Original article

Laparoscopic approach to intrapelvic nerve entrapments

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Abstract: It has been well-established that a large portion of the lumbosacral plexus is located intra-abdominally, in the retroperitoneal space. However, most of the literature descriptions of lesions on this plexus refer to its extra-abdominal parts whereas its intra-abdominal portions are often neglected.The objective of this review paper is to describe the laparoscopic anatomy of intrapelvic nerve bundles, as well as the findings and advances already achieved by Neuropelveology practitioners.

Abreviations:
- LANN – Laparoscopic Neuronavigation
- LION – Laparoscopic Implantation of Neuroprosthesis

Keywords: Sciatic; Nerve Entrapment; Gluteal Pain; Neuromodulation; Laparoscopy.

INTRODUCTION

It is well established that a large portion of the lumbosacral plexus is located intra-abdominally in the retroperitoneal space[1]. However, descriptions of lesions on this plexus in most of the literature refer to its extra-abdominal course. The intra-abdominal location and the potential entrapment of nerves from lumbosacral plexus at these sites are often neglected in the literature[2].

In 2007, Possover et al.[3] described the Laparoscopic Neuronavigation (LANN) technique, opening the doors to accessing the retroperitoneal portion of the lumbosacral plexus through a safe, minimally invasive, and objective way. Since then, multiple causes of intrapelvic nerve entrapments have been described and a new field in Medicine – Neuropelveology – was created.

In this paper, we will review the laparoscopic anatomy of the intrapelvic nerve bundles, describe the symptoms and signs associated with intrapelvic neuropathies, as well as the diagnosis and treatment rationale of these conditions.

LAPAROSCOPIC ANATOMY OF THE INTRAPELVIC NERVES

Ilio-Hypogastric, Ilio-Inguinal And Genito-Femoral Nerves

These nerves are sensitive branches of the lumbar plexus. The ilio-hypogastric and ilio-inguinal nerves enter the retroperitoneal space emerging on the lateral border of the psoas muscle and follow anteriorly and distally to pierce the internal abdominal oblique muscle close to the antero-superior iliac spine[4]. The genito-femoral nerve emerges from the anterior border of the psoas muscle and its two branches leave the abdomen through the femoral (femoral branch) and inguinal (genital branch) canals. Their fibrotic entrapment is related to post-herniorhaphy inguinodynia[5] (Figure 1).

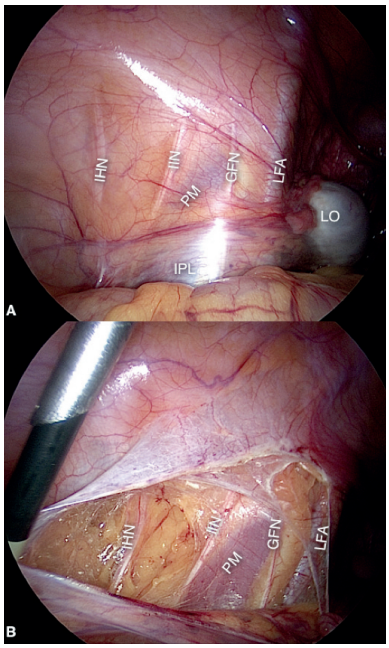

Figure 1. – Laparoscopic view the left abdominal wall exhibiting the Ilio-Hypogastric (IHN), Ilio-Inguinalis (IIN) and Genito-Femoralis (GFN) Nerves, with the overlying peritoneal intact (A) and exposed (B) [PM – Psoas Muscle; LO – Left Ovary; IPL – Infundibulopelvic Ligament; LFA – Left Femoral Artery

Femoral nerve

The femoral nerve is the largest motor and sensory nerve of the lumbar plexus. It emerges from the postero-lateral aspect of the psoas muscle and leaves the abdomen through the femoral canal (Figure 2) to innervate the quadriceps muscle and the skin covering the anterior thigh and medial aspect of the leg.

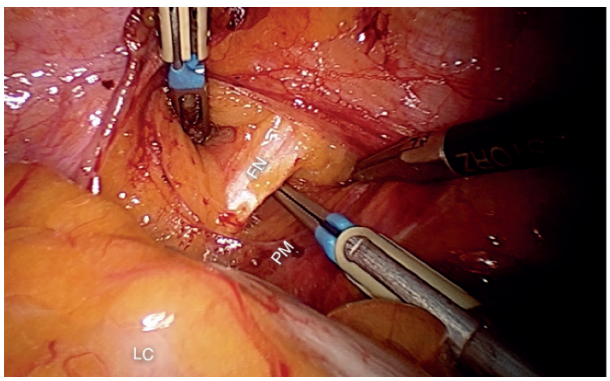

Figure 2. – The Left Femoral Nerve (FN) entering the retroperitoneal space on the posterolateral aspect of the Psoas Muscle (PM). (LC – Left Colon)

Nerves of the Obturator Space

The obturator nerve enters the obturator space at the level of the pelvic brim and leaves the pelvis through the obturator canal. It gives sensory branches to the skin of the medial thigh and motor branches to the hip adductors (Figure 3-A).

The lumbosacral trunk and the distal portions of the S1, S2, S3 and S4 nerve roots merge into the obturator space and form the sciatic and pudendal nerves (Figure 3-B).

The sciatic nerve is formed by the L4 and L5 fibers of the lumbosacral trunk and fibers from the S1, S2 and S3 nerve roots and leaves the pelvis through the greater sciatic notch. It gives out sensory branches to the upper gluteal region, postero-lateral thigh, leg, ankle and foot. It also controls the hip extensors, abductors and rotators, knee flexors, and all the muscles for the ankle and foot.

The pudendal nerve is formed by fibers of the 2nd, 3rd and 4th nerve roots and leaves the pelvis in the interligamentous plane between the sacrospinous and sacrotuberous ligament. It then enter the pudendal (Alcock's) canal. It provides sensory branches to the the perineal skin. It also sends motor branches to the perineal muscles and the anterior fibers of the levator ani muscles. Finally, there are direct motor and sensory branches from the S3 and S4 nerve roots to the posterior fibers of the levator ani muscle[6-8].

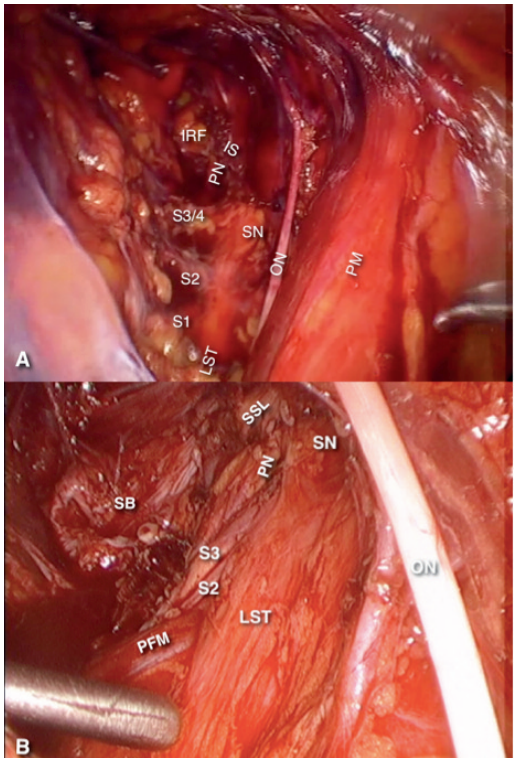

Figure 3. – Nerves of the obturator space (right side). Picture (A) is the final aspect of a laparoscopic approach to Alcock's Canal Syndrome, where the sacrospinous ligament has been transected to expose the pudendal nerve (PN). In picture B, the sacrospinous ligament (SSL) is intact. In both pictures, the internal and external iliac vessels are retracted medially. (ON – Obturator Nerve; PM – Psoas Muscle; SN – Sciativ Nerve; LST – Lumbosacral Trunk; PN – Pudendal Nerve; IRF – Ischiorectal Fossa; IS – Ischial Spine; SB – Sacral Bone; PFM – Piriformis Muscle)

Nerves of the Presacral and Pararectal Spaces

The superior hypogastric plexus, which is formed by fibers from para-aortic sympathetic trunk and gives rise to the hypogastric nerves. The hypogastric nerves run over the hypogastric fascia in an anterior and distal direction. After crossing about two thirds of the distance between the sacrum and the uterine cervix or the prostate, its fibers spread to join the pelvic splanchnic nerves (described below) to form the inferior hypogastric plexus (Figure 4). The hypogastric nerves carry the sympathetic signals to the internal urethral and anal sphincters, rectum and bladder, which cause detrusor relaxation and bladder contraction, thus promoting continence. They also carry proprioceptive and nociceptive afferent signals from the pelvic viscera[9].

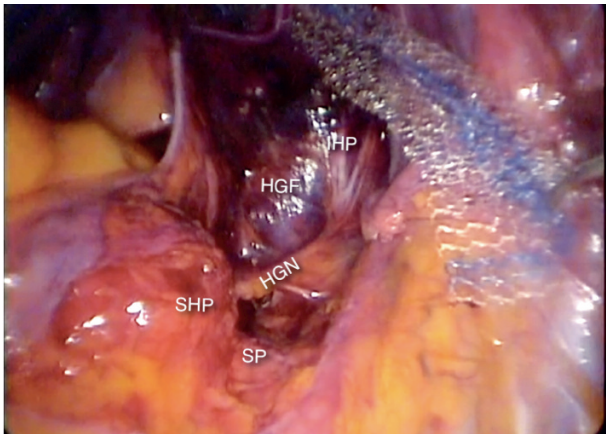

Figure 4. – The hypogastric nerve (HN) emerges from the Superior Hypogastric Plexus (SHP) at the level of the Sacral Promontory (SP) and runs anteriorly and distally, juxta-laterally to the Hypogastric Fascia (HF), to merge with the Pelvic Splanchnic Nerves to form the Inferior Hypogastric Plexus (IHP)

The lateral limit of the presacral space is the hypogastric fascia, which is the formed by the medial most fibers of the endopelvic fascia. The sacral nerve roots can be found juxta-laterally to this fascia (Figure 5). They leave the sacral foramina and run anteriorly and distally, lying over the piriformis muscle and crossing the internal iliac vessels laterally to them, to merge and form the nerves of the sacral plexus[10]. Before crossing the internal iliac vessels, they give out the thin parasympathetic branches called pelvic splanchnic nerves, which promote detrusor contraction and provide extrinsic parasympathetic innervation to the descending colon, sigmoid and rectum. They also carry nociceptive afferent signals from the pelvic viscera[9]. The pelvic splanchnic nerves join the hypogastric nerves to form the inferior hypogastric plexus in the pararectal fossae[10].

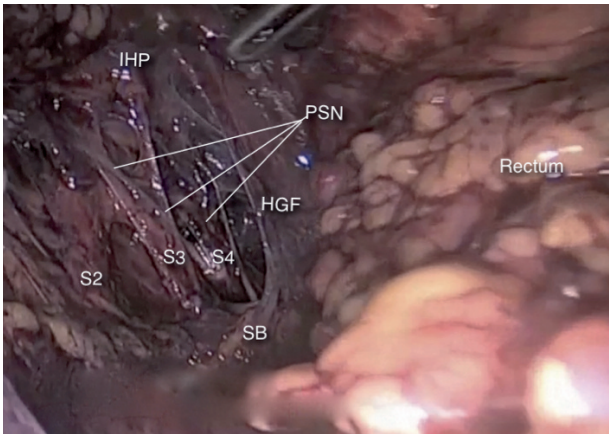

Figure 5. – The Sacral Nerve Roots (S2-S4) can be found juxta-laterally to the Hypogastric Fascia (HGF) and give origin to the Pelvic Splanchnic Nerves (PSN), which run anteriorly and distally to merge the Hypogastric Nerve and form the Inferior Hypogastric Plexus (IHP).

INTRAPELVIC NERVE ENTRAPMENT SYNDROME

Definition and Clinical Features

Nerve entrapment syndrome, or compression neuropathy, is a clinical condition caused by compression on a single nerve or nerve root. The symptoms and signs include pain, tingling, numbness, and muscle weakness on the affected nerve's dermatome and myotome[11]. Intrapelvic nerve entrapments are, therefore, entrapments of the intrapelvic portions of the nerves described in the previous sessions and will produce clinical features related to the affected nerves.

The above definition refers to the entrapment of somatic nerves. Autonomic nerve entrapment will produce visceral and vegetative symptoms, such as urinary frequency or urgency, dysuria, rectal pain, suprapubic and/or abdominal cramps and chills. However, as described, above, the sacral nerve roots give origin to both somatic and parasympathetic nerves. Therefore, entrapments of these roots will produce somatic (such as pain along the dermatome) and visceral (such as urinary and bowel dysfunction) clinical pictures.

In a concise manner, the main symptoms of intrapelvic nerve entrapments are:

- Sciatica associated with urinary symptoms (urgency, frequency, dysuria) without any clear orthopedic cause (spinal or deep gluteal nerve entrapment);
- Gluteal pain associated with perineal, vaginal or penile pain;
- Dysuria and/or painful ejaculation;
- Refractory urinary symptoms;
- Refractory pelvic and perineal pain.

It is important to emphasize that, due to the distance between both plexuses, intrapelvic nerve entrapments will usually cause unilateral symptoms.

Diagnostic Workup

Once the hypothesis of an intrapelvic entrapment is raised, it is mandatory to perform the topographic diagnosis, which is the determination of the exact point of entrapment. So far, careful neuropelveological evaluation, combined with a detailed medical history and neurological examination is the most reliable method for this.

To increase objectivity and accuracy of the diagnosis, we have been examining the use of high definition pelvic MRI and sacral plexus tractography, which is a technique for functional MRI of peripheral nerves[12]. Asymmetries and structures that could entrap the plexus are identified at MRI and those specific portions are investigated on tractography for any gaps in neural activity (Figure 6).

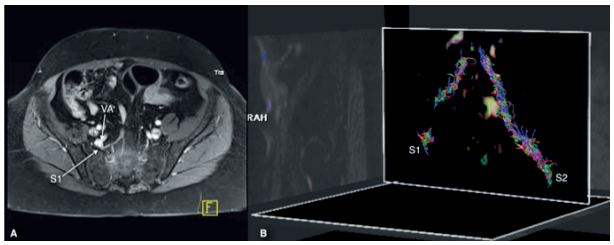

Figure 6. – A: contrasted MRI showing enlarged vessels (VA) in direct contact with S1 nerve root. B: Tractography showing a signal gap in S1.(Courtesy of Dr. Suzan M. Goldman, MD, PhD & Homero Faria)

Our results so far are very promising, but the accuracy of this method still needs to be investigated. Therefore, for further assurance, our next step is a diagnostic block, guided by ultrasound or fluoroscopy and performed by an intervention pain specialist; the exact point where a signal gap is identified at the tractography is infiltrated with 0.5mL to 1mL of lidocaine 0.5%. If a reduction of 50% or more in pain (VAS) is observed, the test is considered positive (Figure 7).

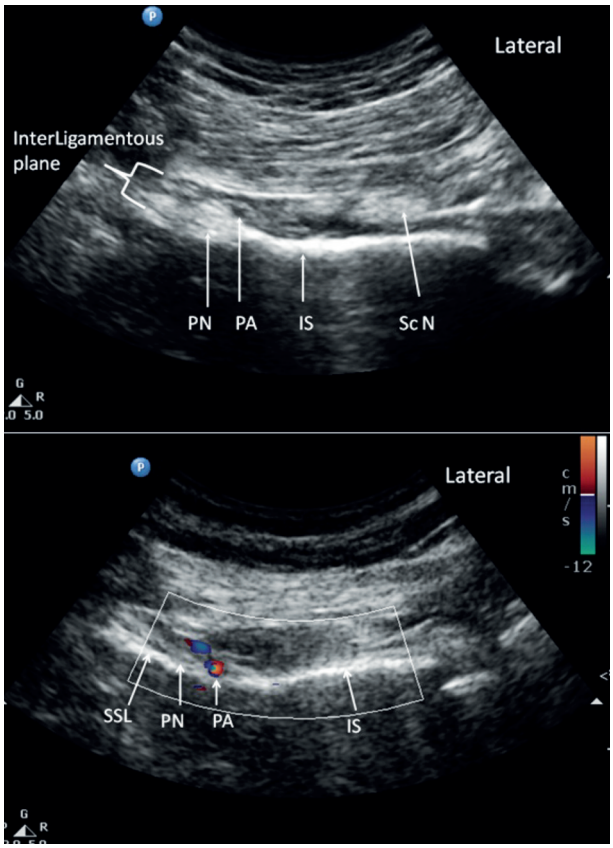

Figure 7. – (A) Ultrasound image of the interligamentous plane at the ischial spine where the pudendal artery and nerve are located between the sacrospinous and sacrotuberous ligaments. (B) Color Doppler of the same picture showed the pudendal artery. Reprinted with permission from Philip Peng Educational Series

Etiology of intrapelvic entrapments

Endometriosis

The first report of intrapelvic nerve entrapment was made by Denton and Sherill[13], who described a case of cyclic sciatica due to endometriosis in 1955. After that, some other case reports and small series were published, until 2011, when Possover et al[2] described the largest series so far, with 175 patients, all treated laparoscopically.

In endometriotic entrapments, the symptoms tend to be cyclic, worsening during the premenstrual and menstrual days and ameliorating or even disappearing during the post-menstrual period[2,14-15].

Evaluation consists of preoperative identification of the symptoms and determination of the topographical localization of the lesions mainly by clinical evaluation, although radiological examination (MRI) is sometimes required. Treatment is achieved by exploring all suspect segments of the plexus through laparoscopic approach, with radical removal of all endometriotic foci and fibrosis[2,14-15] (Figure 8).

The true incidence of endometriosis involving the sacral plexus is unknown, as this presentation of the disease is often neglected. On average, patients undergo four surgical procedures seeking to treat the pain before receiving the right diagnosis[2]. Moreover, about 40% of women with endometriosis refer unilateral pain on the inferior limb[16] and,

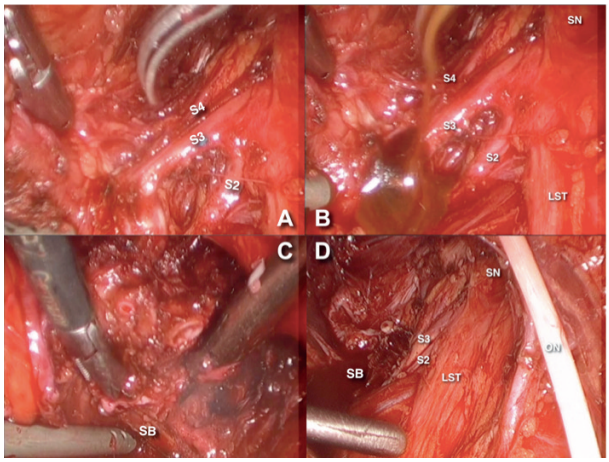

Figure 8. – A – after partial detachment of the nodule, allowing for visualization of S2, S3 and S4 nerve roots, S3 was found to be dilated on its proximal part; B – opening of the S3 nerve root sheath revealed an endometrioma inside the nerve; C – the nodule was detached from the sacral bone (SB); D – final aspect of the right pelvic sidewall; ON – obturator nerve; SN – sciatic nerve.

in 30% of patients with endometriosis, leg pain was demonstrated to be neuropathic[17], which leads to the conclusion that endometriotic involvement of the lumbosacral plexus is probably underdiagnosed and much more frequent than reported.

Fibrosis

This is one of the most frequent causes of intrapelvic nerve entrapments and possibly the most well-known etiology, since Amarenco[18] described the pudendal neuralgia in cyclists, in whom the pain is a consequence of fibrotic entrapment due to continued trauma.

Despite the historical aspect, however, surgical manipulation seems to be the most frequent cause of fibrosis over the sacral plexus (Figure 9). Among the surgeries with higher risks of inducing such kinds of entrapments are the pelvic reconstructive procedures[19].

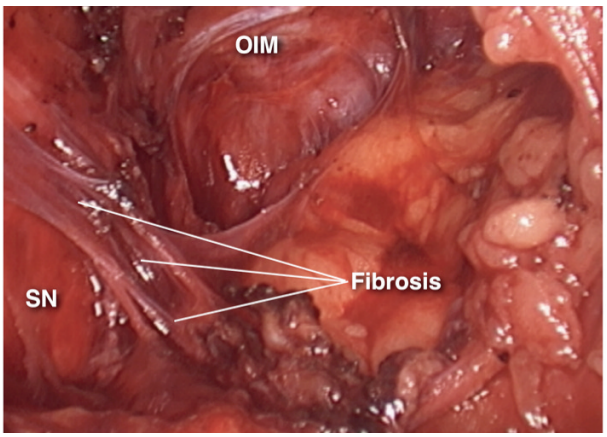

Figure 9. – Fibrotic entrapment of the left sciatic nerve

Vascular Entrapment

Pelvic congestion syndrome is a well-known cause of cyclic pelvic pain. Patients commonly present with pelvic pain without evidence of inflammatory disease. The pain is worse during the premenstrual period and pregnancy, and is exacerbated by fatigue and standing[20].

However, what is much less known is the fact that dilated or malformed branches of the internal or external iliac vessels can entrap the nerves of the sacral plexus against the pelvic sidewalls, producing symptoms such as sciatica, or refractory urinary and anorectal dysfunction[2,21] (Figure 10).

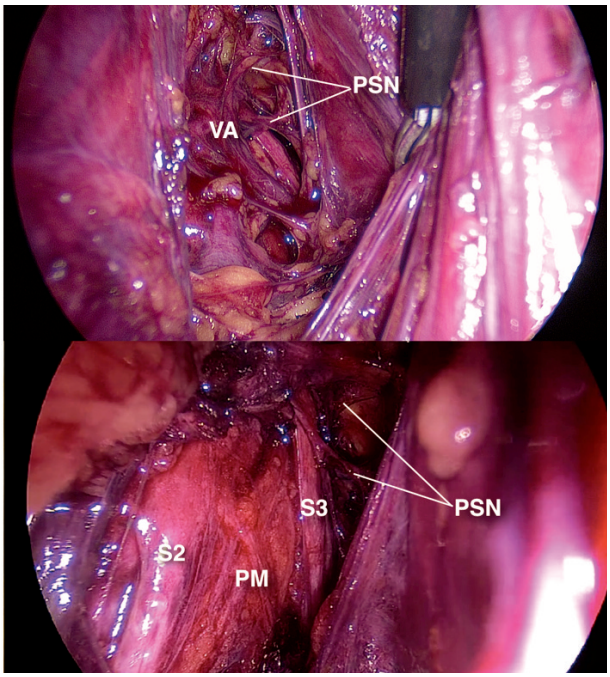

Figure 10. – Varicose tributary (VA) of the left internal iliac vein entrapping the S2 and S3 nerve roots against the left piriformis muscle (PM)

Piriformis Syndrome

Numerous malformations of the piriformis muscle have been described in the deep gluteal space that can entrap branches of the sciatic nerve. The laparoscopic approach has revealed that the intrapelvic fibers of this muscle can also entrap the sacral nerve roots[22]. Usually, these fibers originate from the sacral bone, laterally to the sacral foramina. However, part of the piriformis fibers may originate medially to the sacral foramina and the corresponding nerve roots in some individuals (Figure 11). Differentiating intrapelvic from extrapelvic piriformis syndrome can be very challenging. Bowel and urinary symptoms are a good indication that the entrapment is intrapelvic, but these are not always present.

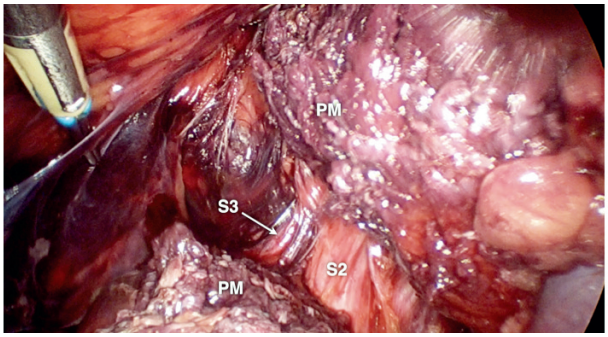

Figure 11. – Muscular entrapment of the right S2 and S3 nerve roots. Observe the transected piriformis muscle bundle (PM) originating from the sacral bone medially from the sacral nerve roots and, therefore, crushing the nerves every time the muscle contracts.

Neoplasms

Tumors can also entrap the nerves or nerve roots. Tumors can be primary neural tumors, such as Schwanomas, or

metastatic tumors, such as pelvic lymph nodes, entrapping the nerves in pelvic malignancies (Figure 12).

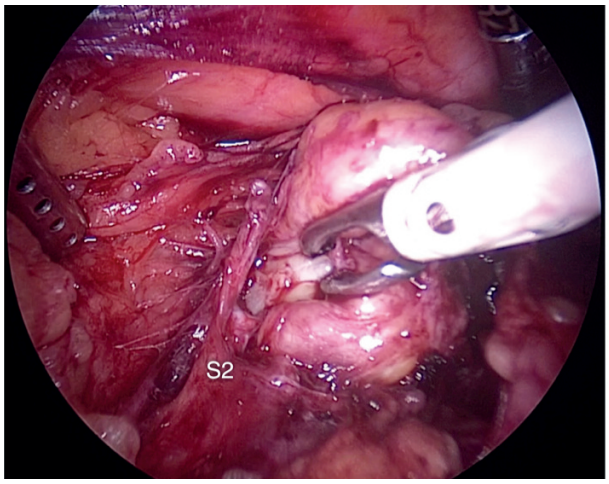

Figure 12. – Schwannoma in S2 (left)

Primary Neuropathic Pain, Nerve Transection and Secondary Neuropathic Pain

All the previously described causes of intrapelvic neuropathies have extrinsic entrapment as the etiology of pain. Intrapelvic radiculopathies can also result from nerve transections and or degenerations or intrinsic dysfunctions of the nerves themselves.

Nerve transections can occur during surgery or trauma and can induce neuroma formation, resulting in phantom pain and anesthesia of the affected nerve dermatome. An example of this is the phantom pain secondary to amputations, where branches of the sciatic and femoral nerves are transected. In the same fashion, pudendal transection will induce perineal pain and perineal anesthesia, as well as unilateral atrophy of perineal muscles, frequently resulting in urinary and fecal incontinence.

In entrapment syndromes, chronic ischemia induces cytoarchitectural changes to the neuron, which do not heal properly after the detrapment, resulting in neuropathic pain. The later the detrapment is performed, the higher the risk of neuropathic pain[23].

Neuropathic pain can also result from metabolic disturbances of the neuron, infectious agents, chronic exposure to neurotoxic substances, or a myriad of other causes.

In cases where there is no suspicion of entrapment as the primary cause of symptoms, extensive neurological investigation must be performed, preferably by a neurologist trained in assessing peripheral nerve pain. The symptoms must be clinically treated by an interprofessional pain team composed of a pain physician (usually an anesthesiologist or neurologist), a physiotherapy team (pelvic and motor), and a mental healthcare team (psychologist and psychiatrist). The pain specialist will prescribe and adjust the pharmacological treatment and, in cases where poor response to medical treatment is observed, perform the appropriate intervention (e.g. anesthetic blocks, pulsed radiofrequency).

Etiology of intrapelvic entrapments

As a rule, once a nerve entrapment has been diagnosed, decompression (usually surgical) is mandatory, since chronic ischemia can lead to endoneurial degeneration[23]. Therefore, the longer the time between the beginning of symptoms and detrapment, the lower the chance of success.

Surgical decompression will lead to complete resolution of pain and other symptoms in about 30% of the patients; around 50% will experience more than 50% reduction in pain and about 20% will not improve or, in some cases, experience worsening of their pain. Approximately 25% of patients will present with post-decompression neuropathic pain and 17% will present neuropathic strength loss, both of which tend to be transient; the former will last, on average, 5.5 months and the latter will last 2.5 months[24].

Patients who present with transient post-decompression pain, persistent post-neuropathic pain or worsening of symptoms, should be treated like patients with primary neuropathic pain, as described in the following session.

Pharmacological Treatment

There are no specific recommendations for the treatment of neuropathic pain of intra-pelvic origin. Management of this group of patient will follow the recommendation of neuropathic pain in general. Antidepressants, anticonvulsants, local anesthetics, N-methyl-D-aspartate (NMDA) antagonists, opioids, cannabinoids, botulinum toxin, capsaicin, and others may be used[25-27]. Most of these drugs were originally developed for other indications (e.g. depression and epilepsy), and their effectiveness for controlling neuropathic pain was later verified. The following tables outline commonly used drugs used for neuropathic pain control:

TABLES

Anticonvulsants	
Carbamazepine	400 to 1600 mg / day
Oxcarbazepine	600 to 1200 mg / day
Diphenylhydantoin	300 to 400 mg / day
Valproate Sodium	500 to 1500 mg / day
Lamotrigine	50 to 400 mg / day
Topiramate	50 to 200 mg / day
Gabapentin	900 at 2400 mg / day
Pregabalin	150 to 300 mg / day
Amitriptyline	50 to 150 mg / day
Nortriptyline	50 to 150 mg / day
Maprotiline	50 to 150 mg / day
Duloxetine	60 mg / day
Antidepressants Neuroleptics	
Fluphenazine	2 to 20 mg / day
Levomepromazine	25 to 500 mg / day
Chlorpromazine	50 at 600 mg / day
Antiarrhythmics	
Lidocaine	5 mg/kg/h/6h
Mexiletine	600 mg / day
Central Acting Muscle Relaxants	
Baclofen	10 to 30 mg / day
Opioids	
Tramadol	100 to 300 mg / day
Oxycodone	20 to 60 mg / day
Morphine Sulfate	20 to 90 mg / day
Methadone	150 to 400 mg / day
Transdermal Fentanyl	Up to 75 mg / day
Local anesthetics	
Capsaicin	
Anti-inflammatories	

Physiotherapy

In pelvic dysfunction resulting from nerve compression, the main goals of physical therapy are to reduce pain, train the pelvic floor muscles, and provide education about dysfunction and lifestyle interventions. This includes teaching awareness of the pelvic muscle group, the correct way to contract the pelvic muscles, coordination, motor control, strength, endurance, and relaxation of the musculature[28,29,30].

In order to reduce the patient's pain after surgical nerve decompression, cryotherapy has proven to be an effective therapeutic resource when applied to the vaginal canal. It is recommended to fill a non-sterile glove finger (or a condom) with ice and insert it into the patient's vagina for less than 20 minutes.

Electrical stimulation is also an important resource in the treatment of pain. It stimulates the rapidly conducting myelinated gross nerve fibers, triggering at the central level the descending inhibitory analgesic systems on the nociceptive transmission conducted by the non-myelinated fibers of small caliber, thus generating pain reduction[31,32].

Manual therapy techniques for myofascial release should be applied when there are signs of muscular tension of the pelvic floor, with the presence of trigger points, due to pain caused by nerve compression. The technique involves firm massage on the levator anus muscle with sliding movements towards the origin and insertion, punctual pressure at the trigger points at the limit of the patient's pain, in addition to perpendicular movements to the muscle fiber[33].

The techniques described for strengthening and awareness of pelvic floor musculature include biofeedback, and electrostimulation. These represent an important form of prevention and treatment for pelvic floor dysfunction.

Biofeedback is one of the most used resources for urogynecological physiotherapy, since it has no side effects. This technique allows the objective awareness of the physiological function that is unconscious in the individual, facilitating the correct learning of the pelvic floor muscle contraction. It can also be used for training and hypertrophy of the muscles. In addition, biofeedback assists in patient motivation during treatment, improving adherence to the physiotherapy program[34,35].

Electrical stimulation, when applied in the vaginal canal acts passively, and has an important effect on the proprioceptive awakening along with stimulating the correct learning of the perineal contraction. In addition, it has shown effective therapeutic results in patients with pelvic floor dysfunction, contributing to training of strength and muscular endurance, increasing the number of activated motor units and generating hypertrophy of the fibers. These benefits promote a strong and rapid contraction of the muscles, increasing urethral pressure and preventing urine loss during an abrupt increase in intra-abdominal pressure[36].

Interventional Treatments

Interventional procedures are an important option for the treatment of pelvic and perineal neuropathic pain. This is true especially for patients in whom conservative treatment did not bring the expected relief from pain, or for those whom the adverse effects of medications are intolerable.

The percutaneous blockade of specific nerves serves both diagnostic and therapeutic roles. In addition to the local anesthetic, it is quite common to add depot steroid for the anti-inflammatory and membrane-stabilizing effect. Imaged guidance with ultrasound[37,38], computed tomography, or fluoroscopy[39] enhanced the accuracy, reduce the volume of injectate and potentially minimize the complication rates.

If the pain relief is temporary, it is possible to apply more lasting techniques, such as radiofrequency, cryoablation, or neurolysis by chemical agents, such as phenol.

In the case of neuralgia caused by nervous incarceration by a muscle, there is the possibility of infiltration of this muscle with local anesthetic at first, followed by specific physiotherapy[40,41]. If this muscle contracts again, resulting again in nervous compression, it is possible to inject botulinum toxin, for a more prolonged relaxation. These techniques are best described in the myofascial pain chapter.

Pulsed Radio Frequency (RFP) is an alternative technique to conventional radiofrequency, and its advantage would be a longer pain relief without neural damage. During RFP application, a high frequency, pulsed current is generated and this allows the heat generated in the tissue to dissipate during the latency periods, not exceeding 45°C, which would be a neurodestructive temperature[42]. Thus, by maintaining the temperature only up to 42°C, there is no neural destruction, and, therefore, can be applied even in mixed nerves (i.e. both sensory and motor). The mechanism of action of the RFP is related to the electric field formed, which would alter painful signaling in a neuromodulatory form, but has not yet been fully elucidated[42,43]. The RFP can be applied distally to the nerve responsible for the patient's pain, or proximal, at its exit in the intervertebral foramen.

The Dorsal Root Ganglia (DRG) block corresponding to the nerve responsible for the pain can be performed with local anesthetic, guided by fluoroscopy. If the blockage alleviates at least 50% of the pain, it is possible to apply RFP thereafter[38].

Phenol Neurolysis has been described in several targets, especially to treat cancer pain, but also for non-cancer pain, and may bring prolonged pain relief. Care must be taken not to inject near motor nerves, because of the risk of flaccid paralysis. Chemical neuritis is another possible complication, although uncommon[44].

Cryoablation is a technique that promotes prolonged analgesia. The application of tissue cold blocks nerve conduction is similar to the local anesthetic. Long-term analgesia is due to freezing, which damages the nerve structure and causes Wallerian degeneration. However, since the myelin sheath and endoneurium remain intact, the nerve can regenerate after a period of time. One of its advantages over other neurolysis techniques, such as phenol for example, is the absence of post-procedure neuritis[39].

The main complications described with these procedures are similar to those experienced with any injection, including hematoma, infection and nerve damage.

Neuromodulation

In cases where medical and intervention pain treatment has failed or in cases where, although the topography of the lesion is determined, its etiology cannot be identified intraoperatively, the laparoscopic implantation of neuromodulation electrodes can be used to specifically modulate the af-

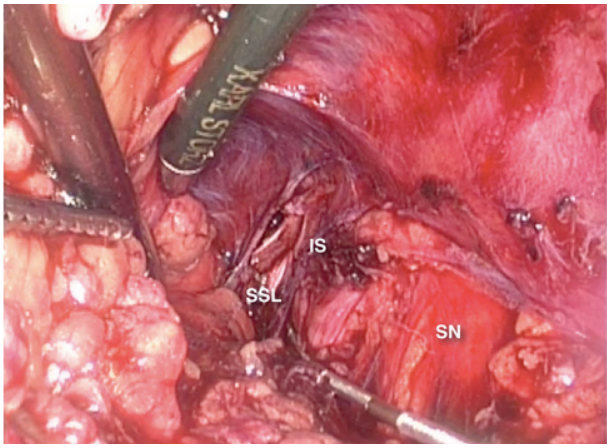

Figure 13. – LION Electrode placed on right sciatic and pudendal nerves (PM – Psoas Muscle; IS – Ischial Spine; SN – Sciatic Nerve; SSL – Sacrospinous Ligament)

fected nerve, producing very encouraging results when compared to the more commonly available epidural neuromodulation[5,45].

The laparoscopic implantation of neuroprosthesis – the LION procedure – was first reported by Possover in 2009 as a rescue procedure in patients with local complications of a Brindley procedure[45]. Due to its successful results and decreased invasiveness, it was then used as a primary procedure in spinal cord-injured patients, aiming to improve locomotion and bladder function[46]. Long term data has shown improvement in voluntary motor function and sensitivity, suggesting positive effects on neuroplasticity[47] (Figure 13).

CONCLUSION

Laparoscopy provides minimally invasive access with optimal visualization to virtually all abdominal portions of the lumbosacral plexus, which are also subject to entrapment neuropathies. Therefore. when facing sciatica, gluteal or perineal pain without any obvious spinal or deep gluteal causes, the examiner should always remember that the entrapment could be in the intrapelvic portions, especially when urinary or anorectal symptoms are present.

The laparoscopic approach to the intrapelvic bundles of the lumbosacral nerves opened a myriad of possibilities to assess and treat this neglected portion of the plexus, by means of nerve decompression or selective neuromodulation.

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INSTEAD OF:
All patients signed informed consent and the principles of the Helsinki Declaration were followed.

CORRIGENDUM:
ETHICS. This was a prospective case study audit. Prior to undertaking this study, each unit obtained EC approval for use of the TFS instrument in prolapse and incontinence surgery as standard hospital practice. All patients signed informed consent and the principles of the Helsinki Declaration were followed.