Vilnius University Press

Acta Medica Lituanica
2026. Vol. 33. No 2. Online ahead of print

ISSN 1392-0138 eISSN 2029-4174
DOI: https://doi.org/10.15388/Amed.2026.33.2.11

Sciatic Nerve Anatomical Variations and Deep Gluteal Syndrome: Clinical Relevance and Current Evidence – A Narrative Literature Review

Mantas Leskauskas
Vilnius University, Faculty of Medicine, Vilnius, Lithuania
E-mail: mantas.leskauskas@mf.stud.vu.lt
ORCID ID https://orcid.org/0009-0001-7330-1694

Laura Nedzinskienė*
Vilnius University, Faculty of Medicine, Institute of Biomedical Sciences, Department of Anatomy, Histology and Anthropology, Vilnius, Lithuania
E-mail: laura.nedzinskiene@mf.vu.lt
ORCID ID https://orcid.org/0000-0003-4925-9323

Abstract. ***Background:*** The aim of the study: Based on the latest literature, this narrative literature review aims to provide a comprehensive evaluation of deep gluteal syndrome (DGS) and to assess the significance of anatomical variations of the sciatic nerve (SN) in relation to the syndrome.

Materials and methods: A review of the scientific literature was conducted by using the *PubMed*, *ClinicalKey*, and *ScienceDirect* databases. Publications were selected from 2015 to 2026. All publications were written in English. The following keywords and their combinations were used during the search: "deep gluteal syndrome", "sciatic nerve variations", "sciatic nerve entrapment", and "piriformis syndrome".

Results: The sciatic nerve, characterized by significant anatomical variability in the deep gluteal space, may result in altered biomechanics and predispose the nerve to compression. Anatomical variations of the sciatic nerve, depending on their relationship with the piriformis muscle, are categorized according to the Beaton and Anson classification (types A–F). In recent decades, the term "deep gluteal syndrome" has come into use, encompassing extrapelvic non-discogenic sciatic nerve compressions. The syndrome most commonly manifests as persistent, 'shooting' pain which worsens after prolonged sitting and radiates toward the back of the knee, hip, and/or gluteal region, as well as dysesthesia and nocturnal pain. There is currently no gold standard for the diagnosis of DGS. Diagnostic criteria may include compression of the SN in the deep gluteal region, sciatica-like pain, and a non-discogenic origin of symptoms. First-line treatment options include physical therapy, nonsteroidal anti-inflammatory drugs (NSAIDs), muscle relaxants, and rest. In addition, intramuscular injections of a local anesthetic may be administered to alleviate symptoms. If conservative treatment proves ineffective, surgical nerve decompression should be considered.

Conclusions: The most common abnormal sciatic nerve variation worldwide is the Type B variant. Although anatomical variations of the sciatic nerve alter its biomechanics, they have no direct, statistically significant association with the development of deep gluteal syndrome, according to the latest literature. Anatomical variations of the sciatic nerve may be considered a predisposing factor that increases the risk of DGS

* Corresponding author

Received: 19/05/2026. **Revised:** 23/06/2026. **Accepted:** 01/07/2026

when additional primary etiological factors are present. However, more comprehensive scientific studies are needed to assess this issue.

Keywords: sciatic nerve, deep gluteal syndrome, anatomical variations.

Sėdimojo nervo anatominiai variantai ir gilaus sėdmens skausmo sindromas: klinikinė reikšmė ir naujausi moksliniai duomenys – aprašomoji literatūros apžvalga

Santrauka. ***Darbo tikslas:*** Remiantis naujausiais literatūros duomenimis, atlikti išsamią naratyvinę apžvalgą apie gilaus sėdmens skausmo sindromą (GSSS) ir įvertinti sėdimojo nervo anatominių variacijų reikšmę.

Darbo metodika: Naudojant *PubMed*, *ClinicalKey*, *ScienceDirect* duomenų bazes atlikta mokslinės literatūros apžvalga. Publikacijos buvo atrinktos nuo 2015 iki 2026 metų. Visos publikacijos parašytos anglų kalba. Paieškos metu naudoti raktiniai žodžiai, jų junginiai: „deep gluteal syndrome“, „sciatic nerve variations“, „sciatic nerve entrapment“, „piriformis syndrome“.

Rezultatai: Sėdimasis nervas, pasižymintis reikšmingu anatominės eigos variabilumu giliojoje sėdmens srityje, gali lemti pakitusią biomechaniką ir predisponuoti nervo kompresiją. Sėdimojo nervo anatominės variacijos ir dėl jų kintantis santykis su kriaušiniu raumeniu yra grupuojamos pagal Beaton ir Anson klasifikaciją (A–F tipai). Pastaraisiais dešimtmečiais pradėta vartoti gilaus sėdmens skausmo sindromo sąvoka, apimanti ekstrapelvines nediskogenines sėdimojo nervo kompresijas. Sindromas dažniausiai pasireiškia: nuolatiniu „šaudančio“ pobūdžio skausmu, stiprėjančiu po ilgesnio sėdėjimo ir plintančiu link pakinklio, klubo ir (arba) sėdmenų srityje, bei dizestezija ir naktiniais skausmais. Aukso standarto GSSS diagnostikoje nėra. Diagnostiniai kriterijai gali būti: SN kompresija giliojoje sėdmens srityje, išialginio pobūdžio skausmas ir nediskogeninė simptomų kilmė. Pirmo pasirinkimo gydymo metodai: fizinė reabilitacija, medikamentinis gydymas NVNU, miorelaksantais ir ramybės režimas. Be to, gali būti skiriamos vietinio anestetiko intraraumeninės injekcijos simptomatikai slopinti. Konservatyviam gydymui nepasiteisinus, skirtinas operacinis gydymas – nervo dekompresija.

Išvados: Pasaulyje labiausiai paplitęs ne normos (A tipo) variantas yra B tipo variacija. Nors sėdimojo nervo anatominės variacijos pakeičia jo biomechaniką, tiesioginio ir statistiškai reikšmingo ryšio su gilaus sėdmens skausmo sindromo išsivystymu, remiantis naujausiais literatūros duomenimis, nėra. Sėdimojo nervo anatominės variacijos gali būti vertinamos kaip predisponuojantis veiksnys, didinantis skausmo sindromo išsivystymo riziką, esant papildomiems etiologiniams veiksniams, tačiau tam įvertinti trūksta išsamesnių mokslinių tyrimų.

Raktažodžiai: sėdimasis nervas, gilaus sėdmens skausmo sindromas, anatominės variacijos.

Introduction

The *Sciatic Nerve* (SN), the largest and longest peripheral nerve in the human body [1], is notable for its anatomically varying course in the deep gluteal space. In 1937, Lindsay E. Beaton and Barry J. Anson described and first classified this phenomenon, identifying as many as six main types of anatomical variations that define the relationship between the sciatic nerve branches and the piriformis muscle [2]. Such variability in the sciatic nerve can significantly affect its biomechanics and predispose it to compression, which is most commonly caused by the adjacent piriformis muscle [2]. In recent decades, following the identification of other potential causes of sciatic nerve compression besides the aforementioned *Piriformis Syndrome* (PS), a broader term, that of *Deep Gluteal Syndrome* (DGS), has been adopted in the scientific literature to encompass extrapelvic, non-discogenic causes of sciatic nerve compression [3]. The persistent 'shooting' pain, radiating to the back of the knee, hip and/or gluteal region, dysesthesia and nocturnal pain [3,4] result in a complex differential diagnosis aimed at determining the exact cause of sciatic nerve compression and establishing a diagnosis of DGS [3]. As a result, etiological treatment may be delayed and ineffective. Pain and

numbness in the lower extremities caused by sciatic nerve compression can significantly impair the quality of life and limit work capacity [4,5]. Modern-day factors such as a sedentary lifestyle, poor posture, and lack of physical activity are the main predisposing factors for DGS and contributors to symptom worsening [6]. Therefore, it is important to focus on preventive measures. The need to identify potential causes of DGS and to clarify the impact of the anatomical variability of the sciatic nerve on the development of the syndrome, in order to achieve more accurate diagnosis and a more favorable management plan, highlights the relevance of this study.

The aim of the study: based on the latest literature, this narrative literature review aims to provide a comprehensive overview of deep gluteal syndrome and to assess the significance of anatomical variations of the sciatic nerve in relation to the syndrome.

Materials and methods

The search for relevant literature was conducted in online scientific databases (*PubMed*, *ClinicalKey*, *ScienceDirect*). Articles investigating deep gluteal syndrome and the influence of sciatic nerve anatomical variations on the development of this syndrome were pursued. Additionally, information on piriformis syndrome was searched for. A number of filters were applied during the search: publications had to be dated from 2015 to 2026, and only written in English. The following keyword combinations were used during the search: "deep gluteal syndrome", "sciatic nerve variations", "sciatic nerve entrapment", and "piriformis syndrome". A total of 50 publications meeting the inclusion criteria were initially found, of which, 33 were later excluded. 17 publications were analyzed in this narrative review.

Scientific articles were selected based on the following inclusion criteria:

- Published between 2015 and 2026;
- Written in the English language;
- Available in full text;
- Studies that were conducted on humans.

Scientific articles were omitted based on the following exclusion criteria:

- published before 2015;
- written in a language other than English;
- without full-text access;
- studies involving animals.

Results and Discussion

Sciatic nerve anatomy and anatomical variations

The sciatic nerve is the largest and longest nerve in the human body, which originates from the ventral rami of the lumbar nerves (L4–S3), entering the sacral plexus in the pelvis [1,5]. The nerve exits the pelvis through the greater sciatic foramen below the piriformis muscle and descends along the posterior aspect of the thigh to the popliteal fossa, where it divides into the *Tibial Nerve* (TN) and the *Common Peroneal Nerve* (CPN) [1, 2]. The location and characteristics of SN bifurcation are particularly variable.

The sciatic nerve courses through the deep gluteal space, a collection of adipose tissue between the middle and deep gluteal aponeuroses, located anterior to the gluteus maximus muscle, posterior to the femoral neck, and adjacent to the greater and lesser trochanters [3]. This space contains important structures, including the piriformis muscle, the superior and inferior gemelli muscles, and the internal obturator muscle [3]. The piriformis muscle divides the deep gluteal space into suprapiriform and infrapiriform parts, through which, the superior and inferior gluteal arteries and

nerves, the posterior femoral cutaneous nerve, the sciatic nerve, and smaller structures pass [4]. After emerging from the pelvis, the sciatic nerve travels posterior to the obturator internus-gemellus complex and the quadratus femoris muscle, between the greater trochanter and the ischial tuberosity, before descending vertically down the thigh, accompanied by a common tendon of the biceps femoris and the semitendinosus muscle [3,7]. Studies conducted on cadavers have shown that, in most cases, the sciatic nerve is located 1.2 ± 0.2 cm from the lateral edge of the ischial tuberosity [3].

The sciatic nerve is able to adapt to body movements within the deep gluteal space [3]. During flexion, abduction, and external rotation of the thigh, the sciatic nerve experiences less tensile force as it slides over the posterior edge of the greater trochanter [8]. In the knee region, when the lower leg is flexed, the sciatic nerve moves posterolaterally; conversely, when the lower leg is extended, the nerve is deeply embedded in the ischiofemoral tunnel and experiences significant tension. Dynamic nerve entrapment may develop when smooth mobility is impaired [8].

A precise understanding and recognition of SN anatomical variations is crucial in order to avoid iatrogenic injury during interventional or surgical procedures [1]. In 1937, the variations were first classified into six distinct types (Types A–F) by Lindsay E. Beaton and Barry J. Anson, based on the relationship between the SN and the piriformis muscle [2]. In the typical case (Type A), the nerve exits below the piriformis and diverges at the level of the ischial tuberosity. Type B variation is characterized by early branching of the SN in the pelvic region, where the common peroneal nerve pierces the piriformis muscle, while the tibial nerve follows its typical course. Other rarer variants include the CPN coursing above the piriformis (Type C), an undivided sciatic nerve piercing the piriformis (Type D), or passing above the muscle (Type F), and the TN piercing the piriformis with the CPN passing superior to it [2, 7]. The main anatomical relationships defined by the Beaton and Anson classification are summarized in Table 1.

Table 1. Beaton and Anson classification of sciatic nerve anatomical variations in relation to the piriformis muscle; based on sources [2,7,9]

Type	Anatomical relationship to the piriformis muscle	Frequency / clinical note
A	The undivided sciatic nerve exits below the piriformis muscle and divides distally.	Typical pattern; reported in approximately 85.2% of lower extremities in a meta-analysis.
B	The common peroneal nerve pierces the piriformis muscle; the tibial nerve passes below it.	Most common anatomical variant; reported in approximately 9.8% of cases and may be clinically relevant when other etiological factors are present.
C	The common peroneal nerve passes above the piriformis muscle; the tibial nerve passes below it.	Rare variant; reported in approximately 1.9% of cases.
D	The undivided sciatic nerve pierces the piriformis muscle.	Very rare variant; may increase susceptibility to dynamic irritation if additional local factors coexist.
E	The common peroneal nerve passes above the piriformis muscle; the tibial nerve pierces the muscle.	Very rare variant.
F	The undivided sciatic nerve passes above the piriformis muscle.	Very rare variant.

A 2016 meta-analysis, which examined 7,210 lower extremities, found that the normal Type A variant was present in 85.2% of cases, while Type B was present in 9.8% of the sample pool. When examining across different continents, it was found that Type B variation was most prevalent in Asia (17.0%) and least prevalent in Africa (2.2%); when analyzed by gender, this variation occurred nearly twice as frequently in women as in men [2]. Other studies show a similar prevalence of ana-

tomical variations: in one cadaveric study, among 294 lower extremities, 10.2% presented variations, while, in another study, among 643 examples of hip *Magnetic Resonance Imaging* (MRI), an average of 13.2% showed variations [9].

The variable relationship between the sciatic nerve and the piriformis muscle may be a predisposing factor for nerve compression at the greater sciatic foramen, otherwise known as piriformis syndrome [2]. Compression is also possible elsewhere in the deep gluteal region. Therefore, accurate knowledge of these variations remains essential for diagnostic evaluation so that to minimize the risk of complications [1].

Deep gluteal syndrome

In recent decades, following the discovery of additional potential causes of sciatic nerve compression besides piriformis syndrome, a broader definition has been adopted in scientific literature. Deep gluteal syndrome is defined as extrapelvic compression of the sciatic nerve of non-discogenic origin in the deep gluteal space [3], characterized by pain and/or dysesthesia in the gluteal, hip, and/or posterior thigh regions, which may cause symptoms similar to radiculopathy [5]. This concept was first introduced in 1999 by P. McCrory and S. Bell in a sports medicine field publication. Although this syndrome typically involves only compression of the sciatic nerve, various sources included other nerve compressions, such as the posterior femoral cutaneous nerve, inferior gluteal nerve, and superior gluteal nerve [8]. In this study, the analysis of DGS is limited to sciatic nerve compression.

Compression of the sciatic nerve in the deep gluteal region clinically manifests as: (1) persistent pain in the hip and/or gluteal region, which intensifies after sitting for a prolonged period (longer than 20–30 minutes) or during rotation of an extended leg (with the hip in flexion) [3]; (2) 'shooting' sciatica-like pain, felt along the back of the thigh toward the knee; (3) dysesthesia in the buttock and/or extremity on the affected side; and (4) nocturnal pain that subsides during the day [4]. In addition, the patient's body is often seen in a forced position at rest in an effort to alleviate pain symptoms [3]. Sometimes, patients complain of a burning sensation in the gluteal region, along with aching, tingling, and/or spasms, which can mimic a hamstring tear or intra-articular hip pathology [5]. Clinical examination and successful diagnosis of DGS may prove challenging, considering other possible nerve and hip joint pathologies in the lumbar and pelvic region, many of which share overlapping symptoms [3].

Thus far, when discussing extrapelvic compression of the sciatic nerve, the majority of publications have generally focused solely on piriformis syndrome [3], as it was long assumed that the piriformis muscle is the only structure capable of compressing the sciatic nerve in the deep gluteal region. However, with the improvement of diagnostic and surgical techniques, a broader spectrum of etiological factors of sciatic nerve compression, both within and beyond the deep gluteal region, has been identified over time. Furthermore, the same nerve can be compressed at multiple sites along its entire length by the surrounding structures simultaneously, resulting in symptoms of sciatic nerve compression [5]. Therefore, it is particularly important to understand the anatomy and biomechanics of the deep gluteal region when searching for potential causes of pain.

Etiopathogenesis

The piriformis muscle, which is a structure located centrally in the deep gluteal space, is responsible for piriformis syndrome, considered the most common cause of extrapelvic compression of the sciatic nerve [5]. PS is diagnosed in 6–8% of the cases of sciatica [2]. However, additional anatomical structures exist in the region that can compress the nerve and cause deep gluteal syndrome: fibrous and fibrovascular bands, gluteal muscles, gemelli-obturator internus complex, hamstring muscles, the ischial tuberosity, trauma, and post-traumatic tissue scarring [8]. In addition, ischiofemoral impingement and greater trochanteric impingement syndrome may contribute to the development of

DGS [5]. According to statistical data, an analysis of 239 patients identified the most common sites of SN compression: the piriformis muscle (67.8%), sciatic foramen (6%), and the ischiofemoral tunnel (4.7%). Another study involving 481 patients showed that surgically treated SN compression was most commonly caused by the piriformis muscle (26%) or other regional muscles (14%) [8].

Piriformis syndrome

Piriformis syndrome occurs when the piriformis muscle compresses the sciatic nerve, causing nerve entrapment in the deep gluteal region. This most commonly occurs due to muscle fatigue or hypertrophy, which can constrict the infrapiriform foramen during hip flexion, adduction, and internal rotation. Furthermore, SN compression can be triggered by spasms, inflammation, anatomical variations of the piriformis muscle and the sciatic nerve, as well as direct trauma to the pelvis and hip [10]. These factors are associated with the primary piriformis syndrome. Secondary PS is associated with precipitating factors such as trauma, piriformis muscle overuse, muscle spasm, or other external pathology [11].

The etiology of the syndrome can also be categorized into static and dynamic types. Static etiological factors include: (1) anatomical variations of the piriformis muscle (split, accessory, hypertrophied, or atrophied muscle) and anatomical variants of the sciatic nerve; (2) scarring due to muscle trauma (it is estimated that 50% of the cases involved are related to trauma [2]); and (3) massive lesions or hematomas around the muscle [4]. Meanwhile, sciatic nerve compression during motion is attributed to dynamic factors, which are best examined endoscopically [3].

An asymmetrically enlarged piriformis muscle due to hypertrophy may alter the anatomical position of the sciatic nerve. However, according to one study, a 2 mm muscle asymmetry is found in as many as 81% of patients without piriformis syndrome, and patients with an asymmetry of 4 mm or greater reported no symptoms [7]. Another study found that among 14 patients diagnosed with piriformis syndrome following trauma, only two had an enlarged muscle on the affected side, while seven were found to have a diminished muscle [5].

Anatomical variations of the sciatic nerve, the most common of which is Type B (according to the Beaton and Anson classification), may predispose individuals to the development of DGS in the presence of other causative factors, such as trauma to the gluteal region, fatigue due to prolonged sitting or stretching, instability of the pelvis and spine, or other orthopedic conditions. These factors may accelerate the onset of SN compression [7]. Nevertheless, the variation itself does not always cause the symptoms specific to the syndrome. This is because there are both symptomatic patients without the aforementioned SN variations and asymptomatic patients with such variations [7].

Anatomical anomalies of the piriformis muscle, due to their unusual anatomical positioning, may also predispose individuals to the syndrome. Sometimes, before attaching to the greater trochanter of the femur, the piriformis muscle tendon may join other tendons of the superior gemellus and obturator internus muscles, or the obturator internus and gluteus medius muscles. Due to these variations, the piriformis muscle may begin to compress the sciatic nerve, particularly during internal hip rotation [8].

Fibrous and fibrovascular bands

Over the past years, thanks to advancements in medical diagnostics, additional factors unrelated to the piriformis muscle have been identified as causes of sciatic nerve compression. One example is fibrous structures attached to the sciatic nerve tissue, restricting the nerve's normal range of motion. Under normal conditions, the nerve can be slightly stretched or move smoothly along the surface of the adjacent structures, thereby adapting to the movements of the lower limb. However, impaired or absent sciatic nerve accommodation can precipitate the development of compression and neu-

ropathy. During endoscopic examination, fibrous and fibrovascular bands adhering to the nerve are a common finding in cases of sciatic nerve compression [3].

At the macroscopic level, these structures can be divided into three main types: (1) fibrovascular bands, which consist primarily of vascular tissue combined with connective tissue; (2) fibrous bands consisting of dense elastic connective tissue; and (3) vascular bands formed solely of vascular tissue. Meanwhile, depending on their location, the bands are classified as proximal (attaching to the SN at the greater sciatic notch level), distal (attaching in the ischial tunnel region between the quadratus femoris muscle and the proximal attachment point of the hamstring muscles), and middle (located at the level of the piriformis muscle and the obturator internus-gemelli complex) [3].

Based on their mechanism of pathogenesis, these bands are also divided into three groups. The first group includes fibrous bands that encircle the sciatic nerve, thereby preventing it from moving posterior to anterior or anterior to posterior. Their typical course runs from the posterior edge of the greater trochanter to the gluteus maximus muscle and the greater sciatic notch. The second type includes bands that firmly attach to the sciatic nerve and restrict any nerve mobility [3].

As a result, the sciatic nerve cannot adapt to the movements of the lower limb and is subjected to greater tensile force. Most often, bands extending from the greater trochanter are found laterally to the sciatic nerve, although some are also found medially, attaching to the sacrotuberous ligament. Lastly, the third type of fibrous bands is characterized by anatomically variable attachment sites, which anchor the sciatic nerve in place from various directions [3].

Gemelli-obturator internus syndrome

The gemelli-obturator internus complex refers to a unit consisting of structurally and functionally similar gemelli and obturator internus muscles. The sciatic nerve runs alongside this structure, creating a potential site for entrapment [8]. Compression of the sciatic nerve due to the gemelli-obturator internus syndrome is a clinically rare finding [3], but significant when assessing potential causes of sciatica-like pain [5]. In terms of pathogenesis, it is similar to piriformis syndrome and should be included among other causes of DGS. In this case, the sciatic nerve, passing between the lower edge of the piriformis and above the superior gemelli and obturator internus muscles, may be compressed from both sides in a 'scissor-like' manner [3]. There is a reported case where the SN runs directly through the internal obturator muscle [5]. Meanwhile, internal obturator hypertrophy is rarely seen, occurring only among bodybuilders [7].

Quadratus femoris muscle and ischiofemoral tunnel pathology

Ischiofemoral Impingement (IFI) syndrome is characterized by an atypical narrowing of the space between the ischial tuberosity and the femur, through which, the sciatic nerve passes. Normally, the diameter of this space is 23±8 mm [3]. The quadratus femoris muscle also plays a significant role in the development of the syndrome. In acute cases, edema and inflammation are typical causes, while chronically, entrapment occurs because of fibrous tissue formation [4].

The space may also narrow in cases of trauma. A tear or weakness in the abductor muscles may contribute to IFI. Weak thigh abductors can cause contralateral pelvic drop, leading to compression of the quadratus femoris muscle, which eventually results in dynamic IFI over time. Additionally, hamstring enthesopathies and tears, which are common in athletes, can narrow the quadratus femoris space and cause dynamic compression of the SN [8].

Hamstring pathology

The sciatic nerve, coursing alongside the ischial tuberosity to which the hamstrings attach [5], can be affected by muscle enthesopathies: partial or complete hamstring strain, tendon detachment, avul-

sion fractures, apophysitis, proximal tendinopathy, calcifying tendinitis, and contusions, resulting in entrapment during hip motion. Acute inflammation is typically characterized by edema of the surrounding tissues, which can irritate the sciatic nerve, whereas chronic inflammation is characterized by the formation of scar tissue between the muscles, tendons, and sciatic nerve. As a result, the sciatic nerve may be compressed during hip motion [3].

Gluteal muscle pathology

The most common gluteal muscle disorder causing SN compression is gluteal contracture. This condition, which is common in school-aged children, manifests as long-term gluteal muscle fibrosis and atrophy, resulting from repeated intramuscular injections into the buttocks. In many cases, fibrosis develops in the region, potentially causing symptoms of sciatic nerve compression. Furthermore, long-term gluteal contracture can lead to the formation of fibrous bands that restrict sciatic nerve mobility, resulting in sciatic nerve entrapment [7].

In addition, changes in the anatomical alignment of the pelvis, femur, and spine, due to orthopedic disorders and gluteal tendinopathies, can affect the natural course of the sciatic nerve and lead to compression [4].

Orthopedic causes

Compression of the sciatic nerve, and, ultimately, DGS, can be caused by adjacent hematoma or tissue edema resulting from severe trauma (acetabular or ischial tuberosity fractures) or from posterior hip dislocation [8]. Hematomas can compress the sciatic nerve directly, or else they can cause indirect damage (ischemia from vasa vasorum compression or by fibrous bands that anchor the SN in place). In addition, the sciatic nerve can be compressed by abnormally healed bone tissue following previous avulsion fractures of the lesser or greater trochanter, or ischial tuberosity [7].

Rare causes of DGS

In recent case reports, a variety of rare and previously undocumented etiologies of deep gluteal syndrome have been described. These include ligament calcification, enthesopathies and amyloidosis. These discoveries broaden the scope of potential causes of SN compression in the deep gluteal space and solidify deep gluteal syndrome as a multifactorial condition.

To date, the role of ligamentous pathology as a cause of nerve compression has been overlooked [12]. Typically, the sciatic nerve exits the pelvic region inferior to the piriformis muscle and descends inferolaterally, adjacent to the *Sacrospinous Ligament* (SSL), averaging a distance of 1.4 cm from the ischial spine. Thus, the smallest anatomical discrepancy resulting from ligamentous pathology can make the SN vulnerable to compression [12].

A case of isolated SSL calcification as the primary etiology of DGS was presented in 2025. Calcium salt deposition (calcification) can theoretically alter SSL biomechanics, increase its volume and reduce elasticity, all of which can contribute to a dynamic SN compression, exacerbated during flexion, adduction and internal rotation of the hip [12].

Enthesopathy-related dynamic SN compression is another concept highlighted in a recent case report [13]. The study describes a 45-year-old man presenting with symptoms consistent with DGS. A hip CT scan helped determine the cause of sciatic nerve compression to be a bony spur at the insertion site of the piriformis muscle. Dynamic ultrasonography successfully showed the dynamic motion-related nature of SN compression. Entesopathy causes friction between the piriformis muscle and the perineural tissues during hip joint movement, which results in pain. This rare case helps demonstrate the multifactorial nature of DGS that includes muscle-tendon pathologies [13].

Lastly, one exceedingly rare case of soft-tissue amyloidoma as the primary cause of sciatic nerve compression has been reported [14]. During nerve decompression surgery, abnormal fibrous tissue was found to be the cause of SN compression at the piriformis insertion site. Pathology revealed Congo red-positive amyloid deposition, identified as transthyretin protein [14]. The study shows that the etiology of DGS can sometimes arise from systemic soft-tissue disorders, as opposed to typical muscle, fibrovascular anomalies of the deep gluteal space.

Clinical assessment

No gold standard diagnostic method for deep gluteal syndrome has been established to date. According to a study, diagnosis of DGS is characterized by three features: sciatica-like pain, a non-discogenic origin of symptoms, and compression of the sciatic nerve in the deep gluteal region [8]. In many cases, the assessment is based on differential diagnosis, ruling out other possible causes of hip and lower extremity pain. The aforementioned symptoms can be triggered by both extrapelvic pathologies, including lumbar spine radiculopathy, facet arthropathy, and deep back muscle disorders, and intrapelvic pathologies. Therefore, it is very important to gather a detailed patient history before performing an examination, which should be followed by diagnostic imaging, injections or provocation tests [8].

First and foremost, the patient's posture should be noted – a limp and an altered standing posture (weight supported by the unaffected leg) are often characteristic [4]. The physical examination consists of several movement tests: the Lasègue, Beatty, and FAIR tests, Pace and Freiberg signs, and the piriformis stretch tests (active and seated) [3]. When checking for the Freiberg sign, the patient lies on their back with legs extended while the examiner rotates the patient's leg inward at the hip joint. If the patient feels an increase in pain, the test is considered positive. Pace's sign is observed when a seated patient, while actively abducting and externally rotating the leg, experiences increasing pain and faster muscle fatigue in the presence of external resistance from the physician [11].

During the FAIR (*Flexion Adduction Internal Rotation*) test, the patient lies on their unaffected side. The physician bends the patient's thigh to a 60° angle and the lower leg to a 60°–90° angle, and then performs adduction and internal rotation of the leg. The piriformis muscle is stretched, so patients may experience symptoms of sciatic nerve irritation [11].

During the Beatty test, the patient also lies on their unaffected side, with the symptomatic leg in slight hip and knee flexion with the knee on the examination table. During the test, the patient attempts to lift and hold the flexed limb off the table (usually at about 10 cm). The test is considered positive if gluteal pain occurs [11].

Meanwhile, the Lasègue test is considered positive when the patient experiences deep pain in the buttock while holding their fully extended leg at 90° hip flexion [11]. There are two types of piriformis muscle stretch tests. The test can be performed with the patient seated, where the subject's leg, extended at the knee, is bent to 90° at the hip, adducted, and rotated inward. In another variation of the test, the patient, with their heel firmly pressed against the table, abducts and externally rotates their leg against resistance. During both tests, the piriformis muscle is palpated [3]. It has been established that these two tests can reliably and accurately detect SN compression, verified by endoscopic examination [5].

The development of DGS is also influenced by other previously mentioned syndromes, which are examined during specific provocation tests. The stride walking test can help diagnose ischiofemoral impingement syndrome. During the examination, the patient walks in long strides, strongly extending and flexing the thigh, which may cause buttock pain lateral to the ischium. The patient can also be placed on their unaffected side while the examiner extends and gently adducts the patient's thigh – this is known as the IFI test. Pain may be perceived while the patient's thigh is in adduction

or maintained in a neutral position [8]. Hamstring tendinopathies can be detected by using the active hamstring test. The patient is asked to sit down and flex their knee at a 30° angle, after which, they attempt to flex it against resistance. The test is considered positive when pain and/or muscle weakness is/are experienced [8].

In addition, palpation of the gluteal region is crucial during a physical examination. With the patient lying prone, palpation is performed near the ischial tuberosity. Pain superolateral to the ischial tuberosity, at the level of the greater sciatic foramen, is characteristic of DGS; pain lateral to the ischial tuberosity may indicate IFI; pain at the ischial tuberosity may be a sign of hamstring tendon injury; pain medial to the ischial tuberosity may hint at possible pudendal nerve compression [5].

Among the most informative diagnostic imaging techniques for assessing sciatic nerve compression is magnetic resonance imaging [5]. The most suitable MRI sequences for visualization are T1- and T2-weighted sequences, evaluated in all three planes, from L5 to a point 2 cm distal to the ischial tuberosity [8]. This imaging method allows for the evaluation of both the anatomy of the nerve itself and the surrounding structures in the deep gluteal space [4], as well as potential causes of compression, including piriformis muscle anomalies, avulsion fracture sequelae, osseous compressions and intrapelvic abnormalities [5].

In standard cases, the sciatic nerve is an easily identifiable oval-shaped structure with distinct fascicles. In T1-weighted imaging, the sciatic nerve appears isointense relative to the surrounding muscles. Perineural and perifascicular tissues appear hyperintense due to their lipid composition [7]. In T2 or fast spin-echo inversion recovery sequences, the sciatic nerve appears isointense or slightly hyperintense relative to the muscle and hypointense relative to the local blood vessels. The nerve fascicles are well distinguished by the low-intensity signal emitted by the surrounding connective tissue [7].

On MRI fluid-sensitive sequences, nerve injury may exhibit a hyperintense signal similar to that of the surrounding blood vessels. However, potential imaging artifacts, such as the 'magic angle effect', should not be ruled out. In addition, changes in the nerve size, arrangement of the fascicles, or blurring of perineural fat may suggest neural injury. The main indirect sign of nerve entrapment is edema of the surrounding muscle due to impaired innervation. MRI also helps assess spine disorders, intra-articular hip pathologies, as well as other differential diagnostic criteria [5].

Gemelli-obturator internus syndrome may be suspected on MRI if neuritis is detected near the obturator-piriformis space [7]. Since the piriformis and obturator internus are functionally and structurally similar, it is often difficult to differentiate between sciatic nerve compression caused by either of these muscles [8]. Quadratus femoris muscle tears appear on MRI as a region of intramuscular fluid intensity with surrounding muscle edema. These findings are best visualized on STIR, fat-suppressed PD or fat-suppressed T2-weighted sequences. Tears of this muscle are usually found on the posteromedial surface of the femur, at the muscle-tendon junction [7].

Ultrasonography can similarly be used in the diagnosis of sciatic nerve inflammation. Although the procedure is prompt and readily available, its value in assessing the deep gluteal space structures is limited due to the depth, obliquity, and size of nerves, potential beam attenuation from intramuscular fatty infiltration, and the operator skill. Under favorable conditions, in cases of DGS, one may observe neural fascicular edema, echogenic perineural adhesions, and/or sciatic nerve compression by variant anatomy [15]. Changes in size, visible asymmetry of the piriformis, and palpable stiffness can also be observed. Another advantage of medical ultrasound is that the image is evaluated in real time. This allows for dynamic monitoring of SN compression and the assessment of its entrapment and reduced mobility [15]. One example is *Musculoskeletal Ultrasound* (MSK-US), which is slowly becoming the method of choice for real-time evaluation and guided management of peripheral nerve entrapments. It allows for nerve mobility assessment during provocative maneuvers, provides high-resolution imaging of superficial structures and enables real-time guidance during injections [12].

Guided injections are an important diagnostic and therapeutic tool in the management of DGS, allowing the exclusion of articular pathology. The procedure can be performed under ultrasound or *Computed Tomography* (CT) guidance with the patient lying prone [7]. In addition, fluoroscopy, electroneuromyography and MRI may be utilized to obtain more precise results [5]. A mixture of local anesthetic and corticosteroid is injected into the perineural sciatic fat or within the nerve sheath. Once injected under the ischial spine, it should disperse along the perineural fat with hip movements. Care should be taken to avoid penetrating into sciatic nerve splits, the pudendal bundle, or the inferior gluteal vessels, as this increases the risk of significant bleeding [7]. Symptom relief following the injection may suggest DGS and, in turn, lead to better treatment outcomes [15].

Electromyography and nerve conduction studies may be used as supplementary tools in diagnosing DGS. An abnormal H-reflex in the tibial and/or peroneal nerves may indicate sciatic nerve entrapment by the piriformis muscle. Comparing both legs and performing a dynamic test (with the knee in extension and hip in adduction and internal rotation) can increase diagnostic accuracy, since in this position the piriformis may compress the sciatic nerve, potentially leading to temporary impairment of nerve conduction distally. However, it is important to note that obesity, peripheral edema and age may hinder the detection of sensory nerve action potentials. Furthermore, asymptomatic older patients often exhibit neurogenic changes, which can complicate the differential diagnosis between lumbosacral and peripheral entrapment [5].

Treatment methods

Treatment for deep gluteal syndrome is divided into conservative and surgical approaches, although there, is no universally accepted protocol [4]. First-line treatment options include physical therapy, NSAIDs, muscle relaxants, and rest [3].

Physical therapy should focus on the site of sciatic nerve compression [5] and involve pelvic muscle stretching exercises (including the piriformis muscle), gluteal muscle massage, transcutaneous electrical nerve stimulation, improvement of spinal flexibility, and intensive therapy focused on the piriformis [11]. Additionally, exercises may be assigned to strengthen specific muscles, such as the hip adductors, flexors (iliopsoas and rectus femoris), the internal obturator muscle, and posterior lower limb muscles (gluteus maximus, the hamstring muscle group, gastrocnemius, and soleus) [11].

Particular attention is given to the piriformis muscle in cases of its hypertrophy. In cases of DGS, this muscle is often shortened, with or without myofascial trigger points. Massage, acupuncture, and injections into the trigger points may help eliminate the issue [11].

Data from studies indicate that stretching exercises involving external rotation and passive adduction of the piriformis muscle, as well as mobilization of the lower lumbar spine and sacroiliac joint, are beneficial [11]. In addition, the piriformis stretch test, or FAIR, involving hip flexion, adduction, and internal rotation, is designed to stretch the piriformis muscle [5]. In cases of IFI, the hip abductor muscles should be included in the physical therapy program. Light, low-intensity movement exercises can promote healing of impaired muscle tendons and reduce fibrotic changes [8]. Lastly, posture correction can help alleviate the symptoms. Physical therapy is supplemented by medications: nonsteroidal anti-inflammatory drugs, which may be combined with muscle relaxants to reduce muscle spasms. Pregabalin or gabapentin may also be considered for the management of neuralgia [4].

If the desired results are not achieved with physical therapy, local injections of an anesthetic (e.g., mepivacaine or lidocaine) or a corticosteroid (e.g., methylprednisolone) may be administered into the relevant muscle to alleviate the symptoms [5]. Local injections around the sciatic nerve are also an option. Data from studies indicate that injections of a specific solution (1 mL of corticosteroid + 4 mL of local anesthetic + 20 mL of saline) under ultrasound guidance helped alleviate symptoms

in patients with refractory piriformis syndrome due to an anti-inflammatory effect and mechanical displacement of the nerve away from the site of compression. Although, symptoms recurred in half of the subjects after approximately 5 weeks [8]. A separate randomized controlled trial, which compared two different mixtures (betamethasone + 2% lidocaine solution; 2% lidocaine solution), found no significant added benefit of corticosteroids in the treatment of piriformis syndrome [11]. In addition, intramuscular botulinum toxin injections may prove to be effective in patients with DGS resulting from muscle hypertrophy [8]. The toxin inhibits presynaptic acetylcholine release, causing temporary muscle paresis and atrophy [3]. Botulinum toxin injections are administered intramuscularly in a mixed solution (100 U botulinum toxin + 6 mL 0.5% bupivacaine + 4 mL 1% lidocaine) under ultrasound or CT guidance [4]. In one study evaluating morphological changes in the piriformis muscle following botulinum toxin injections, a significant reduction in the muscle density, volume, and fatty infiltration was observed [8]. In another retrospective review examining CT-guided injections into the piriformis muscle and perineural spaces, significant added value of Type A botulinum toxin was found when administered in combination with local anesthetics (lidocaine and bupivacaine) [11]. Ultrasound-guided prolotherapy is another form of treatment, involving the injection of irritant solutions, such as hyperosmolar dextrose, in order to achieve a localized inflammatory sequence with fibroblast proliferation and collagen deposition in affected ligaments and tendons [12]. Additionally, it can help modulate pathological innervation and neovascularization at the bone-tendon junction, which is characteristic in enthesopathies [13]. However, its application in treating DGS is novel and has rarely been described in medical literature [12].

If conservative therapy proves ineffective, surgical treatment should be considered. The choice of intervention depends on the diagnostic quality and the specific clinical situation. Response to targeted injections may be a good predictive indicator of surgical success [5]. Surgical options are also determined by the general anatomy of the area where sciatic nerve compression occurs [4]. There are reports in the literature on the efficacy of open and minimally invasive surgeries, including sciatic nerve decompression, in treating pathologies in the posterior hip region that may cause deep gluteal syndrome [5]. Nowadays, the increasingly widespread use of endoscopic surgical techniques allows for successful visualization of the entire course of the sciatic nerve in the deep gluteal space and enables treatment which would be tailored to the various etiological factors of DGS. Nevertheless, this treatment approach requires significant surgical experience and an excellent understanding of the endoscopic anatomy [3]. A retrospective case series, including 35 patients, described the efficacy of various methods for endoscopic sciatic nerve decompression, including resection of abnormal fibrovascular bands, piriformis tenotomy, release of the obturator internus and quadratus femoris muscles, and release or resection of hamstring tendon scarring [11]. SN decompression may also include resection of the lesser trochanter (for treatment of IFI) and arthroscopic SN neurolysis [8]. In the postoperative period, early patient mobilization is crucial to avoid adhesions in the deep gluteal space. The main principle is ensuring hip joint mobility and reducing sciatic nerve stretching with a knee brace [8].

Clinical significance of sciatic nerve anatomical variations

The relationship between sciatic nerve anatomical variations and the piriformis muscle has been commonly cited, whether in textbooks or individual case reports and case series, as one of the most frequent etiological factors in the development of piriformis syndrome [9]. However, to date, no larger study confirming a definitive link between anatomical variations of the sciatic nerve and piriformis syndrome has been published in the medical literature. It is thought that, in the case of a sciatic nerve variation, where the nerve or one of its branches runs directly through the piriformis muscle, there is a higher risk of sciatic nerve compression when the muscle contracts [16].

A majority of studies have not found evidence of such a connection. In a 2010-dated meta-analysis that included all available case series data, the prevalence of sciatic nerve anatomical variants was found to be not statistically significantly different between general population cadaveric studies and surgical case series of PS [9]. In a retrospective study implemented in 2018 involving a cohort of 783 hip MRI images, no statistically significant correlation was found between sciatic nerve variations and sciatica or gluteal pain, both potentially related to piriformis syndrome [9]. A systematic review conducted in 2010 compared the prevalence of SN anatomical variations between the general population (based on cadaveric studies) and among individuals with piriformis syndrome who had undergone surgery. The study found no statistically significant difference between the two groups (16.9% in the general population and 16.2% in the population with PS) [11]. Thus, based on currently available research, there is no conclusive evidence that the existence of sciatic nerve anatomical variations directly contributes to the development of deep gluteal syndrome under normal circumstances.

Yet, it is noteworthy that although patients with DGS may not always present with SN variations, and asymptomatic individuals might still do, it is still crucial to recognize this anomaly. Any other etiological factor, such as trauma to the gluteal region, overuse or overexertion of the surrounding muscles, fibrous bands, or prolonged sitting, could more easily trigger sciatic nerve entrapment when an SN variation is present as a predisposing factor [7]. Two separate cases of piriformis syndrome associated with Type C sciatic nerve variation help to highlight this point [17]. One patient complained of constant left hip and leg pain following blunt trauma to the hip region, while the second patient reported chronic pain in the right leg and buttocks, which continued after lumbar fusion surgery. In both cases, severe sitting intolerance was recorded, which suggests sciatic nerve compression. Hip MRI confirmed Type C SN variation, which occurs at a frequency of 0.3%–7.6% [17]. Both patients later received transgluteal complete circumferential SN decompression surgery, following conservative treatment with insufficient results. Significant improvement in symptoms was reported by both patients after the operation [17]. While the exact mechanism of the abrupt occurrence of the sciatic nerve compression symptoms is unknown, inflammatory mediators may contribute to nerve irritation following trauma to the buttock and the subsequent inflammation of the piriformis. The inflamed and spastic muscle may then potentially compress the SN [17]. Under these circumstances, an anatomical variation of the sciatic nerve could precipitate symptoms of nerve compression.

Limitations

This review has several limitations. First, it is a narrative literature review rather than a systematic review or meta-analysis. The search was limited to selected databases, English-language publications, and articles published between 2015 and 2026; therefore, some relevant studies may not have been included. Second, the available evidence on the association between sciatic nerve anatomical variations and deep gluteal syndrome is limited and heterogeneous, consisting mainly of cadaveric studies, imaging-based retrospective studies, small case series, and case reports. Third, the lack of universally accepted diagnostic criteria for deep gluteal syndrome makes it difficult to compare findings across studies. Finally, the current literature does not allow a direct causal relationship to be established between sciatic nerve anatomical variations and the development of deep gluteal syndrome. Further prospective studies with larger cohorts and standardized diagnostic protocols are needed to clarify this relationship.

Conclusions

Deep gluteal syndrome, first described in 1999, is a multifactorial pathology involving extrapelvic, non-discogenic compression of the sciatic nerve in the deep gluteal space, most commonly caused

by the piriformis muscle. The syndrome is characterized by pain and/or dysesthesia in the gluteal, hip, and/or posterior thigh regions, as well as symptoms resembling radiculopathy. The sciatic nerve anatomy typically varies depending on the pattern of its branching in the deep gluteal space and its relationship to the piriformis muscle. Variations are grouped into types A–F according to the Beaton and Anson classification, although rarer cases of variable sciatic nerve courses exist that do not fit into any of the aforementioned types. The most common variant worldwide is the Type B variant (the sciatic nerve branches off early in the pelvic region, the common peroneal nerve pierces the piriformis muscle, and the tibial nerve follows its typical course). Although anatomical variations of the sciatic nerve alter its biomechanics, according to the latest literature, there is no direct and statistically significant link with the development of deep gluteal syndrome. Anatomical variations of the sciatic nerve may be considered predisposing factors that increase the risk of developing deep gluteal syndrome in the presence of additional etiological factors. However, the evidence is related to case reports, and more comprehensive scientific studies are needed to verify this assumption. A deep understanding of the deep gluteal space anatomy and a careful assessment of sciatic nerve course in the region can help in making a more accurate diagnosis of deep gluteal syndrome, developing a specific etiology-based treatment plan, and avoiding iatrogenic injuries during procedures and surgeries, thus reducing the likelihood of complications.

Disclosures

The authors declare that there is no conflict of interest, and that no specific financial support has been provided in relation to this project.

Author contributions

M. L.: methodology, writing – original draft preparation, formal analysis.
L. N.: conceptualization, methodology, writing – review and editing.

References

1. Kegoye ES, Ojewale AO, Ezekiel W, Usman IM, Aigbogun EO, Adewale AO, et al. Morphologic and morphometric bilateral analysis and sexual dimorphism in sciatic nerves of adult cadaveric specimens in Uganda. *BMC Musculoskel Disord*. 2025;26(1):422. doi:10.1186/s12891-025-08641-9
2. Tomaszewski KA, Graves MJ, Henry BM, Popieluszko P, Roy J, Pękala PA, et al. Surgical anatomy of the sciatic nerve: A meta-analysis. *J Orthop Res*. 2016;34(10):1820-1827. doi:10.1002/jor.23186
3. Carro LP, Hernando MF, Cerezal L, Navarro IS, Fernandez AA, Castillo AO. Deep gluteal space problems: piriformis syndrome, ischiofemoral impingement, and sciatic nerve release. *Muscles Ligaments Tendons J*. 2016;6(3):384-396. doi:10.11138/mltj/2016.6.3.384
4. Kanumuri S, Andrew D, Kumar S, Chhabra A. Piriformis Syndrome and Deep Gluteal Syndrome: Presentation, Diagnostic Imaging, and Management. In: Davies M, Botchu R, Iyengar KP, eds. *Imaging of the Hip & Bony Pelvis. (Medical Radiology)*. Springer; 2024:369-384. doi:10.1007/174_2024_481
5. Martin HD, Reddy M, Gómez-Hoyos J. Deep gluteal syndrome. *J Hip Preserv Surg*. 2015;2(2):99-107. doi:10.1093/jhps/hnv029
6. Batool N, Azam N, Moafa HN, Hafeez A, Mehmood H, Imtiaz N, et al. Prevalence of piriformis syndrome and its associated risk factors among university students in Pakistan: a cross-sectional study. *BMJ Open*. 2025;15(1):e092383. doi:10.1136/bmjopen-2024-092383
7. Hernando MF, Pérez-Carro L, Cerezal L. Imaging Assessment in Posterior Hip Pathology. In: Martin H, Gómez-Hoyos J, eds. *Posterior Hip Disorders*. Springer; 2018:59-146. doi:10.1007/978-3-319-78040-5_5
8. Geler Külcü D. Deep Gluteal syndrome: An underestimated cause of posterior hip pain. *Turk J Phys Med Rehabil*. 2024;70(1):4-16. doi:10.5606/tftrd.2024.14668

9. Bartret AL, Beaulieu CF, Lutz AM. Is it painful to be different? Sciatic nerve anatomical variants on MRI and their relationship to piriformis syndrome. *Eur Radiol*. 2018;28(11):4681-4686. doi:10.1007/s00330-018-5447-6
10. Polat S, Tunç M, Aksay UC, Sönmez Eİ, Özşahin E, Göker P. Assessment of the piriformis muscle and piriformis syndrome via Web of Science database: A bibliometric analysis. *Medicine (Baltimore)*. 2024;103(47):e40416. doi:10.1097/MD.0000000000040416
11. Lo JK, Robinson LR. Piriformis syndrome. *Handb Clin Neurol*. 2024;201:203-226. doi:10.1016/B978-0-323-90108-6.00002-8
12. Yoon Y, Lam KHS, Lee J, et al. Ultrasound-Guided Prolotherapy for Sciatica Secondary to Sacrospinous Ligament Calcification: A Potential and Previously Overlooked Etiological Factor in Deep Gluteal Syndrome-A Case Report and Literature Review. *Life (Basel)*. 2025;15(9):1486. doi:10.3390/life15091486
13. Yoon Y, Sung D, Lee J, Lam KHS. A Case Report of Deep Gluteal Syndrome Secondary to Piriformis Enthesopathy Successfully Treated With Ultrasound-Guided Dextrose Prolotherapy. *Cureus*. 2026;18(1):e102370. doi:10.7759/cureus.102370
14. Parikh HB, Matcuk GR, Leong M, Vrahas MS, Seruya M. Amyloid Deposition: An Unusual Case of Deep Gluteal Syndrome and Sciatic Nerve Compression: A Case Report. *JBJS Case Connect*. 2022;12(3):10.2106/JBJS.CC.22.00075. doi:10.2106/JBJS.CC.22.00075
15. Koh E. Imaging of peripheral nerve causes of chronic buttock pain and sciatica. *Clin Radiol*. 2021;76(8):626.e1-626.e11. doi:10.1016/j.crad.2021.03.005
16. Jabbari B. Botulinum Toxin Treatment of Piriformis Syndrome. In: *Botulinum Toxin Treatment of Pain Disorders*. Springer; 2022:255-267. doi:10.1007/978-3-030-99650-5_13
17. Son BC, Lee C. Piriformis Syndrome (Sciatic Nerve Entrapment) Associated With Type C Sciatic Nerve Variation: A Report of Two Cases and Literature Review. *Korean J Neurotrauma*. 2022;18(2):434-443. doi:10.13004/kjnt. 2022.18.e29