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REVIEW ARTICLE

Deep Gluteal Syndrome: Diagnostic Assessment and Surgical Treatment of Non-discogenic Sciatica in Our Experience

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Abstract

Deep gluteal syndrome (DGS) is a term applied to a medical condition consisting of pain and paresthesias radiating from the buttock area along the sciatic nerve territory and not related to a discogenic etiology.

The spectrum of pathologies related to non-discogenic sciatica is wide. Thus, this syndrome is a complex clinical entity that was difficult to diagnose at least until recent times, when new imaging modalities offered the possibility to investigate the lumbosacral plexus and the intrapelvic/subgluteal course of the sciatic nerve. A thorough diagnostic assessment has resulted in the detection of myriads of causative pathologies, many of which are amenable of surgical treatment with favorable outcome.

In this work, the authors present their experience in the management and surgical treatment of DGS and review its different etiologies.

Keywords: Pelvic MRI, MR neurography, sciatica, piriformis syndrome, neurofibromatosis, ectopic endometriosis.

Introduction

Sciatica is usually described as a unilateral, well-localized leg pain with a sharp, shooting or burning quality radiating from the buttock to the ankle or foot, along the posterior-lateral aspect of the leg (the dermatomal distribution of the sciatic nerve). It is often associated with paresthesias in the same territory. Neurological deficits can occasionally be part of the clinical presentation.

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The algo-paresthetic symptomatology is usually due to pathologies of the lumbar spine (such as herniated disks or canal stenosis) but in a subgroup of these patients, unrelenting sciatica can be secondary to a wide spectrum of intrapelvic or extrapelvic pathologies (tumorous, consequent to entrapments, post-traumatic, vascular or inflammatory disorders etc). Due to its etiological heterogeneity, the more descriptive term "Deep Gluteal Syndrome" (DGS) is often used when referring to non-discogenic sciatica[1].

In recent years, the introduction of pelvic MRI studies combined with high resolution neurography has undoubtedly offered a sensitive modality in identifying and characterizing extraforaminal ventral rami, lumbosacral plexus structures and the intrapelvic/extrapelvic course of the sciatic nerve[2,3,4] : these new imaging modalities applied to the study of peripheral nerves have revealed a variety of conditions related to the onset of DGS and previously often undetected, due to their rarity. Consequently, accurate diagnostic assessment has directly lead to better options of treatment, especially in those cases favorably amenable to surgery.

In this report, the authors present their experience in the surgical treatment of DGS and review its multifactorial etiology.

A description of MRI protocols and specific findings for the causative pathologies related to DGS is beyond the scope of this paper.

Review of etiologies in DGS

A. Cysts and tumors

Primary tumors of the sciatic nerve and masses causing compression along the sacral plexus and the intra-pelvic / subgluteal course of the sciatic nerve constitute the most frequent conditions treated by peripheral nerve surgeons.

A wide range of pseudotumoral lesions as well as benign and malignant tumors is described in the literature[1].

Among the benign primary lesions of the sciatic nerve, schwannomas and neurofibromas, perineuriomas, lipomatosis and intraneural lipomas can be encountered. The primary malignant lesions are represented by the umbrella term MPNST (Malignant Peripheral Nerve Sheath Tumor).

The occurrence of schwannomas (the most common neurogenic tumor) in the lumbosacral plexus or along the subgluteal course of the sciatic nerve is statistically rare (Figure 1) and neurofibromas are exceptionally encountered in the general population. Intrapelvic primary lesions of the sciatic nerve are much more frequent in patients affected by neurofibromatosis (NF), a phakomatosis encompassing about a dozen clinical presentations[5], each related to different specific genetic anomalies and varying statistical incidence. NF I (also known as Von Recklinghausen's disease) is the most frequent form (1 out of 4-5000 individuals) and is associated with peripheral nerve tumors in one third of cases: neurofibromas are the most frequent histotype encountered in the NF subgroup (Figure 2).

Other primary benign lesions are perineuriomas (also known as benign hypertrophic neuropathy), fibrolipomatous hamartoma (lipomatosis) and intraneural lipoma but their occurrence is exceptional.

Among non-primary benign lesions affecting the sciatic nerve, lipomas, osteochondromas and ganglion cysts (Figure 3) are most commonly related to DGS onset. Other benign conditions include ossification of the sacrospinous ligament and tumoral calcinosis. The incidence of MPNSTs is very low and they often develop after radiotherapy (Figure 4).

Common *non-primary malignant lesions* include colorectal carcinoma, followed by uterine, prostatic and ovarian tumors. Lymphomas can cause DGS due to compression on the sciatic nerve by enlarged lymph nodes or extranodal involvement of soft tissues (e.g. piriformis and gluteus muscle). The possibility of direct lymphoma infiltration of the sciatic nerve is extremely rare. Soft tissue sarcomas and intramuscular metastasis have also been described but are certainly uncommon.

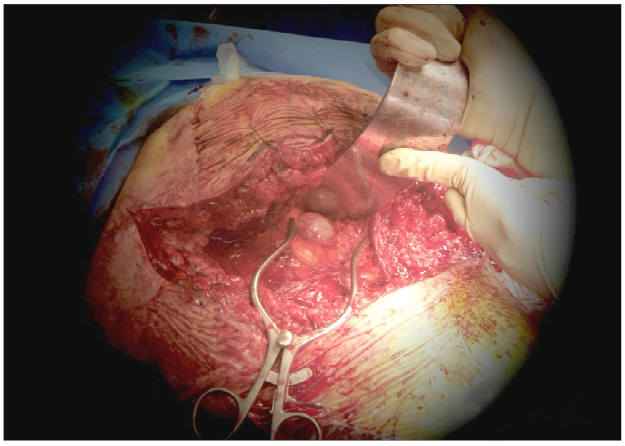

Fig. 1 Schwannoma of the subgluteal sciatic nerve

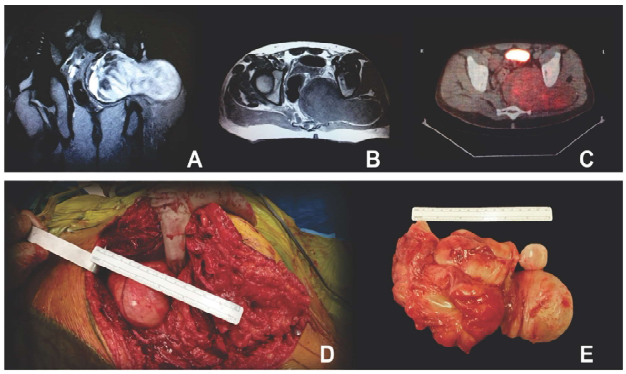

Fig. 2 Bulky intrapevic /extrapelvic dumbbell-shaped neurofibroma in 26 year old patient with NF1.

Fig. 2 (A and 2B) Coronal and axial scans of pelvic MRI.

Fig. 2 (C) FDG PET scan.

Fig. 2 (D) Exposure of the extrapelvic pole of the tumor.

Fig. 2 (E) The specimen of the tumor after en-bloc removal

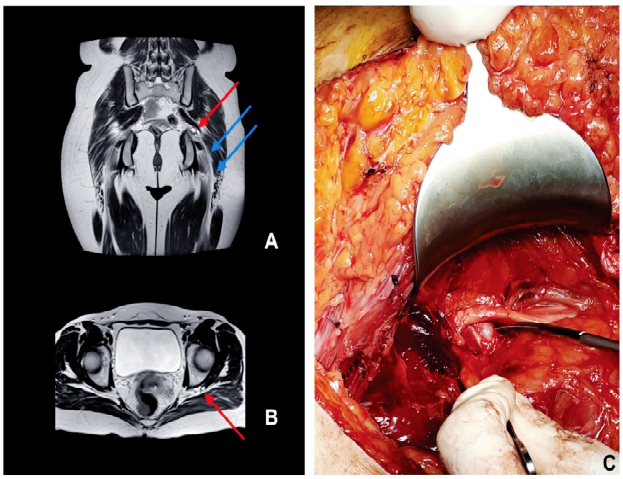

Fig. 3 Ganglion cyst (red arrow) compressing the subgluteal sciatic nerve (blue arrow)

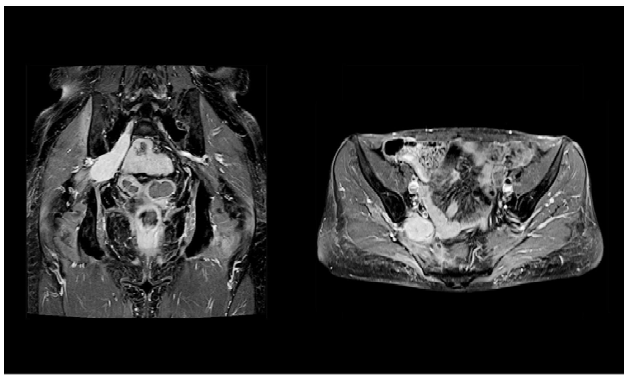

Fig. 4 8 cm long- MPNST of the sciatic nerve with intrapelvic/ extrapelvic growth in a 44 year old lady, previously submitted to radiotherapy due to ovarian cancer.

B. Entrapments

Piriformis syndrome is the best known entrapment syndrome acknowledged to cause DGS, literature estimating its incidence between 5% and 36% of cases[1].

Primary and secondary piriformis syndromes are described. The former is rare and related to the presence of an anatomical cause such as pyriformis muscle hypertrophy and variations in the sciatic nerve course[1,6,7] whereas the latter (consequent to macro and microtrauma, inflammation, infection etc.) seem to be far more common.

Other forms of entrapments have been described in the literature: obturator internus and gemellus syndrome, gluteal compartment syndrome, gluteal maximus contracture, compressions following quadratus femoris and ischio-femoral space pathology, ischio-femoral impingement and harmstring origin enthesopathies[1,8]. The occurrence of these syndromes is very rare.

C. Vascular etiology

DGS can be consequent to compression of the sciatic nerve along its course due to vascular anatomical anomalies or pathologies.

A persistent sciatic artery (PSA) is described among the causes of DGS[1,9]: it is a rare anomaly whose incidence is estimated as ranging from 0.025% to 0.04 of the population. In 12 to 32% of cases, the PSA can be bilateral[9].

Two forms of PSA are described. In the first type, the PSA is hypoplastic and the major blood supply to the lower limb is from the superficial femoral artery; in the second form, the PSA just continues to the popliteal artery with a minimal change in size and the superficial femoral artery is small, providing only collateral branches to the lower extremity.

PSA seems to be prone to atherosclerotic degeneration and may even be associated with aneurysmal formation in 46.1 % of the cases[1]. A PSA aneurysm can cause compression on the sciatic nerve with a clinical presentation consisting of painful pulsatile buttock mass associated with algic radiation in the sciatic territory. It may eventually result in lower limb ischemia due to thrombosis or distal embolization; other complications can be its rupture or fissuring.

Similarly, aneurysms or pseudo-aneurysms of the iliac artery (especially the internal iliac artery) and its branches, arteriovenous malformation or arteriovenous fistula and varicosities can be responsible for the onset of DGS. Another known vascular etiology of DGS is thrombosis of the superior gluteal artery; it usually manifests as buttock claudication after about 150 metres of walking and pain typically disappears with rest.

D. Gynecological etiology

Pregnancy is a well-known cause of DGS. It has been reported that up to 50% of pregnant women complain about back pain and sciatica; the gravid uterus can cause direct compression on the lumbosacral nerve roots or their ischemia by compression on the aorta or vena cava[1].

Hormonal factors induce increases in pelvic stretch and opening up of the sacro-iliac joints while the progressive lordosis tilts the pelvis forward, moving it into increased flexion and leading to elongation and traction on the gluteal muscles in the hip and pelvis. This leads to the possible development of a secondary pyriformis syndrome.

The possibility of a unilateral foot drop resulting from injury to the lumbosacral trunk by the fetal head or by forceps during delivery has also been reported[1].

Lumbar and sciatic pain usually subsides after delivery but in some cases the algo-paresthetic symptomatology can be persistent even for several months afterwards. Occasionally the onset of DGS occurs during the postpartum period.

Beside pregnancy-related sciatica, retroversion of the uterus, adenomyosis and hysteromiomas as well as ovarian cysts or tumors are frequently implicated in the causation of DGS in women.

A very peculiar cause of DGS is catamenial sciatica consequent to ectopic endometriosis[1,10]. Endometriosis seems to affect between 1 and 5% of women of reproductive age but endometriosis–related sciatica is a very rare occurrence. Initially reported by Schlicke in 1946[11], the location of endometrial nodes at the root of the nerve or in the nerve itself is one of the rarest topographic variations of this pathology. It has been suggested that endometrial tissue can migrate from its original genital location to the sciatic nerve thanks to the presence of a peritoneal diverticulum or after retrograde menstruation through the fallopian tubes. In patients without other sites of endometriosis, another hypothesis is hematologic migration after vascular damage due to slight injury or surgical interventions. Endometriosis of the sciatic nerve seems to predominantly occur on the right side: apparently the sigmoid impedes the implantation of endometriotic nodes in the left sciatic nerve[10].

As it is well known, endometriosis is hormone-dependent and occurs in association with the menstrual cycle. In the initial stage of catamenial sciatica, the painful symptomatology is cyclical, usually starting 2-3 days before menstruation or during the period itself. Pain intensity is reported to be extremely severe and tends to gradually subside once menstruation is over. In the long run, pain-free intervals progressively reduce and after some years the algo-paresthetic symptomatology becomes continuous with remarkable re-exacerbation during the menstrual days. Sensorimotor deficits can occasionally occur. Differently from classic endometriosis, pregnancy is usually not associated with disappearance of the pathology.

E. Traumas

Pelvic traumas associated with fractures of the sacrum and the sacroiliac joint, femoral fractures, femoral head dislocation and acetabulum fractures are known to trigger the onset of DGS. Mal-union of ischium avulsions and trochanter fractures were also described among the causes of DGS. The algo-paresthetic symptomatology is related to mechanical compression on the nerves from displaced bone fragment or contact with the bony structures. In such cases MR neurography may be able to detect nerve contusion[1,4].

Gluteal hematoma formation following trauma, hip surgery, or medical conditions interfering with blood coagulation (such as anticoagulation therapy or haemophilia) can result into nerve damage either directly (as consequence of mechanical pressure on the nerve) or indirectly (due to ischemia following vasa vasorum compression). In such cases MRI signal characteristics vary according to the evolution of the hematoma, in terms of size (being affected by progressive reabsorption) and composition (as affected by hemoglobin degradation)[1].

F. Inflammatory and Infectious Etiology

DGS consequent to sacroiliitis is a frequently encountered clinical presentation among individuals with seronegative spondyloarthropathies. Distension of the periarticular bursae of the hip is also a recognized and frequent cause of DGS, as is bursitis of pyogenic or post-traumatic etiology. Gluteal and pelvic abscesses, myositis affecting gluteal, pyriformis or pelvi-trochanteric muscles and sciatic nerve sarcoidosis are also possible causes of the onset of DGS[1].

The Authors' experience

The authors are presenting a surgical series of 16 patients who suffered from DGS, seen over a period of 10 years: 11 males and 5 females, their age ranged from 22 to 73.

All the cases were operated by the first author during her activity in her country of origin and in UAE.

Etiologies of DGS in the authors' series

The etiologies of the cases in our series are the following: (Table 1 represents number of cases for each pathology)

Cysts and tumors

- One patient had a ganglion cyst (Figure 3)
- A subgluteal schwannoma was found in 4 patients (Figure 1). One of them was affected by schwannomatosis (a recently recognized form of neurofibromatosis with an annual incidence of 0.58 cases per 1000000 persons)[12] and presented with two lesions along the sciatic nerve course: a subgluteal schwannoma and another lesion on the proximal course of the nerve in the thigh.
- A subgluteal neurofibroma was found in a 25-year-old woman with NF 1. She had previously undergone surgical removal of several tumoral masses due to her genetic disease (including a brachial plexus MPNST).
- A bulky intraplevic neurofibroma with extrapelvic growth through the sciatic notch was related to

the onset of DGS in a 22-year-old man with NF 1 (Figure 2). This patient had also previously undergone surgery for removal of tumors related to his genetic condition.

- One MPNST of the sciatic nerve developed in a 44-year-old woman subjected to radiotherapy for ovarian cancer 7 years earlier (Figure 4)
- A soft sarcoma with intraplevic location at the sciatic notch, causing compression of the sciatic nerve in a 73- year-old woman.

Entrapments

- We had 2 patients with primary piriformis syndrome and 3 patients with secondary piriformis syndrome: the latter was always related to the occurrence of a post-traumatic intramuscular piriformis hematoma.

Gynecological etiology

- One patient had catamenial sciatica due to an ectopic location of endometriosis.

Infectious/inflammatory

- We encountered one case of gluteal abscess sequalae, consequent to intramuscular injections.

Preoperative clinical presentation in the authors' series

Unrelenting sciatic pain, often associated with paresthesias (mainly in the distal territory of the sciatic nerve), was the usual clinical presentation.

One of the patients diagnosed with secondary piriformis syndrome had sustained a post-traumatic lumbosacral trunk palsy: his neurological deficit had largely resolved before the procedure but sequalae of toe extensors deficit were left.

The patient with catamenial sciatica presented weak toe and foot dorsiflexion (M3+) in association with the algoparesthesic symptomatology .

Duration of symptoms is reported in table 2.

The mean preoperative VAS score was 7 ± 2.0. All the patients reported poor or inconstant relief of the algic symptomatology in spite of daily use of painkillers.

Diagnostic workup

All the cases in our series were subjected to lumbar spine MRI (to rule out spinal pathology), pelvic MRI and electrodiagnostic studies and were subsequently referred to the surgeon after completion of the diagnostic assessment.

FDG PET (Fluorodeoxyglucose-Positron Emission Tomography) Scan was included in the assessment protocol of the two patients with NF.

Surgical technique

Surgery was always performed under general anesthesia without muscle relaxants. Direct nerve stimulation was available and the procedures were always performed under magnification (loupes and operative microscope).

Table 1: Number of cases for each causative etiology in the Authors' surgical series

Findings	No. of cases	Remarks
Ganglion cyst	1	
Sub gluteal schwannomas	4	1 case had schwanomatosis
Sub gluteal neurofibroma	1	Known case of NF I
Intrapelvic neurofibroma with extrapelvic growth through the sciatic notch	1	Known case of NF I
MPNST	1	Past history of radiotherapy for ovarian cancer
Soft tissue sarcoma	1	
Primary piriformis syndrome	2	All cases due to a post traumatic intramuscular hematoma of the piriformis
Secondary piriformis syndrome	3	
Ectopic endometriosis	1	Presented with catamenial sciatica
Gluteal abscess	1	Due to intramuscular injections

Table 2: Duration of preoperative symptomatology

Findings	No. of cases	Duration of preoperative symptoms
Ganglion cyst	1	6 months
Sub gluteal schwannomas	4	Ranging from 6 to 16 months
Sub gluteal neurofibroma	1	8 months
Intrapelvic neurofibroma with extrapelvic growth through the sciatic notch	1	1 year
MPNST	1	5 years
Soft tissue sarcoma	1	4 months
Primary piriformis syndrome	2	Ranging from 1 to 3 years Ranging from 8 to 12 months
Secondary piriformis syndrome	3	
Ectopic endometriosis	1	7 years
Gluteal abscess	1	2 years

Patients were positioned prone with their legs being externally rotated at the hip joint in order to relax the gluteal muscle. The buttock area and the whole lower limb were prepped in order to clearly check the functional response after stimulation.

With the exception of the sciatic MPNST, all the cases were operated via Henry's approach[13] in order to expose the sciatic notch and the subgluteal sciatic nerve course under the buttock: this surgical technique is illustrated in Figure 5. The approach performed in the case of the sciatic nerve MPNST is illustrated in Figure 6.

After appropriate exposure of the sciatic nerve was achieved, the procedures were completed according to the etiology.

In entrapment syndromes, resection of the pyriformis muscle and external neurolysis for possible fibrous bands contributing to the compression on the sciatic nerve were performed.

In primary nerve lesions, tumors were removed working under the microscope and with constant use of nerve stimulation to isolate and identify nerve fibers in order to guarantee the preservation of the nerve structure.

In the cases of extrinsic tumors (e.g. for the removal of the ganglion cyst), the sciatic nerve was isolated and displaced using an elastic band, then the mass was progressively detached from the surrounding lanes and eventually removed. Even in the case of the dumb-bell shaped bulky neurofibroma, the sciatic nerve was progressively detached from the mass; its intrapelvic pole was carefully detached from the surrounding layers via smooth dissection across the sciatic notch. Once pelvic detachment was completed, the tumoral mass was carefully pulled out through the sciatic notch and eventually enucleated (en- bloc removal).

Postoperative management

Drains were removed between the second and third day. Patients were kept in bed for 5 days after the procedure. They gradually resumed sitting, standing and walking afterwards.

Radiotherapy was indicated in the cases of the sarcoma and MPNST.

Complications

Surgery was uneventful in all cases. None of the patients presented postoperative deficits.

Results

Complete resolution of the preoperative painful symptomatology (postoperative VAS score decreased to 0) was achieved in 15 patients. Thus, regular use of painkillers could be discontinued once the patients recovered from the surgery and were discharged.

The patient with the sarcoma presented a postoperative VAS score decreasing from 9 to 4 and was the only case where pain management therapy was prolonged.

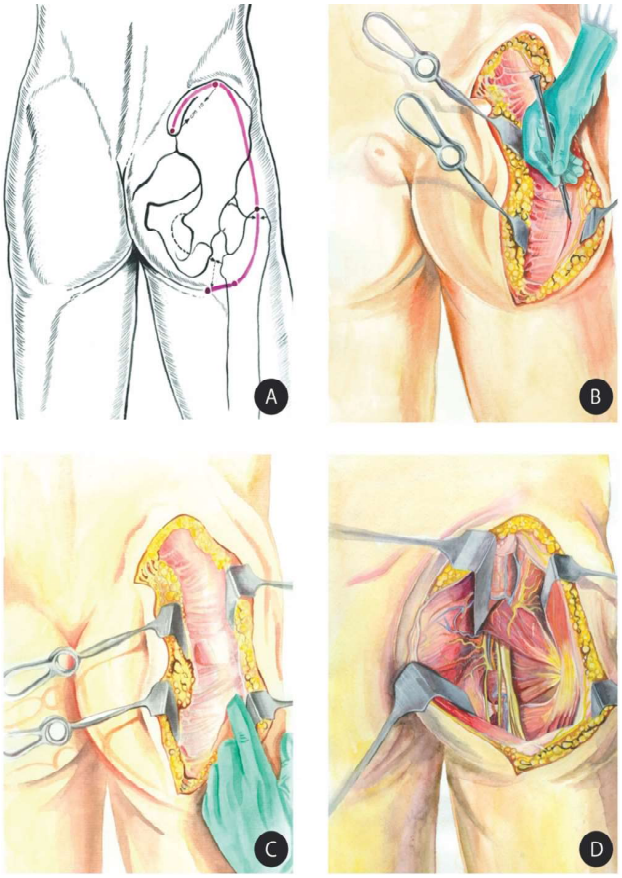

Fig. 5 Henry's approach to expose the subgluteal course of the sciatic nerve. **5 (A)** illustrates the "reverse C" skin incision, starting from the posterior superior iliac spine, partly continuing along the iliac crest, reaching the greater trochanter and along the gluteal fold. Attention must be paid while performing the skin incision to reach but not trespass the deep fascia in order to preserve the posterior cutaneous nerve running immediately below it. This precaution is necessary to preserve the sensitive collaterals supplying the posterior thigh skin and the perineum at the level of the gluteal fold.

5 (B). After its exposure, the gluteal fascia is vertically incised on the external surface of the greater trochanter.

5 (C). Sliding the index finger into the incision allows to palpate the posterior surface of the greater trochanter and to separate the planes, then progressively detaching the lateral insertion of the gluteus maximus. The supero-external border of the muscle (corresponding to the fusion of its superficial and deep aponeurosis) is delimited by a yellow-white line from the border of the gluteus medius: following this line, the gluteus maximus can be completely detached from the iliac crest, otherwise the surgeon would be constantly hindered from obtaining a proper view of the proximal sciatic nerve as it exits the notch. The muscle must be detached from its insertion on the femoral shaft, cutting down to the bone. The fascial edges are usually marked by 0 vicryl stitches every 5-6 cm to facilitate re-approximation of the gluteus maximus during closure.

5 (D). Once its insertion is completely cut, the whole gluteal "lid" is gently lifted and medially displaced thus allowing the exposure of the sciatic nerve course.

During closure, hemostasis must be performed while paying special attention to the gluteal vessels. If overstretched during medial displacement, these vessels may be a source of relevant bleedingonce the gluteal muscle is hinged back. Re- insertion of the gluteus muscle on the trochanter is performed carefully re-approximating the fascial edges (progressively removing the marking stitches) using vicryl 0. The superficial layers are closed after two or three drains are positioned. Subcutaneous suture with steristrips should be preferably used for the skin closure to provide good cosmesis.

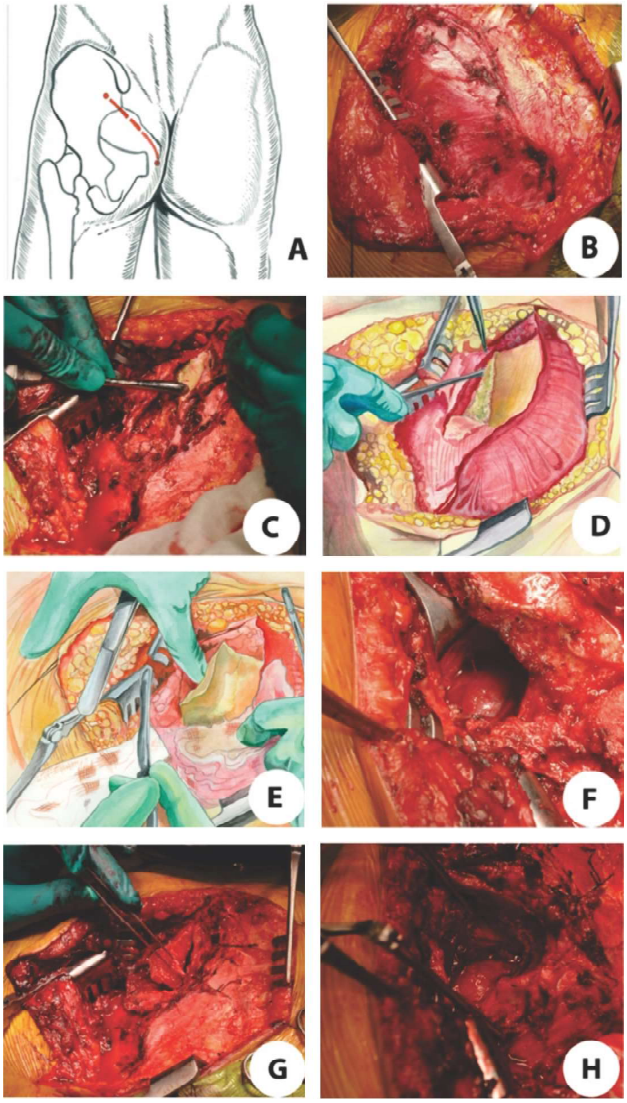

Fig. 6 The surgical approach performed in the removal of the MPNST of the sciatic nerve is illustrated. **6 (A).** Skin incision along the lateral border of the sacrum.

6 (B). Exposure of the medial insertion of the gluteus maximus on the later border of the sacrum

6 (C). Exposure of the sacral surface.

6 (D and E). Detachment of the medial insertion of the gluteus maximus from the sacrum.

6 (F). Exposure of the sciatic notch: the inferior pole of the tumor can be visualized.

6 (G). Removal of a triangular bone fragment.

6 (H). Bone removal resulting in enlargement of the sciatic notch provided better access to the tumor. Once excision of the neoplastic mass was achieved, the bone fragment was repositioned and the muscle and superficial layers were re-approximated. A subcutaneous drain was positioned during closure of the superficial layers.

Follow up

The patients who had catamenial sciatica, sequalae of subgluteal abscess, sarcoma and primary piriformis syndrome were all lost to follow up. The patient who had the ganglion cyst was operated in May 2017 and her last follow up (3 months after the surgery) confirmed persisting resolution of the preoperative symptomatology.

We had up to one year follow up for both patients with secondary pyriformis syndrome and for 3 patients with subgluteal schwannoma. In the remaining tumoral cases, up to a 4 years follow up was possible. All the patients were still pain-free at their last follow up.

For the MNST case, it must be mentioned that in spite of our indication for radiotherapy once histological confirmation of malignancy was received, such treatment was delayed and actually started 6 months after the procedure. A pre-radiotherapy control MRI detected a 1.5 cm recurrence of the tumor but no further change occurred in the investigations (regularly performed every 6 months for the next 3 and half years) following radiotherapy.

Discussion

The onset of sciatic pain is usually related to lumbar spine pathology (e.g. herniated disks or canal stenosis). Although this remains valid for the vast majority of cases, there is actually a percentage of patients with non discogenic sciatica that often remains untreated.

Moreover, herniated lumbar discs or canal stenosis are common findings even in asymptomatic patients: therefore it should be emphasized that the high sensitivity of lumbar spine MRI can sometimes contribute to misdiagnose. In a prospective MRI study of the lumbar spine in asymptomatic patients, Whitman et al[14]. found evidence of a herniated lumbar disc in 20% of patients in the age ranging between 20 and 59 years; in the 60 to 80 year old group the presence of a herniated lumbar disc or lumbar canal stenosis even rose up to 57%. Kulku and Naderi[15] demonstrated that in 20% of cases, sciatic pain is actually related to both discogenic and non discogenic origin.

Therefore, in practice, non discogenic etiology for sciatica is often overlooked or even erroneously ascribed to spinal pathology. Misdiagnosis usually leads to inappropriate treatment that may even result in spinal surgery without providing a successful outcome.

DGS multifactorial etiopathogenesis certainly contributes to the difficulties encountered during the diagnostic assessment. Nowadays, the remarkable progress achieved in imaging has fortunately enhanced the possibility of detecting conditions unknown in the past.

Patients presenting with sciatic pain should always be initially investigated with MRI of the lumbar spine given the statistical preponderance of spinal etiology. However, when MRI does not reveal any spinal pathology or when the findings aren't unmistakably linked to the patient's clinical presentation, further investigations with pelvic MRI should be mandatory. The possibility of detecting a tumoral lesion along the intrapelvic or subgluteal course of the sciatic nerve should be the first consideration to exclude after diagnosing DGS. If a tumoral etiopathology is confirmed, surgical indication should be given as soon as possible.

Pelvic MRI to detect the presence of masses along the lumbosacral plexus and the subgluteal course of the sciatic nerve should be regularly performed in NF patients as part of their assessment protocol, even if they are asymptomatic: these patients often harbor clinically silent tumors that may be responsible of the onset of a DGS only once the mass has achieved bulky size. A major issue in NF patients is the high statistical rate of malignant transformation of previously benign lesions, estimated as ranging from 8 to 13 % and even doubling in the "microdeletion" subgroup (accounting to up to 20% of the overall NF). It must be kept in mind that MRI is sometimes not able to detect signs of aggressive transformations in previously benign neurofibromas. Moreover, there is clear evidence that bulky lesions (> 6 cm diameter) are likely to be malignant. FDG PET scintigraphy should be performed in doubtful cases: a SUV (Standardized Uptake Value) higher than 3.5 is highly suspicious of malignancy and surgical indication should be given promptly.

Once spinal pathology and intrapelvic or subgluteal lesions are ruled out, the algo-paresthetic symptomatology is often attributed to pyriformis syndrome. As a matter of fact, some Authors claim this entrapment syndrome is likely to be under diagnosed[16,17,18]: of note, in 2005 Filler et al[18]. subjected 239 patients with non discogenic sciatica to MR neurography and interventional imaging and concluded that 67,8 % of them had a piriformis syndrome.

Yet it should be emphasized that whereas secondary piriformis syndromes are clearly acknowledged, the very existence of primary piriformis syndrome is even disputed in the medical community[19,20].

In our opinion the diagnosis of entrapment syndromes is not so straightforward. We reckon that even the MRI demonstration of piriformis asymmetry or other anatomical anomalies is not enough to diagnosis a piriformis syndrome unless the clinical history is adamantly in favor of such hypothesis. In their series of 14 patients with a diagnosis of piriformis syndrome and surgically treated with favorable outcome, Benson and Schutzer[21] found the presence of a larger piriformis muscle ipsilateral to the symptomatic side only in 2 cases; in 7 patients the piriformis muscle on the affected side was even smaller than the contralateral one. Russell et al[22]. evaluated the piriformis muscle and its relationship to the sacral roots and sciatic nerve studying 100 asymptomatic patients (200 piriformis muscles) on MRI: they detected a piriformis asymmetry (ranging between 3 to 8 mm) in 19 % of cases.

It must be also considered that a high variability is present in the relationship between the piriformis muscle and the sciatic nerve: 6 categories of anatomical variations were originally described by Beaton and Anson[23].In his review of multiple cadaveric studies and including over 6000 dissections, Smoll estimated 83 % of cases presenting the sciatic nerve running under the piriformis muscle (category A in Beaton and Anson's classification) while the other variations occurred in the remaining 17% with variable incidence[24]. Albeit no clear evidence is evident, it is likely that some anatomical variants might predispose to the onset of DGS: yet their mere presence is not enough to diagnose a piriformis syndrome.

In doubtful cases, the literature mentions the possibility of performing US (Ultrasound), CT (Computerized Tomography) or MRI guided-injections of steroids or local anesthetics into the piriformis muscle with the double aim of confirming the diagnosis and relieving the patient from the symptoms: we would reckon that this is not regularly efficacious in practical terms and not consistently providing useful information. Whereas electrodiagnostic tests contribute no additional information in case of (either primary or non-primary) tumoral masses detected along the course of the nerve, we believe that they still have a major role whenever an entrapment syndrome is suspected. Therefore indication for surgery in primary piriformis syndrome is resulting from a multifactorial evaluation and MRI is actually used to exclude other causes more than to confirm the entrapment pathology.

Recently, the concept of "dynamic entrapment" has been introduced in the diagnostic assessment of peripheral nerve pathologies and it seems to be applicable even to DGS. Using dynamic MRI, Makhsous[25] demonstrated that the soft tissue overlying the ischial tuberosity becomes significantly thinner in a seated posture than it is in the supine position. These findings could point towards the possibility that in some patients the changes in the nerve position during flexion, internal rotation and adduction (causing its lateralization and anterior excursion) can trigger the onset of DGS. However, further evidence is still required to prove this theory.

DGS related to vascular etiology is certainly an exceptional occurrence. In such cases, the diagnostic assessment must include pelvic MRI and angiography. We have no experience with DGS due to vascular pathologies but endovascular intervention or surgical treatment usually lead to the resolution of symptoms, according to what is reported in the literature[1,9].

Obstetrical and gynecological - related DGS is unfortunately very often underdiagnosed. Nevertheless gynecological diseases should be borne in mind, especially when the right side is affected: women in their late 30's or premenopause period and presenting symptoms of DGS should undergo a thorough gynecological examination. Especially when surgical indication for spinal surgery has been given, neglecting the possibility of gynecological etiology in sciatica can lead to false negative cases receiving unnecessary and ineffective treatments.

Finally, we would like to state a few closing remarks concerning the surgical treatment in our series. Henry's approach was our choice in all the cases (with the exception of the MPNST of the sciatic nerve) as in our experience it has proven to be a valid surgical

strategy providing the full visualization of the subgluteal course of the sciatic nerve: in our opinion, this is an absolute requisite in the surgical treatment of DGS, especially during the removal of a tumor.

We favor Henry's approach even in the surgical treatment of piriformis syndrome. The transgluteal approach[26] certainly minimizes dissection and spares muscle attachments, (consequently diminishing recovery time), but provides a more focal exposure of the sciatic nerve and might be exposed to higher rate of postoperative bleeding even after meticulous hemostasis during closure.

Up to the present now, we have not experience with endoscopy in the treatment of DGS. However the reports in literature illustrate favorable outcome and very low rate of complications[27,28,29,30].

Conclusions

Patients with unrelenting sciatica are commonly encountered in clinical practice. Their usual diagnostic assessment include MRI studies of the lumbar spine and in the vast majority of cases causative abnormalities such as herniated lumbar disks or canal stenosis are detected. However, within this population there is a subgroup of patients affected by non discogenic sciatica, consequent to pathologies of the lumbosacral plexus and the sciatic nerve along its intrapelvic and extrapelvic course.

Once routine protocols for MR imaging of the lumbar spine have ruled outs pinal pathologies, patients should be regularly subjected to MRI study of the pelvis with neurography of the sciatic nerve. The spectrum of pathologies related to non discogenic sciatica is extremely wide but once the diagnostic assessment has come to a correct conclusion, it can redirect subsequent treatment, especially in those cases amenable to surgery. In our experience, immediate resolution of the preoperative algoparesthetic symptoms has consistently occurred and follow up has demonstrated a favorable outcome in the vast majority of cases.

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