

Deep Gluteal Syndrome: A Narrative Review of Diagnostic Uncertainty and Management

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Abstract

Deep gluteal syndrome (DGS) conventionally refers to non-discogenic irritation or entrapment of the sciatic nerve within the deep gluteal space. It is one possible cause of buttock pain with sciatica-like symptoms, although definitions and diagnostic criteria vary across studies. This narrative review examines the anatomy, diagnostic assessment, rehabilitation, image-guided procedures, and surgical management of DGS. No single clinical test or investigation has been independently validated to confirm the diagnosis. Assessment combines the history, neurological findings, posterior hip provocation tests, and investigations directed at specific diagnostic questions. Conservative care is generally used first, but rehabilitation studies are heterogeneous and largely concern piriformis syndrome. Small trials have reported some between-group differences without establishing a consistently superior rehabilitation or adjunctive treatment. Image-guided injections may relieve symptoms and provide complementary clinical information, but the response alone does not confirm DGS or identify a specific pain source. Endoscopic decompression has produced favorable outcomes in selected observational cohorts, although direct comparisons with structured rehabilitation are lacking. The clinical framework presented in this review represents the authors' synthesis of the available literature and has not been validated. Its proposed assessment and treatment options do not constitute a mandatory sequence of care.

Categories: Osteopathic Medicine, Physical Medicine & Rehabilitation, Orthopedics

Keywords: deep gluteal syndrome, diagnostic imaging, nerve decompression, piriformis syndrome, posterior hip pain, rehabilitation, sciatic neuropathy

Introduction And Background

Assessment of sciatica-like pain includes examination of the lumbar spine and a neurological assessment. Deep gluteal syndrome (DGS) is an extraspinal consideration because buttock pain and radiating leg symptoms can overlap with lumbar radiculopathy [1-3]. The differential diagnosis also includes hip joint disease, proximal hamstring disorders, pelvic conditions, and other peripheral neuropathies [1,2,4,5]. The absence of an explanatory finding on lumbar imaging does not, by itself, establish DGS.

This review uses the sciatic-focused definition of DGS: non-discogenic irritation or entrapment of the sciatic nerve within the deep gluteal space [1,6]. This definition extends beyond piriformis syndrome because the nerve may be affected near other short external rotators, the quadratus femoris, the proximal hamstring origin, fibrous or vascular structures, and postoperative or post-traumatic scars [1,2,4-6]. These structural findings are considered possible DGS mechanisms when they are concordant with features suggesting sciatic involvement. Such features include the distribution of radiating pain or paresthesia, neurological findings, and mechanosensitivity, interpreted alongside posterior hip examination and any indicated investigations. None independently confirms DGS, and regional musculoskeletal abnormalities may instead represent alternative or coexisting causes of pain [1,2,6].

Different proposed mechanisms may require different treatment targets. A piriformis-directed intervention may not address a fibrous tether, ischiofemoral narrowing, or proximal hamstring pathology. Anatomical variants also occur without symptoms, so an imaging finding must be interpreted in relation to the clinical presentation [1,4,5]. Symptom behavior may inform a working hypothesis, but it does not independently identify the affected structure.

Although individual clinical tests have been evaluated, diagnostic criteria are not standardized across DGS studies, and their applicability beyond the populations studied remains uncertain [6-8]. Rehabilitation evidence is also insufficient to establish which exercise program, dosage, or progression should be used for specific DGS mechanisms [9-12]. These limitations complicate both diagnostic interpretation and the application of treatment findings to individual patients.

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This narrative review asks how the available evidence can inform diagnostic reasoning and the use of conservative management, image-guided interventions, and surgery in adults with suspected DGS. Particular attention is given to the applicability of findings from piriformis-specific populations to other proposed causes of deep gluteal sciatic symptoms.

Review

Search strategy and evidence selection

PubMed was the only bibliographic database searched, supplemented by targeted checks of publisher websites, reference lists, and reviewer-suggested publications. The exact original query and execution date could not be verified from the available records. During revision, an additional PubMed search was conducted on September 30, 2026, with publication-date limits of January 1, 1800, through August 27, 2026. No language, age, species, study-design, or full-text-availability filters were applied to this additional query. The complete strategy and search details are provided in the Appendices. This strategy was developed during revision and does not reconstruct the original search or the contents of PubMed on August 27, 2026.

All 1,112 retrieved titles and available abstracts underwent an AI-assisted relevance assessment using ChatGPT (OpenAI, San Francisco, CA, USA), which was also used for focused source checks. A separate supplementary log records relevance decisions and their rationale. Missing abstracts did not automatically lead to exclusion; English abstracts were assessed when available, including for non-English publications. Source inspection was selective, and the focused-check records distinguish information obtained from abstracts, publisher metadata, and targeted full-text inspection.

The clinical scope concerned adults with suspected DGS or piriformis syndrome. Anatomical studies and reports on alternative causes of posterior hip pain were considered when they informed mechanisms or differential diagnosis. Priority was given to recent reviews relevant to definitions or conclusions, comparative clinical studies addressing gaps, diagnostic studies, and representative treatment cohorts. Older reports were retained for relevant anatomical or diagnostic information. Reports confined to spinal disease without a deep gluteal contribution, unrelated nerves or anatomical regions, or pediatric-only clinical populations were outside the main clinical scope. Null or unfavorable findings were considered under the same relevance criteria. Relevant reports not prioritized for further inspection remained in the log and were not classified as ineligible.

Selected sources were compared narratively by population, diagnostic definition, study design, intervention and comparator, follow-up, and outcomes. Piriformis-specific evidence was distinguished from evidence concerning broader DGS. Reports identified as belonging to the same trial were considered together; possible cohort overlap was retained as an uncertainty. Participant totals from reviews were not added to those of their constituent primary studies.

Quantitative findings were summarized descriptively and attributed to the relevant primary study or published synthesis. Confidence intervals (CIs) were reported where available and verified in the source. Findings from published meta-analyses were identified as results of those syntheses. No new quantitative pooling or cross-study statistical comparisons were performed. Treatment-response proportions were interpreted within their reported outcome definitions, denominators, and follow-up. Nonsignificant between-group differences were not interpreted as evidence of equivalence.

Methodological limitations were discussed narratively without formal risk-of-bias assessment or certainty grading. No prospective protocol registration, independent duplicate human screening, or exhaustive full-text assessment was undertaken. Final literature selection remained selective and cannot establish comprehensive coverage. The screening log, including the AI-assisted relevance assessments and their rationale, is available from the corresponding author upon request.

Clinical anatomy and pathophysiology

After leaving the pelvis, the sciatic nerve travels beneath the gluteus maximus near the piriformis, obturator internus-gemelli complex, quadratus femoris, and proximal hamstring origin. Fascial planes and vessels also cross this region, while the posterior femoral cutaneous nerve follows a nearby but separate course. Hip and knee position alter the sciatic nerve's relationship to these structures [3-5].

Potential local causes of sciatic irritation or entrapment include fibrous or fibrovascular bands, postoperative adhesions, post-traumatic scarring, proximal hamstring pathology, ischiofemoral narrowing, masses, and compression by the short external rotators [1,4,5]. Lopez Castellanos and Feipel reviewed anatomical variations in the sciatic-piriformis relationship and reported associations between selected configurations and piriformis syndrome [13]. The presence of an anatomical variant alone, however, does not establish that it causes the patient's symptoms [1,4,5].

Symptom behavior may help assess the clinical relevance of regional anatomy. Sitting can increase local pressure, while a long stride may provoke pain near the ischiofemoral space or proximal hamstring origin.

Hip flexion and adduction can reproduce symptoms in some piriformis-region presentations, and postoperative or post-traumatic adhesions may restrict nerve excursion [1,4,5]. These observations inform clinical hypotheses; a provoking movement can load several tissues and does not independently identify a symptomatic structure or establish sciatic involvement.

Recent reviews illustrate differences in the scope assigned to DGS. Bisciotti et al. used the term to encompass seven regional conditions involving several nerves [14]. Yoon et al. proposed a broader framework that includes muscle and tendon disorders, including enthesopathy, as potential pain sources even without direct sciatic nerve entrapment [15]. In a separate approach to posterior buttock pain, Reckling and Polly organized the differential diagnosis into six anatomical regions encompassing neural and regional musculoskeletal causes [16]. These approaches broaden consideration of competing and coexisting disorders but do not establish a validated diagnostic classification of DGS.

The present review retains a sciatic-focused definition to distinguish suspected deep gluteal sciatic involvement from regional pain without supporting neural features. Muscle and tendon disorders remain relevant as alternative diagnoses, coexisting conditions, or potential contributors to sciatic irritation. Other regional nerve entrapments are considered within the differential diagnosis rather than assumed to share the same assessment and treatment framework.

Clinical presentation and diagnostic reasoning

Patients may report deep buttock pain, reduced sitting tolerance, and posterior thigh pain or paresthesia. Symptoms can remain proximal to the knee or extend distally. Neither distribution reliably distinguishes DGS from lumbar radiculopathy, and local tenderness is nonspecific [1,2,7,17]. The history should establish the onset, progression, functional consequences, and relationship of symptoms to sitting, walking, hip movement, and changes in position.

Hopayian et al. describe sitting-related pain, tenderness near the greater sciatic notch, and symptom reproduction during maneuvers that lengthen or contract the piriformis [7,17]. These findings may support a clinical hypothesis but do not establish sciatic entrapment. Lo and Robinson distinguish piriformis-region myofascial pain from demonstrated focal neuropathy, emphasizing the uncertainty associated with the diagnostic label [18]. Coexisting lumbar, hip, or proximal hamstring pathology requires consideration even when posterior hip provocation tests reproduce familiar symptoms.

Fever, unexplained weight loss, night pain disturbing sleep, or a worsening course inconsistent with the presumed mechanical diagnosis should prompt reassessment for alternative causes. Depending on the clinical context, these include infection, neoplasm, inflammatory disease, and pelvic pathology [19–21]. Neurological deterioration, including progressive weakness or impaired gait, requires timely assessment, with urgency determined by the severity, progression, and suspected cause [19,22].

New urinary, bowel, or sexual dysfunction, or altered perineal sensation, associated with severe low back pain radiating into the leg warrants immediate assessment for possible cauda equina syndrome [22]. When spinal infection is suspected, magnetic resonance imaging of the affected spinal region is an appropriate initial investigation; absence of fever does not exclude infection [21]. Suspected serious pathology requires assessment outside the proposed rehabilitation framework.

Physical examination

The examination includes gait and a neurological assessment, followed by evaluation of the lumbar spine, hip, sacroiliac region, and proximal hamstring origin as indicated [1,2]. Straight-leg raise and slump testing may reproduce symptoms associated with neural mechanosensitivity, but neither identifies the anatomical site of irritation. Their findings require interpretation alongside neurological localization and examination of regional musculoskeletal structures [1,2].

For the active piriformis test, the patient lies on the opposite side with the tested hip and knee flexed. The patient presses the heel into the examination table and performs hip abduction and external rotation against resistance while the examiner palpates the piriformis region [1,8].

For the seated piriformis stretch test, the patient sits with the hip flexed to approximately 90°. The examiner extends the knee and passively adducts and internally rotates the hip. Palpation is performed with the middle finger approximately 1 cm lateral to the ischium and the index finger proximally at the sciatic notch [1,8]. The location and reproduction of familiar pain or paresthesia should be recorded separately from unfamiliar stretching discomfort.

Martin et al. retrospectively evaluated these tests in 33 patients aged 15–64 years who underwent endoscopic assessment. Sciatic entrapment was identified in 23 patients and was absent in 10 [8]. The active piriformis test had a reported sensitivity of 0.78 (95% CI, 0.58–0.90) and specificity of 0.80 (95% CI, 0.49–0.94). The corresponding estimates for the seated piriformis stretch test were 0.52 (95% CI, 0.33–0.71) and

0.90 (95% CI, 0.60-0.98). For a positive result on either test, the tabulated sensitivity was 0.91 (95% CI, 0.73-0.98) and specificity was 0.80 (95% CI, 0.49-0.94) [8].

The results text, however, described a positive result for either test in 20 of 23 patients with confirmed entrapment (87%), which differs from the tabulated sensitivity. This discrepancy remains unresolved. The estimates also require caution because the sample came from a single surgeon's selected surgical population. Test reliability was not assessed, blinding of the endoscopic assessment to the clinical test results was not reported, and the reliability and validity of the endoscopic criteria had not been established. These findings do not establish diagnostic performance in an unselected population with posterior hip pain [8].

Fishman et al. reported a sensitivity of 0.881 and specificity of 0.832 for an H-reflex criterion during flexion, adduction, and internal rotation (FAIR) positioning within their operational definition of piriformis syndrome [23]. These values do not describe the diagnostic accuracy of the bedside FAIR pain-provocation maneuver against an independent reference standard. Positional electrophysiological findings and pain provocation are different measurements and should not be treated as interchangeable tests.

Imaging and electrodiagnostic studies

Imaging should address a defined diagnostic question. Pelvic or hip magnetic resonance imaging can identify masses, postoperative changes, proximal hamstring lesions, quadratus femoris edema, ischiofemoral narrowing, and competing hip pathology [2,4,5]. Ultrasound permits dynamic assessment and image-guided procedures, although its findings depend on acquisition technique and operator experience. An anatomical abnormality requires clinical correlation and does not, by itself, establish the source of symptoms.

Magnetic resonance neurography (MRN) may provide additional information when persistent symptoms remain unexplained, and a focal sciatic abnormality or postoperative tether is suspected. Filler et al. evaluated neurographic findings and treatment outcomes in a selected referral population with non-discogenic sciatica and piriformis syndrome [24]. Their results do not establish diagnostic performance in an unselected population with posterior hip pain. This review therefore considers MRN a selective adjunct whose use depends on local availability, appropriate acquisition protocols, reader expertise, and whether the findings could change management.

Measurement performance also depends on the feature examined. Eastlack et al. reported stronger interobserver agreement for identifying sciatic nerve variants than for assessing piriformis muscle asymmetry [25]. Preisner et al. found scanner-associated differences in quantitative MRN parameters in healthy volunteers, despite generally good agreement for several measurements [26]. These studies address reproducibility and do not establish the diagnostic accuracy of MRN for DGS.

Electrodiagnostic testing, combining nerve conduction studies with needle electromyography, helps document objective nerve dysfunction, particularly axonal injury, and assess whether the findings favor sciatic neuropathy, lumbosacral radiculopathy, or plexopathy [18,27,28]. Its contribution is primarily to differential diagnosis and characterization of nerve involvement. Findings consistent with sciatic neuropathy do not, by themselves, identify the structure responsible for entrapment within the deep gluteal space.

Electrodiagnostic testing is not a stand-alone confirmatory test for pain-predominant DGS. Normal findings do not exclude suspected DGS without detectable nerve dysfunction, and a normal needle examination does not definitively exclude radiculopathy [18,28]. Positional H-reflex abnormalities require interpretation alongside the clinical examination and conventional electrodiagnostic assessment; they should not be used alone to confirm DGS [18,23,29].

In an exploratory study of 19 participants with unilateral gluteal pain, Kurková et al. reported side-to-side differences in selected sciatic nerve diffusion-tensor imaging parameters and abnormalities of the H-reflex during positional testing. Clinical tests were inconclusive [29]. These observations do not establish diagnostic accuracy or validate advanced imaging and positional electrophysiology as independent confirmatory tests.

Rehabilitation and conservative management

Conservative care is commonly considered when there is no progressive neurological deficit or lesion requiring specific treatment [1,2]. Kirschner et al. reviewed treatment options for piriformis syndrome, including stretching and physical therapy [9]. Tonley et al. described hip strengthening and movement reeducation in a single patient [10].

Shamsi et al. randomized 45 participants with chronic low back pain and a DGS diagnosis based on a positive FAIR test and piriformis tenderness to stretching, self-compression, or control groups. All groups received heat and transcutaneous electrical nerve stimulation over 10 treatment sessions. Pain improved within all

groups, but changes in pain and surface electromyographic measures of the tibialis anterior and gastrocnemius did not differ significantly between groups. Disability improved more with stretching than with self-compression [12]. The small sample, piriformis-based diagnostic criteria, and retrospective registration limit interpretation and generalizability. Because all groups received cointerventions, within-group improvement cannot be attributed to the exercises alone.

The following options represent the authors' clinical synthesis. The cited publications do not validate their combination, sequence, or exercise dosage as a rehabilitation program for DGS. Selection may take account of symptom irritability, identified impairments, functional goals, and patient preferences.

Within this proposed approach, education addresses diagnostic uncertainty and the possible relationship between symptoms and posterior hip loading. Sitting duration, walking distance, sleep disruption, or a patient-selected activity can provide functional baselines. Possible activity modifications include shorter sitting intervals, position changes, reduced direct pressure, and walking within tolerance. These options can be adjusted according to the patient's response.

Exercise options may include gentle hip movement, low-load isometric exercise, trunk exercises, and small-amplitude neural gliding when tolerated. Hip strengthening, gait practice, and activities linked to functional goals may be introduced according to individual capacity. These components do not constitute a mandatory sequence. Persistent distal symptoms or reduced function after exercise warrant reassessment of the load and working diagnosis, particularly when stretching or neurodynamic maneuvers provoke sustained symptoms.

Exercise selection may also reflect a provisional hypothesis about the structures contributing to symptoms. These adjustments are reversible treatment hypotheses, not diagnostic tests or validated rules for assigning DGS subtypes. Suspected piriformis involvement may prompt consideration of graded hip-rotator loading. With suspected ischiofemoral involvement, temporary reduction of stride length or exposure to provocative hip extension and adduction positions may be tried. Suspected proximal hamstring involvement may prompt adjustment of tendon loading. After surgery or trauma, progression may take account of tissue healing and local sensitivity; sensitivity alone does not establish scar-related nerve entrapment.

These modifications may be revised or discontinued according to tolerability and functional response. Improvement does not independently confirm DGS or identify the responsible anatomical structure, while lack of improvement does not independently exclude the suspected mechanism. Persistent disability warrants reassessment of the diagnosis, exercise dose, and barriers to participation. Coexisting lumbar or hip disease and other competing diagnoses require separate assessment.

Image-guided injections and adjunctive interventions

Image-guided injections may relieve symptoms and provide complementary information during clinical assessment. Improvement after a local anesthetic injection does not independently confirm DGS or identify a specific site of entrapment [17]. Interpretation should consider injectate dispersion. A small MRI feasibility study in healthy volunteers documented dispersion after piriformis injection, although visible spread to the sciatic nerve was not demonstrated [30]. Placebo effects, other nonspecific procedural effects, and natural symptom variability may also contribute to improvement [17]. When a corticosteroid is co-administered, subsequent relief cannot be attributed solely to the local anesthetic.

The intended target, injectate composition and volume, timing and duration of relief, and changes in pain and a predefined functional activity should be documented. These observations require interpretation alongside the history, examination, and relevant investigations. Sitting, walking, sleep, or exercise tolerance may provide functional outcomes beyond pain intensity. An inadequate response warrants reassessment of the target, technique, and working diagnosis.

Most injection studies concern piriformis syndrome, with uncertain applicability to other DGS mechanisms. Michel et al. described a single-center series of 250 patients assessed using clinical criteria for piriformis syndrome [31]. Hilal et al. pooled heterogeneous studies of local anesthetic, corticosteroid, and botulinum toxin injections and reported pain reduction after these treatments [32]. Differences in study design and limited direct comparisons constrain treatment ranking. These findings do not establish an optimal agent or target for fibrous tethering, hamstring-related entrapment, or postoperative scarring.

Evidence for adjunctive interventions also requires attention to the comparator. Tabatabaiee et al. randomized 32 patients to ultrasound-guided dry needling or a waitlist control and found less pain with dry needling at one week [33]. This comparison does not establish longer-term benefit or isolate a specific needling effect against a sham procedure.

Fu et al. randomized 70 participants to ultrasound-guided corticosteroid injection or extracorporeal shock-wave therapy, with home stretching in both groups. Outcomes improved at one and five weeks, without a significant between-group difference in improvement rates; baseline pain and disability imbalances

complicated comparison [34]. Dede et al. randomized 48 participants to shock-wave therapy or dry needling, both combined with stretching; 44 completed follow-up and were analyzed. Pain, disability, and lower-limb function improved at one and three months, without significant between-group differences [35]. Neither trial establishes equivalence or applicability to other DGS mechanisms.

Yen et al. analyzed outcomes after ultrasound-guided sciatic nerve hydrodissection in 53 patients. At the final follow-up, 33 patients (62.3%) had a pain reduction of at least 50%; follow-up averaged 5.4 months and ranged from three to 19 months [36]. Three patients lost to follow-up had been excluded from the analysis. The uncontrolled design and an injectate containing dextrose, lidocaine, and betamethasone prevent separation of mechanical, pharmacological, and other treatment effects. These observations do not establish the comparative benefit of hydrodissection or an optimal schedule of repeat procedures.

Surgical management

Elective surgical consultation may be considered for persistent functional disability when the clinical assessment suggests a potentially treatable deep gluteal source. Consultation allows reassessment of the diagnosis and discussion of treatment options; it does not establish an indication for surgery. Imaging findings and any response to a targeted injection require interpretation within the overall assessment. Injection response alone does not establish the site or mechanism of entrapment. Endoscopic decompression and neurolysis aim to release a focal tether or compressive structure and restore sciatic nerve excursion [1].

Martin et al. reported improvements in pain, function, and sitting-related symptoms after endoscopic treatment in 35 patients [37]. Park et al. and Ham et al. also observed postoperative improvement in selected patients with persistent DGS symptoms [38,39]. These series lacked a non-operative comparator and used different diagnostic and outcome criteria. Concomitant procedures also complicate attribution of benefit. Park et al. treated associated intra-articular hip disease in some patients [38].

The reviews by Hopayian et al., Kay et al., and Metikala and Sharma described generally favorable surgical outcomes, although the evidence consisted predominantly of small observational studies [11,40,41]. Sun et al. reported an arthroscopic series incorporating ultrasound assessment in 30 patients [42]. These reports describe postoperative outcomes but do not establish whether decompression is superior to structured rehabilitation.

Elzeiny et al. reviewed seven endoscopic studies involving 312 patients and 316 hips, with a reported mean follow-up of 25.1 months [43]. The evidence was predominantly retrospective and concerned patients selected for surgery. Differences in patient selection, outcome definitions, follow-up, and concurrent procedures limit comparison across surgical series [38,39,43].

An individual series illustrates the importance of specifying the outcome. Ilizaliturri et al. reported excellent Benson ratings in 11 patients, good ratings in three, and a fair rating in one at the reported two-year assessment. Thus, 14 of 15 patients (93.3%) had an excellent or good rating [44]. Follow-up durations were reported inconsistently in the narrative and accompanying table. This proportion describes a particular outcome in a small, selected cohort without a non-operative comparator and should not be interpreted as a general probability of benefit from surgery.

The available evidence does not establish an optimal duration or essential components of conservative treatment before surgical referral [11,40,41]. The authors' proposed framework therefore does not require completion of a fixed rehabilitation period or sequence of procedures. Assessment considers functional disability, symptom progression, neurological findings, competing diagnoses, and concordance between the clinical presentation and any suspected anatomical lesion. Previous treatment, its tolerability, and its effects on function should be reviewed.

Decisions about surgery also require discussion of potential benefits, uncertainty about the outcome, operative risks, alternatives, and patient preferences. Progressive neurological loss or a lesion requiring specific treatment warrants timely assessment according to its severity and suspected cause, without waiting for completion of a routine rehabilitation sequence.

Quality of evidence and limitations

Interpretation is limited by inconsistent terminology, variable diagnostic criteria, small samples, and selective referral. Studies of piriformis syndrome cannot be assumed to represent all proposed causes of deep gluteal sciatic symptoms [6,18]. Broader definitions that include regional muscle, tendon, and other nerve disorders also change the populations and mechanisms encompassed by DGS [14,15]. These differences require attention when comparing studies or applying their findings.

Reported diagnostic performance depends on patient selection and the reference standard. Estimates from a selected endoscopic cohort do not establish accuracy in an unselected population with posterior hip pain [8]. Treatment response presents a separate interpretive problem: improvement after a targeted intervention

may support a clinical hypothesis, but it does not independently confirm the diagnosis or identify the symptomatic structure. Natural symptom variation and nonspecific treatment effects remain possible explanations [17].

The selected rehabilitation literature does not establish a validated, mechanism-specific exercise program or treatment sequence [9-12]. Common cointerventions complicate attribution of within-group improvement, and nonsignificant between-group differences do not establish equivalence [12,34,35]. Surgical evidence is predominantly observational. The case series discussed in this review lack structured non-operative comparators, while patient selection and concurrent procedures complicate attribution of benefit [37-39,42,44]. The available evidence therefore limits conclusions about comparative effectiveness and the choice of treatment for an individual patient [11,40,41,43].

This narrative review cannot establish comprehensive coverage of the literature. PubMed was the only bibliographic database searched, supplemented by targeted source checks, and full-text inspection was selective. The AI-assisted relevance assessment did not constitute independent duplicate human screening. No formal risk-of-bias assessment or certainty grading was performed; methodological concerns were considered narratively. Relevant or conflicting evidence may, therefore, have been missed or given insufficient weight.

Clinical implications for rehabilitation practice

Clinical assessment combines the symptom history, neurological localization, and examination of the lumbar spine and posterior hip. Table 1 organizes these assessment domains; its clinical interpretations represent the authors' synthesis. Symptom behavior may guide questions for the differential diagnosis. The associations summarized in Table 2 are clinical hypotheses and do not independently establish DGS, sciatic nerve involvement, or a specific anatomical source of pain.

Domain	Finding to seek	Clinical interpretation
History and load response	Deep buttock pain, sitting intolerance, posterior thigh symptoms, and aggravating positions or activities [1,2,7,17].	Defines the clinical pattern and provides functional baselines; it is not diagnostic by itself.
Neurological localization	Strength, reflexes, sensation, gait, and symptom distribution [1,2,18].	Helps assess possible nerve root, plexus, sciatic nerve, or distal peripheral nerve involvement and identifies neurological deterioration.
Neurodynamic provocation	Reproduction of familiar pain or paresthesia during straight-leg raise or slump testing [1,2].	May indicate mechanosensitivity but does not independently localize the lesion or distinguish DGS from radiculopathy.
Posterior hip provocation	Reproduction of familiar symptoms during the active piriformis test or seated piriformis stretch test [8].	May support clinical suspicion when findings correspond to the history; diagnostic estimates come from a selected surgical cohort.
Anatomical assessment	Findings on targeted palpation, MRI, ultrasound, or magnetic resonance neurography considered in relation to the clinical presentation [1,2,4,5,24].	Addresses a specific differential diagnosis or suspected anatomical contribution. An abnormality does not independently establish the source of symptoms.
Electrodiagnostic assessment	Evidence of nerve dysfunction on nerve conduction studies and needle electromyography, interpreted alongside the neurological examination [18,27,28].	Helps assess sciatic neuropathy, radiculopathy, or plexopathy but does not independently identify a compressive structure within the deep gluteal space. Normal findings do not exclude pain-predominant DGS.

TABLE 1: Assessment domains in suspected DGS

Abbreviations: DGS, deep gluteal syndrome; MRI, magnetic resonance imaging.

Evidence basis: The assessment findings are informed by the cited clinical and diagnostic literature. Their organization into assessment domains and the "Clinical interpretation" column represent the authors' synthesis. The table has not been validated as a diagnostic instrument.

This table was prepared by the authors for this review based on the cited literature. It was not reproduced or adapted from a previously published table.

Clinical pattern	Possible interpretation	Next question or examination
Pain with prolonged sitting or direct buttock pressure [1,2,7,17].	Possible deep gluteal or regional musculoskeletal contribution; this pattern alone does not establish sciatic nerve involvement.	Measure sitting tolerance and the response to standing, walking, or weight shifting.
Pain during hip flexion, adduction, and internal rotation [1,2].	Several deep gluteal tissues are loaded; a piriformis contribution is possible but not proven.	Compare the response with resisted testing, local palpation, the contralateral side, and the remainder of the examination.
Pain beside the ischium during a long stride [1,2,4,5].	Possible ischiofemoral, quadratus femoris, or proximal hamstring involvement; the pain pattern alone does not identify the symptomatic structure.	Assess stride, hip extension and adduction, and hamstring loading; consider pelvic MRI when indicated.
Pain at the hamstring origin with resisted knee flexion or hip extension [1,2,4,5].	Possible proximal hamstring tendon involvement; the loading response alone does not establish associated sciatic nerve irritation.	Assess tendon loading responses alongside neurological findings and competing regional diagnoses.
Weakness, reflex asymmetry, progressive numbness, or a dermatomal pattern [18,27,28].	Possible nerve root, plexus, or peripheral nerve involvement requiring localization.	Assess neurological localization and progression; consider targeted imaging and electrodiagnostic testing. Progressive neurological loss warrants timely assessment [19,22].
Progressive night pain, systemic symptoms, unexplained weight loss, or a non-mechanical course [19-21].	Possible serious alternative cause, including infection, neoplasm, or inflammatory disease; interpretation depends on the overall clinical context.	Reassess for alternative causes and select investigations according to clinical suspicion and urgency. Absence of an explanatory lumbar finding does not establish DGS.

TABLE 2: Symptom behavior and clues to the differential diagnosis

Abbreviations: DGS, deep gluteal syndrome; MRI, magnetic resonance imaging.

Evidence basis: The symptom patterns are drawn from the anatomical and diagnostic literature discussed in the text. The "Possible interpretation" and "Next question or examination" columns represent the authors' clinical synthesis. These proposed associations and assessment suggestions are not validated diagnostic rules.

This table was prepared by the authors for this review based on the cited literature. It was not reproduced or adapted from a previously published table.

Table 3 summarizes selected publications and methodological concerns relevant to their interpretation. Applying these findings requires attention to the population studied, particularly whether participants had piriformis-specific symptoms, broader suspected DGS, or findings used to select patients for surgery.

Source	Design or population	Main contribution	Key methodological or applicability concern
Kizaki et al. [6]	Systematic review	Synthesized the definition of DGS as a non-discogenic sciatic nerve disorder involving entrapment in the deep gluteal space.	Variation in definitions and diagnostic procedures limits comparison across studies.
Hopayian et al. [7,17]	Systematic reviews of clinical features	Identified recurring features of piriformis syndrome, including buttock pain, sitting-related symptoms, tenderness, and symptom reproduction with provocative maneuvers.	Piriformis-specific populations; clinical features do not independently establish the diagnosis or represent every proposed DGS mechanism.
Martin et al. [8]	Retrospective diagnostic study; 33 patients undergoing endoscopic assessment	For either piriformis test positive, the source table reported sensitivity of 0.91 (95% CI, 0.73–0.98) and specificity of 0.80 (95% CI, 0.49–0.94).	The results text instead reports 20/23 positive cases (87%). Selected surgical population; test reliability was not assessed, assessor blinding was not reported, and endoscopic criteria were not validated.
Cohort using an			The electrophysiological criterion differs from the

Fishman et al. [23]	operational definition of piriformis syndrome, with treatment follow-up	Applied an H-reflex criterion during FAIR positioning and reported outcomes after injection and physical therapy.	bedside FAIR pain-provocation maneuver; its reported accuracy does not validate that clinical maneuver against an independent reference standard.
Kirschner et al. [9]; Tonley et al. [10]	Review and single-patient case report	Review of piriformis syndrome treatment options; single-patient report of hip strengthening and movement reeducation.	Neither source validates the combined rehabilitation framework proposed in this review.
Hilal et al. [32]	Systematic review and meta-analysis	Reported pain reduction after several piriformis-directed injection treatments.	Heterogeneous study designs and limited direct comparisons constrain treatment ranking and applicability to other DGS mechanisms.
Martin et al. [37]; Park et al. [38]; Ham et al. [39]; Sun et al. [42]	Observational surgical series	Reported postoperative improvement after decompression in selected patients.	No structured non-operative comparator; concomitant procedures may contribute to improvement.
Kay et al. [40]; Metikala and Sharma [41]; Hopayian et al. [11]	Systematic reviews	Reviewed surgical outcomes; Hopayian et al. also assessed conservative treatments.	Limited direct comparisons and heterogeneous patient selection restrict conclusions about comparative benefit.
Geler Külcü [3]; Lo and Robinson [18]	Clinical reviews	Discussed regional anatomy, differential diagnosis, and uncertainty surrounding piriformis syndrome.	Narrative interpretations do not establish independent diagnostic accuracy or validate a treatment algorithm.
Shamsi et al. [12]	Randomized trial; 45 participants with chronic low back pain and piriformis-based DGS criteria	No significant between-group difference in pain or surface EMG; disability improved more with stretching than with self-compression.	Small sample, common cointerventions, retrospective registration, and limited applicability to other DGS presentations.
Yen et al. [36]	Uncontrolled case series; 53 analyzed; mean follow-up, 5.4 months (range, three to 19 months)	At final follow-up, 33/53 patients (62.3%) had at least 50% pain reduction after sciatic nerve hydrodissection.	Three patients lost to follow-up were excluded. The uncontrolled design and combined dextrose, lidocaine, and betamethasone injectate prevent attribution to individual treatment components.
Kurková et al. [29]	Exploratory multimodal study; 19 participants with unilateral gluteal pain	Examined MRI, diffusion-tensor imaging, positional H-reflex findings, and clinical tests.	Clinical tests were inconclusive; diagnostic accuracy and independent confirmatory value were not established.
Elzeiny et al. [43]	Systematic review; seven studies, 312 patients and 316 hips; reported mean follow-up, 25.1 months	Summarized postoperative pain and functional outcomes after endoscopic sciatic nerve release.	Predominantly retrospective evidence from selected surgical populations. Heterogeneity limits comparison; reported outcomes should not be interpreted as a pooled probability of benefit.
Fu et al. [34]	RCT; 70 participants; assessments at one and five weeks	Compared corticosteroid injection with shock-wave therapy; both groups also performed stretching and showed improvement.	Baseline pain and disability imbalances; no significant difference in improvement rates; piriformis-specific population.
Dede et al. [35]	RCT; 48 randomized, 44 analyzed; assessments at one and three months	Compared shock-wave therapy with dry needling; both groups also received stretching and showed improvement.	No significant between-group differences; equivalence was not established; piriformis-specific population.
Lopez Castellanos and Feipel [13]	Systematic review of sciatic–piriformis anatomy	Examined anatomical variants and reported associations with piriformis syndrome for selected configurations.	Anatomical associations do not establish that a variant causes symptoms in an individual patient.
Bisciotti et al. [14]	Descriptive systematic review; 84 studies	Used a broader DGS definition encompassing seven regional conditions involving several nerves.	The proposed scope differs from the sciatic-focused definition used in this review.

Ilizaliturri et al. [44]	Surgical case series; 15 patients undergoing piriformis tendon release and sciatic nerve exploration	At the reported two-year assessment, 14/15 patients (93.3%) had excellent or good Benson ratings.	Small, selected cohort without a non-operative comparator. Follow-up durations differ between the narrative and table. The proportion does not establish a general probability of surgical benefit.
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TABLE 3: Selected evidence and its relevance to deep gluteal syndrome

Abbreviations: CI, confidence interval; DGS, deep gluteal syndrome; EMG, electromyography; FAIR, flexion, adduction, and internal rotation; MRI, magnetic resonance imaging; RCT, randomized controlled trial.

Evidence basis: The study-design descriptions and reported findings summarize the cited publications. The "Key methodological or applicability concern" column presents the authors' narrative assessment of limitations relevant to interpreting and applying these findings. No formal risk-of-bias assessment or certainty grading was performed. Quantitative findings are attributed to the cited publications; this review did not generate pooled estimates.

This table was prepared by the authors for this review based on the cited literature. It was not reproduced or adapted from a previously published table.

Table 4 presents the authors' proposed, non-validated framework for considering rehabilitation options and further assessment. It does not prescribe a treatment sequence. Potential adjustments to activity or exercise reflect provisional clinical hypotheses. Their suitability depends on tolerability, functional goals, neurological findings, competing diagnoses, and patient preferences. Suspected serious pathology requires assessment outside this proposed rehabilitation framework. Progressive neurological loss warrants timely assessment according to its severity and suspected cause. Symptoms suggesting cauda equina syndrome require immediate assessment and should not be managed by progressing through rehabilitation options [19-22].

Clinical domain	Possible components	Clinical purpose	Findings prompting reassessment
Clinical framing	Assessment of neurological deficits, the lumbar spine and hip, and reproduction of familiar posterior hip symptoms.	Identify warning features, competing diagnoses, and functional baselines.	Progressive deficit, systemic features, a mass, or another suspected lesion requiring specific assessment.
Symptom irritability	Adjustment of sitting and provoking loads; tolerable movement, walking, and low-load exercise.	Support daily activity while monitoring symptoms and function.	Worsening symptoms, a course inconsistent with the working diagnosis, or inability to identify a tolerable activity level.
Functional capacity	Hip and trunk strengthening, neural gliding when tolerated, gait practice, and activities linked to functional goals.	Assess whether individualized loading improves function without sustained symptom aggravation.	Persistent disability, sustained distal symptoms, or deterioration in function after exercise.
Targeted procedure	An anatomically directed procedure addressing a defined clinical question, with documentation of the target, injectate, and pain and functional response.	Assess symptom relief and functional response in relation to a defined clinical question, without assuming anatomical specificity.	No functional gain, an implausible target, or repeated short-lived responses without sustained benefit.
Surgical consultation	Review functional limitations and progression, neurological findings, competing diagnoses, prior treatment and response, and any concordant anatomical findings.	Assess whether a surgical option offers a favorable individual balance of potential benefits and risks, considering patient goals and preferences.	Progressive neurological loss warrants prompt assessment. Surgical candidacy cannot be inferred from persistent pain, an isolated positive test, or an incidental anatomical variant alone.

TABLE 4: Proposed non-validated clinical framework for rehabilitation and further assessment.

Evidence basis: This framework represents the authors' clinical synthesis. The sources informing it address clinical assessment and differential diagnosis [1,2,6,8]; selected rehabilitation approaches and limitations of conservative-treatment evidence[9-12]; therapeutic injections for piriformis syndrome and sciatic nerve hydrodissection in deep gluteal syndrome [32,36]; and surgical outcomes in selected cohorts and systematic reviews [11,38-41,43]. These publications do not validate the framework as a whole, its proposed combinations of care, or exercise dosage. The options do not constitute a mandatory treatment sequence. Their use requires consideration of neurological findings, competing diagnoses, comorbidities, access to care, and patient preferences. Study designs and methodological limitations are summarized in Table 3.

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Changes in sitting tolerance, walking, or another predefined activity can help assess clinical benefit. Improvement does not establish the underlying mechanism, while persistent disability warrants reconsideration of the diagnosis and treatment approach. Table 5 summarizes interpretive cautions concerning clinical tests, anatomical findings, and treatment response.

Interpretive point	Rationale	Pitfall
Clinical concordance	Concordant symptoms and examination findings may support clinical suspicion; isolated tenderness is nonspecific.	Treating a positive FAIR pain-provocation maneuver or an anatomical variant as diagnostic proof.
Neurodynamic test interpretation	Straight-leg raise and slump findings may reflect mechanosensitivity but do not independently establish the site of irritation.	Assigning the anatomical level from symptom reproduction alone.
Functional response to loading	Changes in sitting tolerance, exercise tolerance, or a predefined activity can inform adjustment of the proposed rehabilitation approach.	Treating improvement as confirmation of a specific mechanism, or continuing a load that causes sustained distal symptoms or reduced function.
Injection response	Changes in pain and function help assess clinical benefit, but do not establish the anatomical source or mechanism of symptoms.	Treating an injection response as a diagnostic gold standard or attributing improvement solely to one component of a mixed injectate.
Surgical outcome interpretation	Observational series report postoperative improvement in selected patients, but comparative evidence is limited.	Presenting surgery as superior to structured non-operative care or applying a selected cohort's outcome proportion as an individual probability of benefit.

TABLE 5: Interpretive cautions in suspected deep gluteal syndrome

Abbreviation: FAIR, flexion, adduction, and internal rotation.

Evidence basis: This table summarizes the authors' interpretation of key evidentiary limitations and is not a validated diagnostic or treatment guideline. It draws on the diagnostic and treatment evidence discussed in the text, with selected publications summarized in Table 3. Suggestions concerning loading and functional reassessment are proposed clinical options.

This table was prepared by the authors for this review based on the cited literature.

The relation of symptoms to position and loading can guide the differential diagnosis, without identifying a single cause on its own (Table 2). The selected studies differ in population, diagnostic criteria, and design, which limits direct comparison of their findings (Table 3). The proposed clinical pathway uses functional response and reassessment to guide changes in care (Table 4). Interpretation of test results and treatment response requires attention to the specific limitations of each finding (Table 5).

Conclusions

DGS remains a clinical consideration when symptoms suggest non-discogenic sciatic nerve involvement within the deep gluteal space. The available evidence supports assessment of the clinical pattern and competing diagnoses but does not establish a single confirmatory test or a preferred treatment sequence. Rehabilitation options remain incompletely defined, injection responses lack independent diagnostic specificity, and postoperative improvement in selected cohorts does not establish superiority over structured non-operative care. The proposed clinical framework is an unvalidated synthesis of the reviewed evidence. Treatment decisions should account for functional disability, neurological findings, the plausibility of the suspected mechanism, and patient preferences, with reassessment when the clinical course does not support the working diagnosis.

Appendices

PubMed search and supplementary narrative relevance assessment

- Search Record
- Database. PubMed, using NCBI E-utilities ESearch and EFetch.
- Execution. September 30, 2026; initiated at 13:14:34 UTC.
- Publication-date limits. January 1, 1800 through August 27, 2026, inclusive, using Date - Publication.
- Filters. No language, age, species, study-design or full-text-availability filters.
- Retrieval. 1,112 unique PMIDs, including 1,096 article records and 16 book records. All returned PMIDs were

exported; PubMed returned no query warnings or errors.

Provenance. The strategy was developed for this revision. It does not reproduce the original search or the historical PubMed state on August 27. Later indexing and record updates can affect results.

Source record. The archived ESearch response preserves the exact query translation; XML and tab-delimited exports preserve the retrieved records.

Assessment Completed

All retrieved titles and available abstracts underwent an AI-assisted relevance assessment on September 30, 2026. Of the records, 108 had no abstract. These records were not automatically excluded. This was a narrative prioritization exercise, not independent duplicate human screening or an exhaustive full-text eligibility assessment.

The 99 priority candidates are not 99 included studies or completed full-text assessments. They include linked corrections. Focused source checks are documented separately, with the access level for each source. The 37 uncertain records remain unresolved and were not used to support new clinical claims. The retracted report PMID 11477008 is flagged and is not proposed as clinical evidence.

Complete PubMed Strategy

```
(
  "Piriformis Muscle Syndrome"[Mesh]
  OR "deep gluteal"[Title/Abstract]
  OR "piriformis syndrome"[Title/Abstract]
  OR "pyriformis syndrome"[Title/Abstract]
  OR (piriformis[Title/Abstract] AND (syndrome*[Title/Abstract] OR sciatic*[Title/Abstract]))
  OR (
    sciatic*[Title/Abstract]
    AND (entrap*[Title/Abstract] OR compress*[Title/Abstract] OR neuropath*[Title/Abstract] OR irritat*
[Title/Abstract])
    AND (
      gluteal[Title/Abstract] OR subgluteal[Title/Abstract] OR piriformis[Title/Abstract]
      OR ischiofemoral[Title/Abstract] OR hamstring*[Title/Abstract]
      OR "obturator internus"[Title/Abstract] OR gemelli[Title/Abstract]
      OR "ischial tunnel"[Title/Abstract]
    )
  )
  OR "non-discogenic sciatica"[Title/Abstract]
  OR "nondiscogenic sciatica"[Title/Abstract]
)
AND ("1800/01/01"[Date - Publication] : "2026/08/27"[Date - Publication])
```

Search terms combined the Piriformis Muscle Syndrome MeSH descriptor with title/abstract synonyms and regional sciatic terms. No mandatory diagnostic-test or treatment term was imposed. A record returned by

the date filter was subsequently found to have a September 25, 2026 electronic publication date and was classified after the cutoff (PMID 42799040).

Technical and Verification Sources

<https://pubmed.ncbi.nlm.nih.gov/help/>

<https://www.ncbi.nlm.nih.gov/mesh/68055958>

<https://pmc.ncbi.nlm.nih.gov/articles/PMC10782553/>

<https://pubmed.ncbi.nlm.nih.gov/39593660/>

<https://www.ejcrim.com/index.php/mltj/article/view/6435>

Relevance Criteria and Selection

Priority was given to adult clinical studies and reviews that informed definitions, diagnostic assessment, rehabilitation, procedures or surgery, together with anatomical work needed to interpret proposed mechanisms. Focused source checks addressed reports that could change a clinical statement, clarify a disputed definition, supply a relevant comparison, or identify overlapping evidence. Reports with null or unfavorable findings were retained under the same relevance criteria. Studies were compared by population, diagnostic definition, design, intervention and comparator, follow-up, and outcomes. Evidence limited to piriformis syndrome was distinguished from evidence concerning broader DGS.

Reports sharing a trial registration were treated as a study family, and potentially overlapping institutional cohorts were flagged. Complementary outcomes could be drawn from different reports without counting them as independent samples. Systematic reviews were treated as secondary syntheses, and their participant totals were not added to those of constituent primary studies. Where overlap could not be resolved, no combined unique-participant count was calculated.

Reports confined to lumbar disease without a deep gluteal contribution, unrelated nerves or regions, or pediatric-only clinical populations were outside the main clinical scope. Older anatomy and diagnostic reports could be retained for a specific interpretive purpose. A new or statistically positive finding was not sufficient by itself for selection. Relevant papers not prioritized were retained in the log rather than labeled ineligible.

Language and Access

The database query had no language restriction. English abstracts were assessed when available, including for non-English articles. Full-text access was selective and source-specific; no claim is made that all non-English full texts or all 99 priority candidates were evaluated. Missing methods, unavailable full text, and uncertain cohort identity were retained as limitations.

Identification of Recent and Supplementary Reports

Recent articles were identified within the same PubMed result set. Electronic dates were distinguished from later issue dates and indexing dates. Thirty-one of the 32 references in the original submitted manuscript matched the retrieved records by their own DOI or PMID. Bisciotti et al. (DOI 10.32098/mltj_2026_6435) was verified separately on the official journal site, which gives August 14, 2026 as its publication date. Its absence from this retrieval does not establish that it is permanently unindexed. The publisher check was conducted during revision and is not backdated as an original search.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

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