, most primary studies demonstrated low risk of bias according to the JBI appraisal tools.

When stratified by condition, the available evidence was distributed as follows:

- Ischiofemoral impingement syndrome: the most extensively studied entity, supported by multiple case series, systematic reviews, and imaging-based studies, with relatively higher diagnostic consistency on MRI.
- Piriformis syndrome: supported mainly by case series and narrative reviews, with limited high-level evidence and significant heterogeneity in diagnostic criteria.
- Pudendal neuropathy: supported by a combination of clinical studies, systematic reviews, and anatomical descriptions, with strong reliance on clinical diagnostic criteria (Nantes criteria).
- Proximal hamstring syndrome: supported by clinical studies and systematic reviews, with moderate evidence strength and relatively consistent imaging correlation.
- Posterior femoral cutaneous nerve entrapment:

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Reference	Study design	Level of evidence	JBI score	Risk of bias	Synthesis of the study
Meknas et al., 2009[56]	Randomized clinical trial	I	90/100	Low	Surgical release of the internal obturator tendon in 12 patients affected by trochanteric pain syndrome
Robert et al., 2005[67]	Randomized clinical trial	I	89/100	Low	Description of decompression and transposition procedure of the pudendal nerve in patients suffering from pudendal neuralgia
Labat et al., 2017[66]	Randomized double-blind controlled trial	I	90/100	Low	The use of corticosteroids during nerve block for pudendal neuralgia
Singer et al., 2015[31]	Meta-analysis	I	91/100	Low	A meta-analysis of ischiofemoral impingement syndrome
Smoll, 2010[47]	Meta-analysis	I	90/100		The clinical consequences collating anatomical variations in the sciatic nerve path in correspondence to the piriformis muscle
Park et al., 2020[5]	Systematic review	I	90/100	Low	The DGS as a cause of posterior hip non-discogenic pain
Kizaki et al., 2020[10]	Systematic review	I	78/100	Low	Sciatic nerve entrapment in the DGSp and description of non-discogenic sciatic nerve disorder
Gollwitzer et al., 2017[14]	Systematic review	I	66/100	Moderate	Treatment algorithm of ischiofemoral impingement and review of the literature.
Nakano et al., 2020[21]	Systematic review	I	81/100	Low	Treatment strategies for ischiofemoral impingement
Bisciotti et al., 2024[27]	Systematic review	I	90/100	Low	Groin Pain Syndrome Italian Consensus Conference update 2023
Barbosa et al., 2019[48]	Systematic review	I	90/100	Low	Anatomic variation of SN and the possible association with piriformis syndrome
Fall et al., 2004[65]	Systematic review	I	89/100	Low	Guidelines on chronic pelvic pain of European Association of Urology
Hopayian et al., 2022[74]	Systematic review	I	78/100	Low	Systematic review of conservative and surgical treatments for DGS
Leibovitch et al., 2005[75]	Systematic review	I	90/100	Low	Bicycling related urogenital disorders
Labat et al., 2008[77]	Systematic review	I	65/100	Moderate	Diagnostic criteria (Nantes criteria) for pudendal neuralgia
Andiman et al., 2004[79]	Systematic review	I	81/100	Low	The pain therapy of pudendal neuralgia
Bisciotti et al., 2019[83]	Systematic review	I	75/100	Low	Account of “return to play decision making process” according to the guidelines of the Italian consensus statement on return to play after lower limb muscle injury in football
Rhim et al., 2024[84]	Systematic review	I	74/100	Moderate	The use of extracorporeal shockwave therapies in athletic population
Bharadwaj et al., 2023[12]	Retrospective single center study	III	76/100	Low	Variant sciatic nerve anatomy in relation to the piriformis muscle on magnetic resonance neurography
Jennette et al., 2022[3]	Case series	IV	86/100	Low	Persistent neuropathy caused by unidentified branches of the posterior femoral cutaneous nerve
Johnson, 1977[15]	Case series	IV	71/100	Moderate	Account of three cases of impingement of the lesser trochanter on the ischial ramus following total hip arthroplasty.
Miller and Webb, 2008[18]	Case series	IV	72/100	Moderate	The proximal origin of the hamstrings and surrounding anatomy.
Torriani et al., 2009[22]	Case series	IV	79/100	Low	The MRI abnormality of the QFM in 12 patients affected by ischiofemoral impingement syndrome
Tosun et al., 2012[23]	Case series	IV	71/100	Moderate	MRI ischiofemoral impingement: evaluation
Gómez-Hoyos et al., 2016[26]	Case series	IV	78/100	Low	Description of clinical semeiotic tests in patients with posterior hip pain
Kivlan et al., 2017[30]	Case series	IV	90/100	Low	Anatomical study of the lesser trochanter-ischial space.
Jo and O'Donnell, 2015[40]	Case series	IV	90/100	Low	Surgical description of the endoscopic resection of the lesser trochanter in patients with ischiofemoral impingement
Hatem et al., 2015[42]	Case series	IV	89/100	Low	Outcome of the two years follow-up after endoscopic intervention for ischiofemoral impingement.
Howse et al., 2014[43]	Case series	IV	74/100	Moderate	Description of the surgical decompression of the IFS through postero-lateral approach
Wilson and Keene, 2016[44]	Case series	IV	80/100	Low	The outcomes of diagnostic injections and arthroscopic decompression of the lesser trochanter in a series of patients affected by IFIS:
Hernandez et al., 2017[45]	Case series	IV	75/100	Low	The outcomes of endoscopic decompression of the lesser trochanter in two patients affected by IFIS
Ilizaliturri and Camacho-Galindo, 2010[46]	Case series	IV	81/100	Low	Description of two different endoscopic techniques for internal snapping hip.

Reference	Study design	Level of evidence	JBI score	Risk of bias	Synthesis of the study
Al-Al-Shaikh et al., 2015[49]	Case series	IV	77/100	Low	MRI evaluation of piriformis muscle morphology changes induced by botulinum toxin injections in a series of 12 patients affected by piriformis syndrome.
Russell et al., 2008[50]	Case series	IV	73/100	Moderate	The MRI assessment of the sacral plexus and piriformis muscles
Lewis et al., 2006[51]	Case series	IV	85/100	Low	The role of magnetic resonance neurography in extraspinal sciatica
Jeong et al., 2015[52]	Case series	IV	89/100	Low	The clinical outcomes of ultrasound-guided steroid injections in 63 patients affected by piriformis syndrome
Fishman et al., 2002[53]	Case series	IV	84/100	Low	The diagnosis, treatment, and outcome in a series of 918 patients affected by piriformis syndrome with a 10-year follow-up
Indrekvam and Sudmann, 2002[54]	Case series	IV	90/100	Low	A series of 19 patients affected by piriformis muscle syndrome treated by tenotomy with a follow-up between 1 to 16 years.
Ilizaliturri et al., 2018[55]	Case series	IV	72/100	Moderate	Piriformis tendon endoscopic release procedure in a series of 15 patients affected by DGS
Darnis et al., 2008[59]	Case series	IV	65/100	Moderate	Anatomy and surgery of the inferior cluneal nerves
Ploteau et al., 2017[61]	Case series	IV	80/100	Low	Description of the anatomical variants of the pudendal nerve in a series of patients affected by pudendal neuralgia.
Ploteau et al., 2017[62]	Case series	IV	65/100	Moderate	Anatomical basis of inferior cluneal nerve neuralgia and surgery procedure
Ozsoy et al., 2007[68]	Case series	IV	67/100	Moderate	Anatomical predispositions for lesions of the superior gluteal nerve and gluteus medius muscle
Plihal et al., 1989[72]	Case series	IV	77/100	Low	Etiopathogenesis of superior gluteal nerve entrapment neuropathy and relative conservative treatment and surgery
Murer et al., 2023[76]	Case series	IV	80/100	Low	The risk of pudendal neuralgia in horse riding
Habib et al., 2023[80]	Case series	IV	85/100	Low	Nerve decompression in two patients affected by pudendal neuralgia
Martin et al., 2018[85]	Case series	IV	83/100	Low	The accuracy of 3 clinical semeiotic maneuvers for the diagnosis of proximal hamstring injuries
Coppola et al., 2024[82]	Retrospective case series	IV	82/100	Low	Magnetic resonance imaging as predictor for the days lost from training and competition in professional football players
Patti et al., 2008[17]	Case report	IV	80/100	Low	Description of the clinical framework caused by the impingement of the lesser trochanter onto the ischium
Tosun et al., 2012[23]	Case report	IV	80/100	Low	Description of the clinic cases of 11 female patients suffering from IFIS
Corrales et al., 2018[32]	Case report	IV	81/100	Low	Endoscopic lesser trochanter decompression and in a patient affected by IFIS.
Papoutsi et al., 2016[33]	Case report	IV	82/100	Low	Description of one case of ischiofemoral impingement due to a lipoma located in the IFS
Viala et al., 2012[34]	Case report	IV	78/100	Low	IFIS due to multiple exostoses.
Schatteman et al., 2015[35]	Case report	IV	79/100	Low	IFIS due to a solitary exostosis
Lee et al., 2013[36]	Case report	IV	81/100	Low	Description of a case of IFIS in a 48-year-old female patient
Ali et al., 2011[37]	Case report	IV	78/100	Low	Description of surgical treatment of a snapping hip coexisting with IFIS
Chen and Jenkins, 2018[38]	Case report	IV	81/100	Low	IFIS treated with botulinum toxin
Kim et al., 2014[39]	Case report	IV	82/100	Low	The ultrasound-guided prolotherapy with polydeoxyribo nucleotide sodium in IFIS
Safran and Ryu 2014[41]	Case report	IV	78/100	Low	IFIS surgically treated in a 19-year-old female
Cox and Bakkum, 2005[58]	Case report	IV	79/100	Low	Gemelli-obturator internus complex syndrome in a patient with deep persistent aching pain in the retro-trochanteric region
Rask, 1980[69]	Case report	IV	77/100	Low	Surgical treatment in a patient affected by entrapment of the superior gluteal nerve can occur as a result of compression by anterior-superior tendinous fibers of the piriformis muscle
Probst et al., 2019[1]	Narrative review	V	Not applicable	Not applicable	Anatomy, diagnosis, and treatment of piriformis syndrome
Geler Külcü, 2024[2]	Narrative review	V	Not applicable	Not applicable	Description of the clinical situation caused by DGS
Hernando et al., 2015[4]	Narrative review	V	Not applicable	Not applicable	The sciatic nerve entrapments in the DGSp anatomy, imaging, and management
McCrory and Bell, 1999[6]	Narrative review	V	Not applicable	Not applicable	Anatomical basis of the nerve entrapment syndromes

Reference	Study design	Level of evidence	JBI score	Risk of bias	Synthesis of the study
Carro et al., 2016[11]	Narrative review	V	Not applicable	Not applicable	Anatomical description and surgical approach to piriformis syndrome and IFIS
Taneja et al., 2013[13]	Narrative review	V	Not applicable	Not applicable	Anatomical and clinical concepts of IFIS
Kassarjian, 200[8][16]	Narrative review	V	Not applicable	Not applicable	Interpretation of MRI signal abnormalities in the quadratus femoris muscle
Martin et al., 2015[19]	Narrative review	V	Not applicable	Not applicable	Clinical assessment of DGS
Martin et al., 2016[20]	Narrative review	V	Not applicable	Not applicable	Hamstring syndrome and sciatic nerve impingement as causes of posterior hip pain
Nakano et al., 2107[24]	Narrative review	V	Not applicable	Not applicable	The extra-articular hip impingement syndromes: diagnosis and management
Stafford and Villar, 2011[25]	Narrative review	V	Not applicable	Not applicable	Anatomical concepts and clinical assessment of IFIS
O'Brien and Bui-Mansfield, 2007[28]	Narrative review	V	Not applicable	Not applicable	Quadratus femoris muscle tears cause of hip pain
Kassarjian et al., 2011[29]	Narrative review	V	Not applicable	Not applicable	Anatomic considerations and pathologic injuries of the quadratus femoris muscle
Miller et al., 2012[57]	Narrative review	V	Not applicable	Not applicable	Diagnosis and management of piriformis syndrome
Drăghici et al., 2024[60]	Narrative review	V	Not applicable	Not applicable	Anatomical and clinical concepts concerning the rare entrapment neuropathies of the lower extremity
Isu et al., 2018[63]	Narrative review	V	Not applicable	Not applicable	Clinical assessment and anatomical concepts of superior and middle cluneal nerve entrapment
Jottard et al., 2022[64]	Narrative review	V	Not applicable	Not applicable	Description of the diagnosis and treatment of pudendal and inferior cluneal nerve entrapment syndrome
Petchprapa et al., 2010[70]	Narrative review	V	Not applicable	Not applicable	MR imaging of entrapment neuropathies of the pelvis and hip
Buxton and Dominick, 2006[71]	Narrative review	V	Not applicable	Not applicable	The use of electromyography data of the lower extremity
Lo et al., 2022[73]	Narrative review	V	Not applicable	Not applicable	Anatomical description of nerve entrapments in the pelvis
Schmid et al., 2013[78]	Narrative review	V	Not applicable	Not applicable	Mechanisms, diagnosis and management of the entrapment neuropathies
Bisciotti et al., 2020[81]	Narrative review	V	Not applicable	Not applicable	The hamstring injuries prevention in football
Gonzalez-Lomas, 2021[86]	Narrative review	V	Not applicable	Not applicable	The clinical assessment of the pain in the DGSp

Table 1. *Overview of the analyzed literature.*

Regarding the risk of bias reference indices, if the studies satisfied ≥75% of the requested criteria, the risk of bias was considered low; if the criteria were satisfied by a percentage 60-74%, the risk of bias was considered moderate; finally, if the criteria were satisfied by <60%, the risk of bias was considered high[8].

primarily based on anatomical and case series data, with limited high-level evidence.

- Superior gluteal nerve entrapment: mainly supported by anatomical studies and case reports, with low overall evidence strength.
- Gemelli–obturator internus syndrome: represents the least studied condition, with very limited clinical evidence and predominantly case-based literature.

Overall, the evidence base for DGS remains heterogeneous and predominantly composed of low-to-moderate level studies, with a limited number of randomized controlled trials.

To improve interpretability of the available evidence, findings were interpreted according to study design and methodological quality. Greater weight was assigned to primary clinical studies, randomized controlled trials, and systematic reviews with low risk of bias, whereas conclusions derived mainly from case reports, narrative reviews, and anatomical descriptions were interpreted cautiously and considered hypothesis-generating.

Deep gluteal space anatomy

The posterior extension of the peritrochanteric space into the gluteus maximus muscle is known as the DGSp and is defined anatomically by an anterior border formed by the posterior acetabular column, a posterior border marked out by the gluteus maximus, a medial border fashioned by the sacrotuberous ligament, a lateral border shaped from the gluteal tuberosity, a superior border represented by the sciatic notch and an inferior border coinciding with the ischial tuberosity[4,10]. The muscles contained within the DGSp include the piriformis, the internal and external obturators, the gemelli (superior and inferior), the quadratus femoris, and the origins of the hamstrings. Furthermore, the sacrotuberous and sacrospinous ligaments, the origin of the ischiofemoral

ligament, the lateral ascending branches of the medial femoral circumflex artery, and the superior and inferior gluteal arteries can all be found within the DGSp. Seven

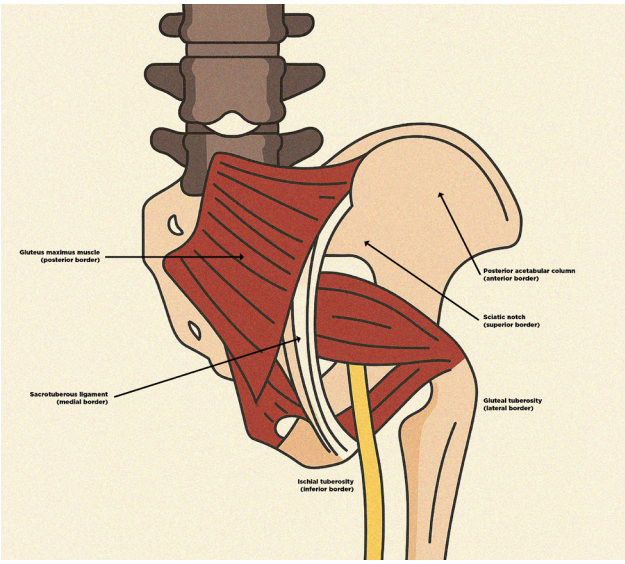


Figure 2. *Schematic anatomical map of the DGSp. The figure summarizes the anatomical relationships between osseous, muscular, and neural structures potentially involved in deep gluteal syndrome and illustrates the principal regions where extraspinal nerve entrapment may occur.*

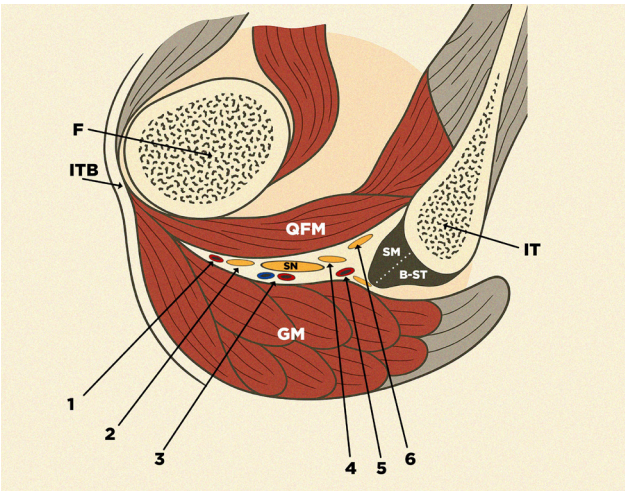


Figure 3. *Axial schematic representation of the principal muscular and neurovascular structures contained within the DGSp. The figure highlights the close anatomical relationships between the sciatic nerve and adjacent muscular, tendinous, and vascular structures that may contribute to dynamic or static nerve entrapment mechanisms described in the included studies.*

Legend: GM, gluteus maximus muscle; QFM, quadratus femoris muscle. SN, sciatic nerve; SM, semimembranosus tendon; B-ST, conjoint tendon of the biceps femoris and semitendinosus muscles; ITB, iliotibial band; F, femoris; 1, ascending circumflex femoral artery; 2, nerve to the short head of the biceps; 3, vasa nervorum of the sciatic nerve; 4, posterior cutaneous nerve of the thigh; 5, inferior gluteal artery and nerve; 6, nerve of the long head of the biceps femoris.

nerves are contained within the DGSp that exit the pelvis through the greater sciatic notch: the sciatic, pudendal, posterior femoral cutaneous, superior and inferior gluteal nerves, the internal obturator/gemellus superior nerve and the quadratus femoris/ gemellus inferior nerve[5,11]. The origin and innervation of these nerves is shown in Table 2. The course of the SN warrants some clarification: it runs distally through the space anterior to the piriformis muscle and posterior to the obturator/gemelli muscle complex and quadratus femoris. However, there are some anatomical variations that are relatively common, including the passage of the SN into the space that either cuts through the piriformis or that lies behind it. These variants have been documented in approximately 17% of cadaver dissection studies[1]. The SN is located, on average, 1.2 ± 0.2 cm from the most lateral aspect of the ischial tuberosity. The SN, along with the inferior gluteal nerve and the posterior femoral cutaneous nerve, descends between the greater trochanter and the ischial tuberosity[1]. Under physiological conditions, the SN is capable of stretching and sliding to accommodate stresses and compressions that occur with hip movement. Indeed, during leg elevation and knee extension, the SN undergoes a proximal excursion of approximately 28 mm medially towards the hip joint[12]. The most important muscular and neurovascular structures within the DGSp are schematically shown in *Figures 2 and 3*, while a schematic view of the DGSp is shown in *Figure 4*.

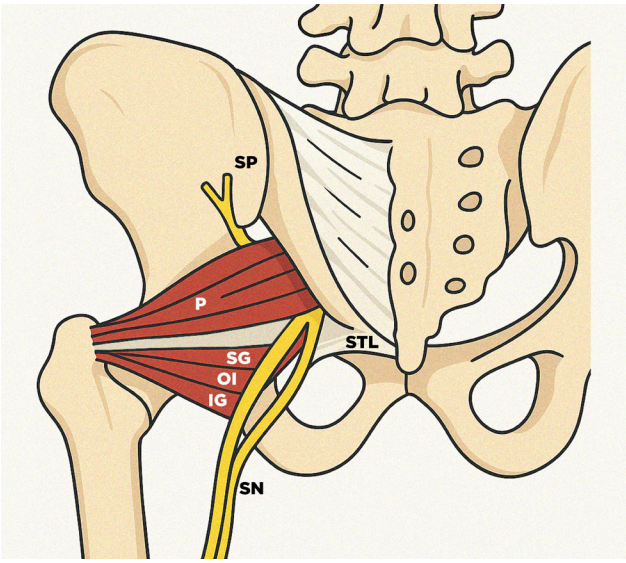


Figure 4. *Coronal schematic view of the principal muscular, ligamentous, neural, and osseous structures of the DGSp. The figure illustrates the spatial relationships among the sacral plexus, sciatic nerve, piriformis muscle, obturator internus-gemelli complex, and sacrotuberous ligament, which represent the anatomical basis of several deep gluteal entrapment syndromes discussed in the present study.*

Legend: SP, sacral plexus; SN, sciatic nerve; STL, sacrotuberous ligament; P, piriformis muscle; SG, superior gemellus muscle; OI, obturator internus muscle; IG, inferior gemellus muscle.

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Neural structure	Origin	Innervation
Sciatic nerve	L4-L5-S1-S2-S3	Motor innervation of hamstring, extensor portion of adductor magnus, all the muscles of the leg and foot. Sensory innervation of the leg and foot except the saphenous nerve dermatome
Posterior femorocutaneous nerve	S1-S2-S3	Sensory innervation of the gluteal region, perineum, posterior thigh and popliteal fossa
Superior gluteal nerve	L4-L5-S1	Motor innervation of gluteus medius, gluteus minimus and tensor fascia lata
Inferior gluteal nerve	L5-S1-S2	Motor innervation of gluteus maximus
Pudendal nerve	S2- S3-S4	Motor innervation of the musculus transversus perinei superficialis, musculus ischiocavernosus, musculus bulbospongiosus. Sensory innervation of perineum and external genitalia
Internal obturator/ gemellus superior nerve	L5-S1-S2	Motor innervation of obturator internus and superior gemellus
Quadratus femoris/ gemellus inferior nerve	L4-L5-S1	Motor innervation of quadratus femoris and inferior gemellus. Sensory innervation of the hip capsule

Table 2. *The origin and innervation of the neural structures of the DGS.*

Deep gluteal syndrome definition

Current literature describes the DGS as a non-discogenic painful condition caused by compression of neural or soft tissue structures within the DGSp. Although no universally accepted classification system currently exists, the available literature suggests that DGS may encompass several distinct but clinically overlapping conditions, including ischiofemoral impingement syndrome, piriformis syndrome, gemelli–obturator internus syndrome, posterior femoral cutaneous nerve entrapment, superior gluteal nerve entrapment, pudendal neuropathy, and proximal hamstring syndrome[5,11].

Therefore, the concept of DGS as an "umbrella term" should be interpreted as a descriptive clinical framework derived from the current anatomical and clinical literature rather than as a formally standardized diagnostic classification. Moreover, substantial overlap exists between DGS-related disorders and lumbar spine, pelvic, and extra-articular hip pathologies, often making differential diagnosis particularly challenging.

The ischiofemoral impingement (IFIS)

Ischiofemoral impingement syndrome (IFIS) is a clinical condition with an etiopathogenesis originating from the excessive narrowing of the anatomical space between the ischial tuberosity and the lesser trochanter, aka "ischiofemoral space" (IFS). An extreme tightening of this IFS can entrap the soft tissues found there, specifically the quadratus femoris muscle, and compress the SN[13,14]. Johnson[15] first described IFIS and its surgical treatment after observing a narrowing of the IFS in three patients complaining of persistent hip pain following surgery: —two cases arose after total hip arthroplasty and one arose following proximal femoral osteotomy. These initial cases were successfully treated with resection of the lesser trochanter. Later, other authors described several clinical cases of hip pain related to a narrowing of the IFS in patients without a history of surgery or trauma[16,17]. These findings are a wake up call for clinicians, orthopedic surgeons, and radiologists to pay due consideration to IFIS.

Indeed, IFIS diagnosis must be based on both physical examination and radiologic assessment; worthy of note is the fact that the imaging available is almost entirely that of MRI scans[16,17]. Among DGS-related disorders, IFIS is supported by the most consistent imaging and clinical evidence, including multiple case series, systematic reviews, and imaging-based studies[13-47].

Anatomy

The quadratus femoris muscle (QFM) is a flat, quadrilateral muscle that runs inferior and parallel to the inferior gemellus muscle and is part of the muscle group of short external rotators positioned below it. This muscle originates from the ischial tuberosity and inserts distally onto the intertrochanteric crest of the femur; it is innervated by the nerve to the quadratus femoris and is vascularized by the medial circumflex femoral artery. The principal function of the QFM is the external rotation of the hip but this muscle also serves as an accessory adductor[18,19]. As far as the etiopathogenesis of IFIS is concerned, it is important to remember that, on leaving the pelvic region, the SN runs posteriorly to the obturator, the gemelli, and the QFM. In this stretch of its course, the SN passes along the most lateral aspect of the ischial tuberosity as shown in *Figure 5*. Within the IFS, the QFM separates the SN from the lesser trochanter[18,19], and any narrowing of the IFS could cause compression of both the QFM and the SN and thereby leading to sciatic neuropathy[20]. Furthermore, since the SN runs between the anterior surface of the gluteus maximus and the posterior surface of the QFM, any injury or inflammation involving the QFM could also lead to SN neuropathy[21].

Etiopathogenesis

The narrowing of the IFS can be classified into three types: 1) positional, 2) congenital, and 3) acquired.

Positional IFS narrowing

Factors affecting the IFS and QFM include: internal/external rotation of the lower limb, adduction/abduction, and flexion/extension movements[21].

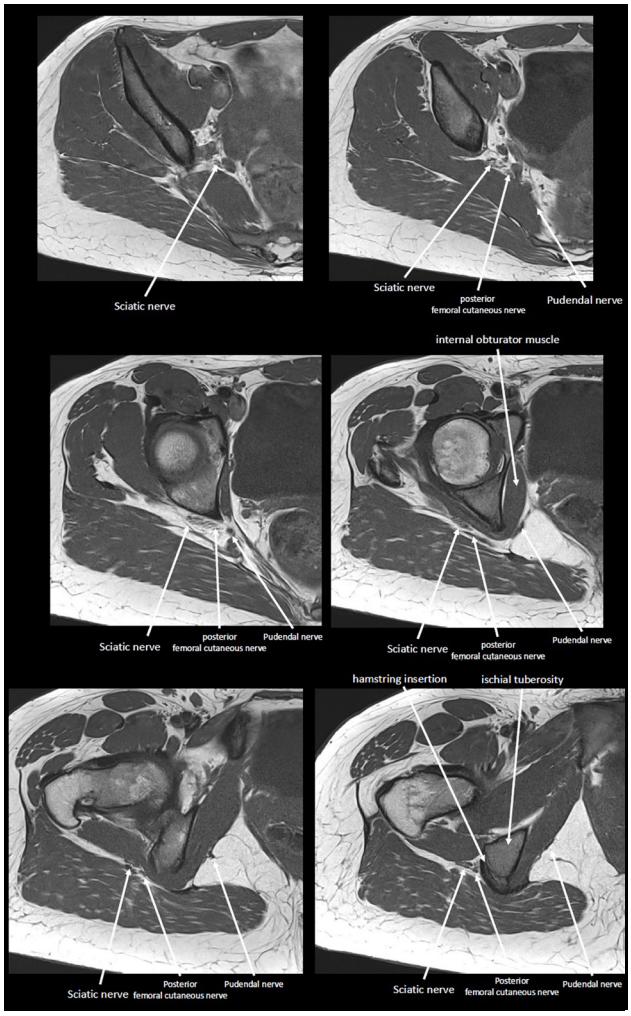


Figure 5. *Axial T1-weighted magnetic resonance imaging (MRI) demonstrating the cranio-caudal course of the sciatic nerve, pudendal nerve, and posterior femoral cutaneous nerve within the DGSp The figure provides an imaging correlation of the neural anatomy potentially involved in DGS and highlights the anatomical complexity underlying differential diagnosis of extraspinal neuropathic pain.*

Congenital IFS narrowing

A congenital IFS narrowing can be caused by[22-25]:

A larger cross-section of the femur at the level of the lesser trochanter.

- A congenital posteromedial position of the femur
- A lower ischiopubic ramus with an angle closer to the coronal plane.
- A prominence of the lesser trochanter.
- Coxa profunda
- Coxa valga
- A configuration of the pelvic bony anatomy in females that presents a greater width and smaller antero-posterior dimensions compared to that in males.

Acquired IFS narrowing

An acquired IFI narrowing may be caused by[15,17,21]:

- Fractures involving the lesser trochanter.
- Valgus-producing intertrochanteric osteotomy.
- Osteoarthritis leading to superior and medial migration of the femur in older patients,
- Enthesopathy of the proximal hamstring insertion.
- Legg–Calve–Perthes disease.
- Abductor muscle injury causing uncompensated hip adduction during gait
- Expansive bone lesions.
- Multiple or isolated exostosis.

Clinical presentation

Patients affected by IFIS complain of deep inguinal or gluteal pain, with no recollection of past injuries[26]. The pain radiates distally to the lower extremity, probably because of pressure on the SN[17,2]. The clinical framework was also related to other symptoms such as snapping, crepitation, or locking[23-24]. Gait analysis revealed that the patient experienced pain when walking in long strides, whereas with shorter strides the pain was alleviated[25]. There is no specific diagnostic clinical test for IFIS, as the pain may recur when the hip is extended, in adduction and/or undergoes external rotation[26]. Therefore, the clinical criteria for IFIS diagnosis ought to aim at provoking an impingement in extension with a neutral or adducted hip to re-create the posterior pain lateral to the ischium, and then relieve the pain in extension with an abducted hip with a subsequent abduction[27]. A diagnostic block can be extremely useful to confirm the clinical diagnosis[28].

The differential diagnosis includes[29,30]:

- QFM indirect injury without IFS narrowing.
- QFM denervation.
- Iliopsoas tendinopathy.
- Hamstring tendinopathy (hamstring syndrome).
- Iliopsoas bursitis.
- Delayed-onset muscle soreness.
- Mass lesions.

Imaging

In IFIS, the hip plain radiographs are usually normal, although sometimes it is possible to observe chronic osseous changes such as sclerosis and cystic changes of the lesser trochanter and ischium. However, plain radiographs are useful for diagnosing osseous abnormalities that cause acquired IFI or other forms of hip pain. Hip pain radiographs should include standard antero-posterior view and frog- view[17,30]. MRI is the standard method for diagnosing IFIS and a standard MRI protocol is sufficient for detecting and evaluating IFIS. The MRI imaging protocol for the pelvis requires the feet to be rotated internally and secured by adhesive tape. Pelvic MRI imaging should be carried out on a 1.5 or 3.0 T scanner equipped with a selection of phased array coils that allow visualization of both hip joints. The recommended pulse sequences are: coronal T1-weighted, axial fast spin echo (FSE), proton density-weighted axial FSE, fat-suppressed T2-weighted, coronal FSE short-tau inversion recovery and sagittal FSE fat-suppressed T2-weighted. Sequences requiring the use of contrast medium are not necessary. Whereas the sequences most appropriate for assessing

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IFIS are axial sequences, both coronal and sagittal sequences can provide additional information about the QFM and surrounding anatomical structures[13]. IFIS is characterized by a narrowing of the IFS and a consequent edema of the QFM belly[21] and is generally unilateral[13] although IFIS manifestation may be bilateral in 25-40 % of cases[25]. In addition to the IFS, another anatomical space useful for the diagnosis of IFIS is the quadratus femoris space (QFS)[21]. A narrowing of the IFS and the QFS can be evaluated *(Fig. 6)* from the measurements obtained from a MRI T1 axial view[21], specifically:

- The IFS is the narrowest distance between the lateral cortex of the ischial tuberosity and medial cortex of the lesser trochanter;
- The QFS is anatomically the narrowest space for passage of the QFM delimited by the superolateral surface of the hamstring tendons and the posteromedial surface of the iliopsoas tendon or lesser trochanter.

  During lateral rotation, with the hip in extension and at a 10° adduction, the lesser trochanter draws closer to the ischium, whereas it moves away from the ischium when the hip is in neutral flexion-extension or abduction-adduction[31].The IFS, with the hip in adduction, external rotation and extension, is about 20-28 mm[15,31]. Several studies specifically show that the IFS is, on average, equal to 18.6 mm in females and 23 mm in males and that it decreases by 0.09 mm with each year of age[25]. Other studies show that, in patients affected by IFIS, the IFS and the QFS were respectively 13 ± 5 mm and 7 ± 3 mm[21] with the leg in internal rotation, whereas when the leg was maintained a neutral position, the IFS and QFS were respectively 12.9 mm and 6.71 mm[23,24]. Finally, IFIS can be diagnosed from MRI images that reveal an IFS <15 mm associated with the presence of edema of the QFM[32,33] *(Fig. 6)*.

Additional MRI findings that may be present in patients affected by IFIS are[23,24,29]:

- Edema surrounding the iliopsoas tendon insertion.
- Edema surrounding the hamstring tendons.
- Bursa-like formations.
- Reduced muscle volume and fatty infiltration in patients affected by long-standing IFIS.

In the case of QFM belly edema it is important to differentiate between an edema caused by IFS narrowing and an edema arising from indirect muscle injury. A QFM indirect injury may be caused by a QFM eccentric contraction as the muscle tries to control internal hip rotation[26,29]. Furthermore, it is important to note that some studies report that, during MRI, the QFM muscle signal changes or that symptoms of IFIS could be observed in patients without a true IFS narrowing but due to lipoma[34] or exostosis[35,36].

Conservative treatment

To date, there is no consensus concerning the optimal treatment for IFIS. Nonetheless, patients affected by IFIS benefit from conservative treatment based on rest, activity restriction, non-steroidal anti-inflammatory drugs and physical therapy[17,23,24,37]. Several studies report that computed, tomography guided injection of anesthetics and steroids into the QFM is a valuable alternative to surgical intervention in selected patients[21,38]. However, palliative therapy providing only temporary relief of symptoms[21,38] is also an option. One study reports a positive outcome with botulinum toxin type-A infiltration[39], whilst another study describes the successful treatment of IFIS with prolotherapy using polydeoxyribonucleotide sodium[40]. In general, all the studies based on the conservative treatment of IFIS show good short-term results, ranging from 2 weeks to 1 year without any complications.

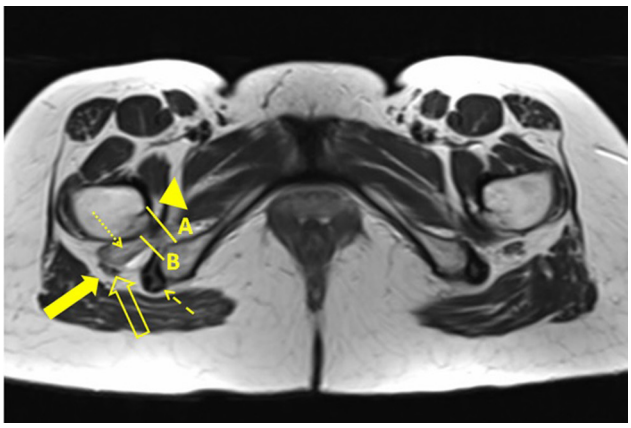


Figure 6. *Axial T2-weighted MRI of the right hip demonstrating imaging findings consistent with ischiofemoral impingement syndrome. The figure shows narrowing of the ischiofemoral space (line A) and quadratus femoris space (line B), associated with edema of the QFM (dotted arrow), supporting the role of mechanical compression as a potential source of DGS and SN irritation described in the reviewed literature.*

Legend: Solid arrow, QFM; open arrow, SN; arrowhead, iliopsoas tendon; dotted line, common tendon of the biceps femoris and semitendinosus muscles.

Surgical treatment

Cases which do not improve with conservative treatments and for which recalcitrant symptoms persist, may require surgical treatment[41]. The excision of the lesser trochanter (LT) may represent an effective treatment by decompressing the IFS and although LT excision is recommended if conservative treatment fails, it is important to remember that this procedure has still to be standardized[33,41]. Johnson[15] described an LT excision, performed in open surgery, in patients suffering from IFIS after total hip arthroplasty. Since this first description by Johnson[15], improvements in arthroscopy techniques and devices have made it possible to access the entire LT endoscopically. Safran and Ryu[41] described an arthroscopic procedure. Compared to open surgery, this endoscopic technique allows for an anatomical decompression with fewer complications[41-47] and less

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soft tissue damage[42] as it is minimally invasive. This technique can be carried out either anteriorly or posteriorly. The endoscopic posterior approach to DGSp allows for easy visualization of the QFM and the SN and facilitates endoscopic osteoplasty of the LT[43], enabling an IFS of at least 17 mm to be obtained and leaving most of the iliopsoas insertion intact. Therefore, the advantage of this technique lies in being less surgically injurious to the iliopsoas tendon[43]. However, it is important to note that this endoscopic posterior approach carries the disadvantage of a greater risk of damage to the SN[33].

Nonetheless, it is important to note that the psoas tendon shows a certain potential for regeneration after its release following endoscopic surgery[47]. For this reason, Jo and O'Donnell[40] described an anterior LT decompression that reduces the risk of damage to the SN. More recently, 58et al.[32] described a surgical procedure for the treatment of IFIS by excising the LT and reinserting the iliopsoas tendon by means of 2 anchorages placed in the footprint after performing a trochanter-plasty. It is important to note that there is a lack of evidence concerning evidence-based recommendations for IFIS surgery in today's literature and, to the best of our knowledge, a systematic descriptive review on this topic has not yet been published[21]. All the studies that focused on the IFS arthroscopic decompression procedure reported a positive outcome at the medium-term follow-up[41-46] whereas, the longer-term outcome of this treatment remains uncertain[41]. Some authors recommend QFM surgical detachment[42]. However, since the medial femoral circumflex arteries run over the upper border of the QFM, it is important to contemplate the risk of avascular necrosis of the femoral head in this procedure[42]. Furthermore, it is important to note that only one study reported data concerning the return to sport after IFS decompression[43]. In this study, Hatem et al.[42] reported an average return to sport period after surgery of 4.4 months (range: 1-7 months).

Piriformis syndrome

Anatomy

The piriformis muscle, in the coronal plane, is located above the superior gemellus muscle and is an important anatomical landmark for identifying the neurovascular structures of the DGSp. The piriformis is an external rotator of the hip; furthermore, it limits the posterior translation of the femoral head with the hip flexed. Hip flexion together with adduction and internal rotation movements stretch the piriformis muscle and cause a narrowing of the space between the lower border of the piriformis, the superior gemellus, and the sacrotuberous ligament. Distally, the piriformis presents: the inferior gluteal artery, the inferior gluteal nerve, the posterior femoral cutaneous nerve, the SN, the internal obturator/superior gemellus nerves, and the quadratus femoris/inferior gemellus nerve *(Fig. 5)*. In literature, 13 anatomical variants of the relationship between the SN and the piriformis muscle have been described[48,49]. The SN passes beneath the belly of the piriformis in 85.2% of subjects. The peroneal division of the SN may perforate the piriformis, while the tibial component passes distally in 9.8% of cases. Under non-pathological conditions the SN elongates and slides and is able to withstand moderate mechanical tension and compression. Several authors have suggested that abnormal anatomical variants, hindering the normal sliding of the SN during the hip joint movements, may be a risk factor for the onset of DGS[48,49].

Etiopathogenesis

Piriformis syndrome may have an etiopathogenesis related to overuse or repeated microtraumas in the gluteal area. Many patients show post-traumatic entrapment of the SN by constrictive fibrous and/or fibrovascular bundles that, starting from the fibers of the piriformis muscle, exert a compressive action on the SN[4].

The potential etiopathogenetic sources of piriformis syndrome can be classified into three categories[11]:

- Hypertrophy of the piriformis muscle (generally asymmetrical) causing anterior displacement of SN.
- Dynamic SN entrapment by the piriformis muscle.
- Anomalous course of SN.

Among DGS-related disorders, the piriformis syndrome is supported by case series and narrative reviews[1,4,5,11,28,48-56]. Therefore, the overall evidence strength is low-moderate.

Clinical presentation

Generally, patients complain of pain which can be either intermittent or persistent, at the level of the buttock, the back of the hip, or the thigh. Sometimes, in the same anatomical areas, there may be dysesthesia. Often, the clinical situation may mimic low back pain of discogenic origin. However, classic neurological signs of low back pain, such as foot droop, are not typically present[1]. Numerous clinical maneuvers that reproduce the symptoms of piriformis syndrome have been described in literature. Among these are the Freiberg stretching maneuver, the Pace maneuver, the Lasègue sign, the Beatty resisted contraction maneuver, and the FAIR test (Flexion–Adduction–Internal Rotation test)[5]. As with IFI, a diagnostic block can be extremely useful for clinical diagnosis[28].

Imaging

The MRI is the gold standard for evaluating piriformis syndrome[50]. MRI of the spine is essential to exclude a discogenic etiopathogenesis. An MRI assessment of the pelvis can also be useful to identify anatomical anomalies that are a predisposition for the piriformis syndrome[50], such as different muscle morphology on the two sides of the body. However, some authors highlight that piriformis dysmetria is observed only in 19% of patients affected by piriformis syndrome[51]. Some authors recommend the use of MRI neurography, which suppresses signals from

surrounding tissues to better visualize peripheral nerves[52]. Finally, electro-diagnostic tests often result normal in cases of piriformis syndrome and are therefore mainly used to exclude other discogenic conditions[5].

Conservative treatment

Non-steroid anti-inflammatory drugs (NSAIDs), muscle relaxants, and neuropathic agents such as gabapentin or pregabalin, in conjunction with physical therapy, represent the first line of choice in the conservative treatment of piriformis syndrome[1]. Intramuscular corticosteroid injections, administered either in isolation or in combination with a local anesthetic under ultrasound or fluoroscopic guidance, are also to be considered a valid, minimally invasive therapeutic intervention[53].

Surgical treatment

Several studies have reported positive outcomes with surgical intervention in piriformis syndrome[54-56]. The surgical procedure typically involves tenotomy of the piriformis muscle tendon and decompression of the SN. However, surgical treatment should be considered only after failure of conservative measures and following careful patient selection[1].

Gemelli-internal obturator syndrome

Anatomy

The superior and inferior gemellus muscles are small muscles located within the DGSp. The superior gemellus originates from the ischial spine, while the inferior gemellus arises from the ischial tuberosity. Both muscles insert onto the greater trochanter, along with the tendon of the obturator internus[11] and, as they both belong to the short external rotator muscle group, they function as external rotators of the thigh. The superior gemellus is innervated by the obturator internus and gemellus superior nerves (L5-S1-S2), whereas the inferior gemellus receives innervations from the nerve to quadratus femoris and the gemellus inferior (L4-L5-S1). The obturator internus is a flat, and roughly rectangular shaped muscle that originates from the ischiopubic ramus and the posterior surface of the obturator membrane. It inserts distally onto the greater trochanter together with the tendons of the gemellus muscles[28]. This muscle also acts as an external rotator of the thigh and is innervated by the internal obturator/gemellus superior[11,28].

Etiopathogenesis

Gemelli-internal obturator syndrome is a relatively rare condition caused by dynamic compression of the SN, often due to stretching or alterations in the function of the internal obturator muscle[11]. However, it is important to note that the specific role of the internal obturator muscle in this syndrome is not yet well understood or extensively described in the literature[11,28]. Evidence regarding gemelli–obturator internus syndrome remains extremely limited and is based predominantly on isolated case reports and small case series[11,28,57-59].

Clinical presentation

The symptoms are similar to those seen in piriformis syndrome and IFIS. The key diagnostic sign is tenderness upon palpation at the insertion point of the external rotators behind the greater trochanter[57]. As with other conditions similar to IFIS and piriformis syndrome, a diagnostic nerve block can be very helpful in confirming or ruling out the diagnosis[28].

Imaging

MRI is considered the gold standard for evaluating gemelli-internal obturator syndrome. It is primarily used to exclude other causes such as discogenic pathology or different forms of DGS[57,58].

Conservative treatment

There are few reports in the literature regarding conservative management. The main options include physical therapy and injections of local anesthetics, with or without corticosteroids[59].

Surgical treatment

To the best of our knowledge, there is only one randomized clinical trial involving six patients affected by gemelli-internal obturator syndrome who underwent surgical treatment. The surgical technique involved sectioning the tendon of the internal obturator near its insertion on the greater trochanter. At 8 years of follow-up after surgical release of the internal obturator muscle, patients experienced a significant decrease in pain compared with a control group of six patients treated conservatively[57].

Posterior femoral cutaneous nerve entrapment

Anatomy

The posterior femoral cutaneous nerve is a sensory nerve formed by fibers from S1, S2, and S3. Along with its collateral branches, it innervates a large anatomical area, including the posterior surface of the thigh, the infragluteal fold, the scrotum in males, the labium majus in females, and the lateral anal region. During its course, the nerve accompanies the SN, usually passing medially or posteriorly *(Fig. 5)*. The posterior femoral cutaneous nerve gives rise, medially, to the inferior cluneal nerve[60]. The inferior cluneal nerve is a sensory nerve that innervates the lower and medial parts of the skin of the buttocks. It follows a path parallel to the SN and pudendal nerve as they pass through the sciatic notch[61].

Etiopathogenesis

The site of entrapment typically occurs at two specific points: first, at the level of the perineal branch where the nerve passes under the ischium toward the perineum; second, proximally at the intersection between the

piriformis muscle and the sciatic spine. The presence of fibrous expansions from the ischium constitutes the main source of nerve entrapment[62,63]. Among DGS-related disorders, the posterior femoral cutaneous nerve entrapment is supported by case series and anatomical studies[60-68]. Therefore, the overall evidence strength is low.

Clinical presentation

Patients often report burning, tingling, or numbness in specific anatomical areas, including[64]:

- Lower and medial buttocks.
- Dorsal and proximal thigh.
- Lateral edge of the anus and the skin of the scrotum in men or the labia majora in women.

Palpation of the sciatic notch often increases pain but does not typically cause radiating symptoms. The absence of motor involvement signs can mimic pudendal nerve entrapment syndrome. An anesthetic nerve block of the posterior femoral cutaneous nerve is a valuable diagnostic tool[65]. Nerve compression becomes symptomatic during sitting or thigh rotation, and symptoms worsen if the patient sits on a hard surface, such as a bicycle seat[61].

Imaging

Ultrasound, X-rays, CT scans, and MRI are generally unable to provide definitive information but may be useful for diagnosis via exclusion[64].

Conservative treatment

The initial step in conservative management involves avoiding painful stimuli[65]. Pulsed radiofrequency is considered an effective treatment modality[65-66]. Pharmacological options include tricyclic antidepressants and anticonvulsants[66]. Conversely, adding corticosteroids to nerve blocks (if used diagnostically) does not seem to improve the situation compared to local anesthetic alone[67].

Surgical treatment

Surgical intervention involves decompression of the posterior femoral cutaneous nerve and its inferior cluneal branches. The procedure typically involves an incision along the axis of the gluteus maximus fibers, lateral to the ischial tuberosity. Nerve decompression is usually performed at the caudal and medial margins of the piriformis muscle. The success rate of surgery is approximately 70%[62,68].

Superior gluteal nerve entrapment

Anatomy

The superior gluteal nerve originates from the posterior rami of the spinal roots L4, L5 and S1 and then crosses the greater sciatic notch above the piriformis muscle. Its course then continues, together with the superior gluteal artery. Between the gluteus medius and the gluteus minimus. The superior gluteal nerve is a mixed motor and sensory nerve; its sensory innervation affects exclusively the greater trochanter and its motor component affects the gluteus medius innervated by both its superior and inferior branches, the gluteus minimus and the tensor fasciae latae muscle which receives only the inferior branch of the nerve[69].

Etiopathogenesis

The primary cause of superior gluteal nerve entrapment is iatrogenic and results from the patient's posture during surgery, specifically flexion and adduction of the lower limb[69]. Another common iatrogenic cause is related to hematoma formation due to incorrect intramuscular injection techniques[61]. Traumatic causes include nerve injury from a fall onto the buttock, motor vehicle accidents, and excessive rotation of the hip or thigh[61]. Current evidence is derived mainly from anatomical studies and case reports[61,69-75]. Therefore, the overall evidence strength is low.

Clinical presentation

Patients typically report pain in the buttock region that radiates to the popliteal fossa. Symptoms tend to worsen when sitting, standing for long periods, or during walking or running. The Trendelenburg sign may be present. Usually, patients with superior gluteal nerve entrapment exhibit three specific symptoms[61,70]:

- Pain in the buttocks.
- Weakness in the hip abductors, leading to a gait known as "waddling gait" or "swayed walk".
- Tenderness on the lateral side of the sciatic notch.

A key clinical sign is pain upon pressure applied to the posterior superior iliac spine and the greater sciatic notch, laterally and superiorly, between the piriformis and the gluteus medius muscle[61]. A diagnostic nerve block is strongly recommended to confirm the diagnosis[61].

Imaging

MRI can be useful to identify changes in the muscles innervated by the superior gluteal nerve[71]. Electromyography (EMG) of these muscles may also aid in diagnosis by revealing ongoing denervation[72].

Conservative treatment

Initial management involves local infiltration of corticosteroids. If symptoms persist, surgical intervention may be necessary[73].

Surgical treatment

Surgical intervention is considered for chronic cases and involves decompression of the nerve via neurolysis. Neurolysis is achieved by performing an incision that extends from the posterosuperior iliac spine to a point about two fingerbreadths above the greater trocanther. With access gained, the superior margin of the gluteus maximus is separated from the tensor of the fasciae latae. The gluteus maximus is then moved inferomedially aside to

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expose the inferior margin of the gluteus medius which, in turn, is moved superolaterally aside, to permit access to the superior gluteal nerve which undergoes neurolysis[70,74,75].

Pudendal neuropathy

Anatomy

The pudendal nerve is a mixed nerve that arises from the ventral branches of S2, S3, and S4. It runs beneath the piriformis muscle and exits the pelvic cavity through the greater sciatic foramen. It then passes through the lesser sciatic foramen, near the internal obturator muscle, and enters the ischioanal fossa *(Fig. 5)*[58]. At the level of the ischial tuberosity, it divides into its terminal branches: the perineal nerve and the dorsal nerve of the penis/clitoris. The sensory component innervates the skin of the perineum and external genitalia. The motor component innervates the superficial transversus perinei, ischiocavernosus, and bulbocavernosus muscles[5,58].

Etiopathogenesis

The pudendal nerve may become entrapped in several locations. The most common anatomical site of entrapment is the space between the sacrotuberous and sacrospinous ligaments at the level of the ischial spine. Additionally, the pudendal nerve may be compressed medially to the ischium by the obturator internus muscle[5,62]. Pudendal nerve compression can also be caused by sitting while cycling. The repetitive motion of pedaling and the direct compression of the nerve between the tip of the saddle and the pubic bone make cyclists particularly susceptible to pudendal neuropathy affecting its motor and sensory terminal branches[5,76]. Horse riding, which causes high levels of pressure peaks in the perineal region, can also cause pudendal neuropathy[77]. Among DGS-related disorders, the pudendal neuropathy is supported by clinical studies, systematic reviews and anatomical descriptions[5,58,62,71, 76-81]. Therefore, the overall evidence strength is moderate.

Clinical presentation

Pudendal nerve entrapment causes pain medial to the ischium, involving the penis, scrotum, labia, perineum, and/or anorectal regions. The diagnosis of pudendal neuropathy is primarily clinical, as there are no specific tests or clinical signs that definitively confirm it. However, the following criteria (Nantes criteria) can be suggestive of the diagnosis[78]:

- Pain perceived within the pudendal nerve territory.
- Pain worsens when sitting (except when sitting on a toilet seat).
- The patient does not wake up during the night due to pain, as pain during rest hours is absent.
- No sensory deficits are detected on clinical examination.
- The patient has a positive response to a diagnostic pudendal nerve block.

Exclusion criteria for pudendal neuralgia include coccygeal, gluteal, or hypogastric pain that is purely paroxysmal, pruritus alone, or imaging abnormalities that can explain the symptoms[78].

Imaging

Some authors suggest that T2-weighted MRI neurography can evaluate changes in perifascicular and endoneurial signal intensity, which may be useful in diagnosing pudendal neuropathy[71]. However, the most specific diagnostic tests remain electromyography and nerve conduction velocity studies[79].

Conservative treatment

The most common conservative treatments include corticosteroid injections, pulse radiofrequency, and spinal cord stimulation[80]. If these treatments fail, surgical options may be considered.

Surgical treatment

In cases where conservative treatments are unsuccessful, laparoscopic transperitoneal pudendal neurolysis is performed. This surgical technique involves decompression and mobilization of the pudendal nerve until it enters Alcock's canal[81].

Proximal hamstring syndrome

Anatomy

The term "hamstring" refers to any of the three posterior thigh muscles in human anatomy (i.e., biceps femoris, semimembranosus, and semitendinosus). The long head of the biceps femoris originates from the ischial tuberosity and shares a common tendon with the semitendinosus muscle. The SN is in close anatomical relationship with the common tendon of the biceps femoris and semitendinosus muscles; therefore, any pathology of the tendon and/or its insertion area can exert a compressive action on the SN[2,82].

Etiopathogenesis

The SN can be involved in several pathologies, whether acute or due to overuse, affecting the hamstrings, which may be involved both in isolation and in combination. The main clinical situations include[2,82]:

- Partial or complete indirect lesion of the biceps femoris and semitendinosus in their proximal portion and their sequelae
- Detachment of the common tendon of the biceps femoris and semitendinosus
- Avulsion fractures (acute or chronic/non-consolidated) of the ischial tuberosity
- Ischial tuberosity apophysitis and its sequelae
- Enthesopathy of the common tendon of the biceps femoris and semitendinosus

Among DGS-related disorders, the proximal hamstring syndrome is supported clinical studies and systematic

reviews[5,2,20,82-86]. Therefore, the overall evidence strength is moderate.

Clinical presentation

Proximal hamstring syndrome causes buttock pain, weakness, and sciatic neuralgia. Patients, especially in chronic cases, often report pain when sitting, particularly on hard surfaces. Palpation at the insertion of the common tendon of the biceps femoris and semitendinosus, as well as active and passive stretching of the hamstrings, can evoke pain[82]. The "long stride walking test" helps differentiate between proximal hamstring syndrome and IFIS[5]. Specifically, in proximal hamstring syndrome, pain appears during the initial phase of "heel strike," when the hip is flexed. Conversely, in IFIS, pain occurs during hip extension, which narrows the ischiofemoral space.

Imaging

Magnetic resonance imaging (MRI) is considered the gold standard for diagnosis, as it can reveal the presence of the clinical conditions described above[83]. Ultrasound (US) can also be useful for diagnostic purposes, while X-ray examination may be helpful in cases of avulsion or fracture of the ischial tuberosity[84].

Conservative treatment

Conservative management includes stretching exercises, extracorporeal shock wave therapy, corticosteroid injections, and physiotherapy primarily focused on eccentric stimulation of the proximal muscle-tendon area[20,85].

Surgical treatment

If conservative therapy fails, surgical decompression of the SN at the entrapment zones may be considered. This decompression can be performed either endoscopically or via open surgery[86].

DISCUSSION

The aim of this systematic descriptive review was to identify the different clinical situations capable of generating DGS and to define a general diagnostic pathway for DGS, as well as to analyse its occurrence in the athletic population. The findings highlight that the current literature on DGS is highly heterogeneous, encompassing randomized controlled trials, cohort studies, case series, case reports, anatomical investigations, and narrative or systematic reviews.

This heterogeneity reflects the still evolving nature of evidence on DGS, where high-quality comparative clinical studies remain limited and most available data derive from low-to-moderate level evidence. From an interpretative perspective, this composition of the literature suggests that current knowledge on DGS is driven more by cumulative clinical experience and anatomical rationale than by large, controlled investigations. Consequently, any synthesis must be considered primarily qualitative, and the coexistence of primary and secondary sources introduces a risk of overlap and interpretative redundancy. For these reasons, the present findings should be interpreted as a structured qualitative synthesis rather than a quantitative aggregation of evidence.

Evidence synthesis and clinical classification

Across the included studies, DGS can be conceptualized into seven main clinical entities: IFIS[13,14,16,17], piriformis syndrome[48,49], gemelli-internal obturator syndrome[11,28,57-59], posterior femoral cutaneous nerve entrapment[60-67], superior gluteal nerve entrapment[61,69,70,73-75], pudendal neuropathy[5,58,62,63,76,78], and proximal hamstring syndrome[2,5,20,82,84,85].

However, it is important to underline that this classification reflects a synthesis of heterogeneous and partially overlapping clinical descriptions rather than a universally validated nosological framework. The convergence of symptoms -primarily deep gluteal pain of non-discogenic origin exacerbated by sitting[10] - limits the possibility of clear clinical stratification based solely on symptomatology.

In particular, the overlap between pudendal neuropathy and posterior femoral cutaneous nerve entrapment is frequently reported, both in terms of anatomical distribution and clinical presentation[62,63], making differential diagnosis particularly challenging[62,63].

Similarly, several of the described syndromes share common biomechanical and compressive mechanisms, suggesting that DGS may be better interpreted as a spectrum of related entrapment neuropathies rather than strictly independent entities.

Diagnostic considerations

From a diagnostic standpoint, clinical history and physical examination alone appear insufficient to reliably distinguish between the different DGS entities[10]. Imaging plays a variable role depending on the condition. MRI provides more direct diagnostic information in IFIS[13,14,16,17] and proximal hamstring syndrome[2,5,20,82,84,85], whereas in other conditions it is mainly useful to exclude alternative diagnoses[64].

Advanced imaging techniques such as T2-weighted MR neurography may improve future diagnostic accuracy by allowing better visualization of peripheral nerve involvement[71]. Nevertheless, at present, electrodiagnostic studies remain particularly relevant in superior gluteal nerve entrapment and pudendal neuropathy[72,79]. In addition, diagnostic nerve blocks represent an important functional tool supporting both diagnosis and clinical decision-making across several DGS conditions[28,61,65,67,78].

Overall, the diagnostic complexity of DGS appears to arise from the combination of overlapping symptom patterns and limited specificity of currently available diagnostic tools, resulting in a condition that remains largely diagnosis-of-exclusion in many clinical settings[10].

DGS in the athletic population

In athletes, IFIS may present with pain radiating anteriorly toward the inguinal region[28], and for this reason it has been included among extra-articular causes of groin pain in the Groin Pain Syndrome Italian Consensus Conference update 2023[28].

Several DGS-related conditions appear to have particular relevance in sport-specific contexts. Pudendal neuropathy has been associated with activities involving prolonged perineal compression such as cycling[5,76] and horse riding[77], where repetitive mechanical stress may act as a triggering factor.

Proximal hamstring syndrome represents another frequently reported DGS entity[2,5,20,82,84-86]. Its occurrence can be interpreted in relation to the close anatomical relationship between the proximal hamstring tendon complex and the SN, where tendon overuse or traumatic alterations may contribute to nerve irritation or compression[2,5,20,82,84,85]. In football players, proximal tendon pathology with increased tendon volume has been suggested as a potential predisposing factor for this condition[83].

In a general sense, sporting activities characterized by repeated gluteal loading and exposure to microtraumatic events (such as rugby, American football, or goalkeeping in soccer) have been hypothesized to be potentially associated with DGS-related conditions, based on limited evidence mainly derived from observational studies and case reports[87].

However, the exact prevalence and risk stratification across sports disciplines remain insufficiently defined in the current literature.

Limits of the study

This systematic descriptive review has several limitations. First, the included studies were highly heterogeneous in terms of study design, patient populations, diagnostic criteria, and treatment strategies, which precluded a quantitative synthesis or meta-analysis. Second, a large proportion of the available literature consisted of narrative reviews, case series, and case reports, resulting in an overall low level of evidence for several DGS-related conditions. Third, the lack of standardized diagnostic criteria and the clinical overlap with lumbar spine and pelvic disorders may have affected the interpretation and comparison of the reported findings. Finally, due to the absence of homogeneous epidemiological data, this review was not designed to estimate the true incidence or prevalence of DGS, particularly in the athletic population.

CONCLUSION

Based on the currently available literature, DGS may be considered an “umbrella term” encompassing several distinct but clinically overlapping conditions involving neural and soft tissue entrapment within the DGSp. However, the diagnostic boundaries between these entities and other lumbar, pelvic, and extra-articular hip disorders remain incompletely defined. This overlap, together with the limited specificity of imaging findings in several conditions, continues to make the diagnosis of DGS challenging in clinical practice.

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