Diagnosis and Recent Management of Meralgia Paresthetica

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Abstract

Meralgia Paresthetica (MP) is a condition when the Lateral Femoral Cutaneous Nerve (LFCN) is compressed by the surrounding tissue. MP causes pain, paresthesia, and sensory loss according to the distribution of LFCN. This condition can cause the patient's quality of life to be disturbed and lead to disability, and financial loss if they do not get proper and adequate treatment. MP causes anything, for example, depression, and anxiety, including individual, social, and global burdens. Proper diagnosis and therapy are important for doctors to know. Knowledge of how to diagnose MP will lead to appropriate MP management so that it can reduce the existing burden. This article will discuss comprehensively the diagnosis and treatment of MP based on the latest references about MP both from journals, research results, and guidelines.

Keywords: Lateral Femoral Cutaneous Nerve (LFCN); Meralgia Paresthetica (MP); pain; diagnosis; management.

1. Introduction

Meralgia paresthetica (MP) is a mononeuropathy pain in the Lateral Femoral Cutaneous Nerve (LFCN). The clinical symptoms appear as pain, paraesthesia, and unpleasant numbness of the anterolateral thigh. This term was first coined by Bernhardt in 1878. The symptoms are pretty complex, it is preceded by pain, tingling, paresthesia, and numbness of the anterolateral thigh that is not related to surgical procedures [1]. Roth is credited with using the term "Meralgia paresthetica", which comes from the Greek words "meros" and "algos", meaning thighs and pain. Hager was the first person that correlates MP with LFCN compression [2].

The incidence of MP is observed in a population study in the Netherlands. Using a computerized network, there were 173,375 patients diagnosed with MP from the year 1990 to 1998 in Rotterdam general hospital. The incidence rate is 0.43 per 10,000 people [1]. Until now there has been no consensus on whether there is gender dominance [2]. However, one study evaluating 150 cases of MP found a higher prevalence in men [3]. One study evaluated a family with MP across four generations, demonstrating the inheritance of an autosomal dominant trait[4]. MP usually shows a greater incidence rate in obese, diabetic, and pregnant women. Several cases have also been reported in children with thin postures [5].

MP is diagnosed through a series of history taking, physical examination, and supporting examinations. Therapy at MP includes non-pharmacological and pharmacological therapy. Knowledge to establish a diagnosis and therapy in treating MP patients is important for doctors, especially neurologists. This article will discuss the diagnosis starting from history taking, physical examination, supporting examinations, and both non-pharmacological and pharmacological therapies in cases of MP.

2. Anatomy And Pathophysiology

Knowledge of the various forms of anatomy and etiological factors of MP is important to be known by doctors. LFCN is a sensory nerve that is originated from the second and third lumbar nerve roots [6]. It arises from the lateral border of the psoas major muscle, then travels across the iliacus muscle to the ASIS (anterior superior iliac spine). The nerve travels under the inguinal ligament medially near the ASIS. When entering the anterior compartment of the thigh region, this nerve travels laterally and caudally before it bifurcates into anterior and posterior branches. The diameter of LFCN at the level of the inguinal ligament is 3.2 + 0.7 mm [7]. Generally, the LFCN passes 1 cm medial to the ASIS at the level of the inguinal ligament, although there is considerable anatomical variation. It is crucial to understand the anatomy of the general shapes and variations of the LFCN pathway that can induce constriction and increase susceptibility to injury.

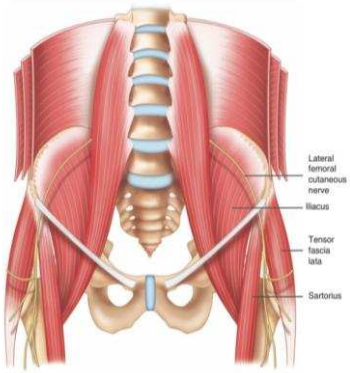

Figure 1. Schematic diagram that shows the LFCN innervation. Notice that the nerve travels under the inguinal ligament and superficial to the sartorius muscle and then between the sartorius and tensor fascia lata muscle. (Usra via www.usra.ca)[8].

Assmann et al 2011, described the frequency of five LFCN variant locations within 52 cadavers [9] (Fig. 2). In type A, the nerve surrounds the iliac crest (which occurred in 4% of cadavers in that study). Meanwhile, the LFCN is covered by the inguinal ligament in type B (27%). For type C, the nerve is enclosed by tendons of the sartorius muscle (23%). For the type D variant, the nerve is near the inguinal ligament and medial to the sartorius muscle (26%). In type E, the LFCN is medially above the iliopsoas muscles (20%). As

a note, 34 (65%) of the cadavers had asymmetrical neural distribution.

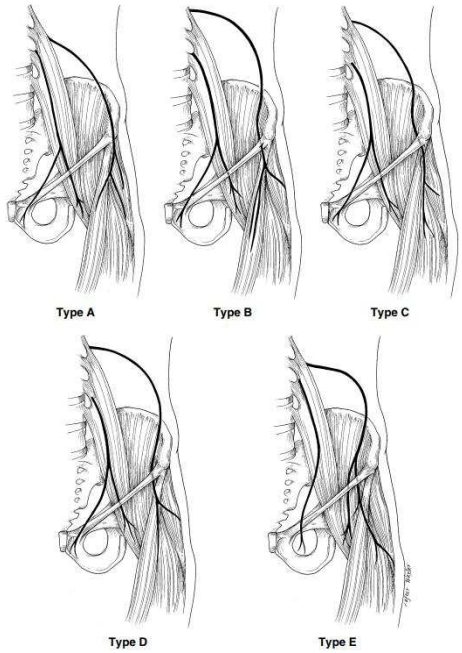

Figure 2. Five locations of the common variant of LFCN on exit from the abdomen (Aszmann OC et al., 1999)[5].

The passage of the LFCN varies widely, especially in where it approaches and exits the inguinal ligament. The variation is summarized below.

1. The LFCN mostly appears as a single branch that travels distally to the inguinal ligament [7]. However, in 28% of cases, the nerve bifurcates before passing through the inguinal ligament (around 0-5 nerve branches)
2. The mean distance of LFCN is approximately 29mm medial to ASIS according to a recent study, however, it can vary between 6 to 73mm [10].
3. Although the passage of nerves is medial to ASIS in the majority of cases, they can also pass through or even be posterior to ASIS in 4% - 29% of cadavers [3].
4. The LFCN generally penetrates the thigh beneath the fascia lata and superficially to the sartorius. However, in 22% of cases, it passes through the sartorius muscle itself. The LFCN intersects the lateral border of the sartorius muscle as far as 22-113 mm inferior to the ASIS [11].
5. LFCN can be located below, past, or above the inguinal ligament [12].

3. Etiology

MP is a peripheral nerve disorder that can be categorized and frequently correlated to entrapment neuropathy with various differential diagnoses, some of them are general and applicable to most peripheral nerve disorders, while others highlight specifically LFCN dysfunction. The etiology of MP consists of mechanical factors for instance pregnancy, obesity, and other conditions with increased intra-abdominal pressure and spinal surgery.

The compression effect of uterine fibroids[7] or tumors on the iliac crest[13] has been linked to the etiology of MP. The risk of MP is higher in pregnant and obese people or people with increased intra-abdominal pressure and abdominal protrusion [13]. This might be due to the adjacent location of the LFCN and the iliac fascia, and thus, the protrusion of the abdominal wall anterior part will cause traction of the LFCN and iliac fascia. Patients that wear belts, corsets, and tight trousers with a low waist such as the hip-hugger can also cause direct pressure on the nerves [14]. This becomes significant when the LFCN travels sideways over and besides the inguinal ligament [9]. Meralgia paresthetica is also correlated to several non-orthopedic surgeries such as laparoscopic for cholecystectomy, myomectomy and herniation surgery, aortic valve surgery, bariatric surgery for obese cesarean patients, and coronary bypass grafting [5,7].

The etiological factors of MP can be classified as metabolic, iatrogenic, and mechanical factors (Table 1) [9]. However, in a majority of MP patients, the exact etiology is still unknown.

Table 1. Etiology of Meralgia paresthetica [9]

Metabolic Factor
Diabetes mellitus
Alcoholism
Lead poisoning
Mechanical Factor
Braces/ corsets
Pelvic tumors (e.g. fibroids or iliac bone tumors)
Trauma (e.g. pelvic crush injury)
Increased abdominal pressure (such as in obesity, pregnancy, and ascites)
Low cut trousers
Iatrogenic Factor: Orthopedic Surgery
Pelvic fixation or osteotomy
Hip arthroplasty or fracture reduction/fixation
Spinal surgery
Iliac crest bone graft
Iatrogenic Causes: Non-Orthopedic Surgery
Bariatric surgery
Cesarean delivery
Laparoscopic surgery (e.g. myomectomy, cholecystectomy, hernia repair)
Aortic Valve surgery
Coronary bypass transplant

4. Diagnosis

4.1 History Taking And Physical Examination

From the history taking, the patient can describe the symptoms as a thick feeling, tingling, pain, burning sensation, and decreased sensitivity to pain sensation, touch, and temperature according to the LFCN dermatome. Hypersensitivity to tactile stimulation and dysesthesia also occasionally occurs. Physical examination usually indicates a tenderness of the lateral inguinal ligament which is the location where the nerve crosses the inguinal ligament. In this condition, exacerbations often occur when the extension of thigh joint during walking. Symptoms of MP include uncomfortable paresthesia of the upper and lateral thighs. In 80% of the patients, the symptoms appear unilaterally, however, the rest of the patients had bilateral complaints [3,7,9].

Symptoms decrease with sitting, but sometimes symptoms are reported to be exacerbated by sitting. Other gastrointestinal, neurological, and urogenital clinical signs that do not match the MP characteristics indicate that the leg pain is caused by another condition. The patient may also complain of hair loss on the anterior thigh as a result of continuous scratching of the area by the patient, and this is an important diagnostic marker [15].

MP has also been associated with lumbar radiculopathy. Although the term MP refers only to dysesthetic pain in LFCN neuropathy, pathologically it may only cause hypoesthesia in the distal distribution without any pain. In rare cases, pain associated with nerve trapping can radiate proximally to the spine, such as in carpal tunnel syndrome, which can make it more difficult to make the diagnosis. The diagnosis of MP is generally made based on a coherent history taking and physical examination relevant to the anatomical position. However, there must be a clinically suitable differential diagnosis when evaluating the patient. Metastases of the iliac crest [3,16] and lumbar disc herniation, sometimes might have symptoms like MP, so it should be ruled out with MP. The symptoms of avulsion fractures of the anterior superior iliac spine and chronic appendicitis might appear similar to MP. If patients came with motor or sensory deficits or reflex changes that are not specific to LFCN, further evaluation should be performed [5,15,17].

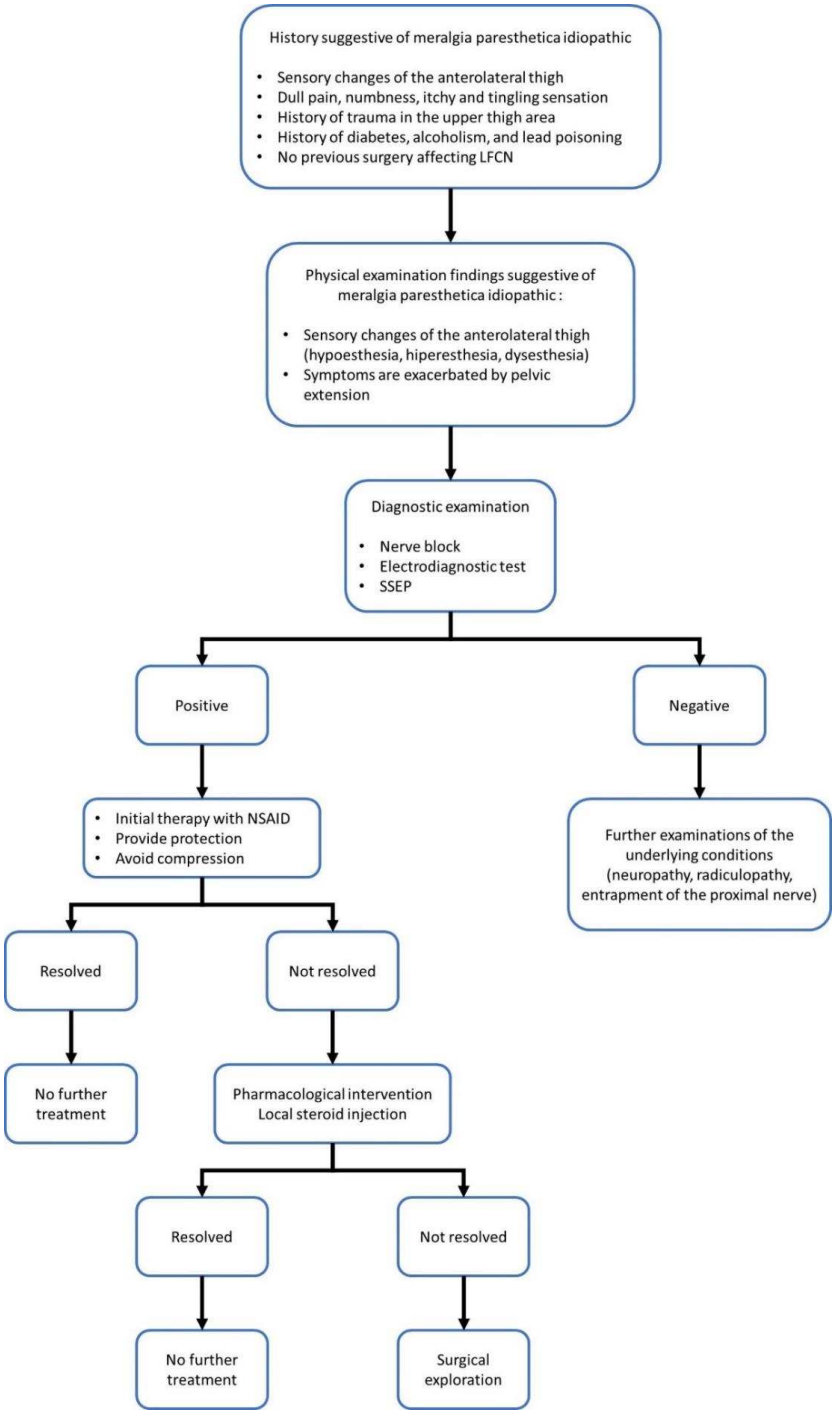

Figure 3. The algorithm for evaluation and treatment of Meralgia Paresthetica[10].

When evaluating patients with a suspected diagnosis of MP, clinicians are expected to find other possible etiologies and important clinical features (Table 1). Other factors such as mechanical or metabolic can be overcome by lifestyle modification such as avoiding wearing tight clothes or routine laboratory tests for instance blood glucose, HbA1c, and lead testing. LFCN is a sensory nerve, therefore if there is a symptom of dermatomal sensory disturbances along with motor and sphincter dysfunction, disorders in the vertebrae should be considered as an etiology. In that case, a radiologic examination of the spine (magnetic resonance imaging) should be performed. Symptoms such as changes in appetite, weight loss, as well as acute severe pain on pressure are red flags that indicate avulsion fracture of ASIS or metastasis to the iliac crest [9,18].

4.2 The Supporting Investigation

The diagnosis of MP is primarily based on clinical symptoms. In situations that are still uncertain, both diagnostic nerve block and electrophysiological tests can be helpful to establish the diagnosis. A CT scan of the lumbar and x-ray of the pelvis should be done to rule out the possibility of disc herniation or tumor. Magnetic Resonance Imaging (MRI) and ultrasound can be performed to analyze the retro-peritoneal areas. From the physical exam, there was no tenderness on the sciatic notch and a negative on the Laseque test. A complete hematologic examination should be carried out, including the function of the thyroid, because MP can be correlated with hypothyroidism [11,19].

An electrodiagnostic test can be performed if there is still uncertainty after anamnesis and physical examination. Routine electrophysiological tests to verify the diagnosis of LFCN neuropathy include sensory nerve conduction velocity (SNCV) with a sensitivity of 81.3% and somatosensory-evoked potentials (SSEP) that is placed on the scalp after LFCN stimulation that has a sensitivity of 65.2% [10,20]. Electrophysiological testing and nerve blocks have an important role when the diagnosis is still unconfirmed. However, the score of SSEP in establishing the diagnosis of MP is still debatable. A study where the evaluation of SSEP in 20 volunteers compared to 22 patients, concluded that SSEP is helpful as a diagnostic tool. A different study also showed that SSEP was useful to determine if the MP was a result of an injury in the proximal area of ASIS. In contrast, research conducted by Seror concluded that only a very severe neurological injury will be detected as abnormal in SSEP, therefore it is not recommended as a routine diagnosis of MP. Measuring SSEP after stimulation is only beneficial for patients where the SNCV cannot be measured, such as people with obesity [3,9,21]. Another examination that can be conducted to support the diagnosis is the Nerve Block Test at the exit location of LFCN, which is around the pelvic area in the inguinal ligament. This nerve block test is performed by injecting 1% lidocaine around 1cm medial and inferior to ASIS or at the location of the worst pain. The examination result is said to be positive if the patient experiences a significant reduction in pain

within 30-40 minutes post-injection [10].

4.3 Diagnostic Injection with Local Anesthesia and Steroids

Even though the use of LFCN blocks cannot yet be compared with other tests, a diagnostic injection might be useful to confirm the diagnosis after the initial assessment. The role of diagnostic injection will be discussed further in the section on management. Neurologists are generally required in the management of MP when performing diagnostic and therapeutic blocks, details regarding the technique are described here. Generally, LFCN block is conducted using marker-based or imaging-guided techniques. Marker-based techniques can be assisted by nerve stimulators and guided imaging techniques are mainly referred to as the use of ultrasound.

5. Treatment

MP management focuses on addressing the underlying causes (if any) and conservative treatment.

5.1 Conservative Measures

Most patients respond well to conservative treatment. This mainly includes lifestyle modification, pharmacological therapy, and injection. Lifestyle modifications include losing weight and avoiding wearing tight trousers. Obesity can increase the risk of MP two times higher, possibly because of the increased intra-abdominal pressure. The use of tights with a waistline that is at hip level can cause MP, especially in people who are thin and have an anomaly in LFCN. Some studies showed a result of MP patients responding well to the conservative management (complaints reduced by 85% in 4 to 6 months) [4,12,14].

5.2 Pharmacological Therapy

5.2.1 Interventional Pain Management

Neuroscientists are involved in the pain management of MP and LFCN blocks. LFCN blockade using local anesthetics and steroids serves both diagnostic and therapeutic roles. on-steroidal anti-inflammatory drugs (NSAIDs) are mostly used as first-line pharmacological therapy. Starting with injecting a local anesthetic such as Bupivacaine 0.25% and followed by injection of methylprednisolone with a dose range of 20-120 mg per day for the next few days. The use of tricyclic anti-depressants and gabapentin for neuropathic pain is very well known. LFCN blockade with anesthetics that are given locally and steroids is normally administered to patients that are included in multi-modal therapy with pharmacological treatment and lifestyle modification [4,6,14]. Although the ideal frequency and dose have not yet been measured, this formula can be repeated 3 to 5 times if needed. Various researches have shown outstanding results from LFCN blocks. Symptoms can be reduced by 66% to 91%. Apart from anti-inflammatory function, steroids also have a membrane-stabilizing effect by suppressing transmission of unmyelinated C fibers and inhibiting ectopic release from experimentally formed neuromas. Partial loss of function in small fiber neurons has been

associated as the cause of pain symptoms in MP patients, especially in those who suffer the disease longer [5,9,14].

Pulsed radiofrequency nerve lesioning for the treatment of Intractable MP has been studied, however large-scale studies are needed to confirm its effectiveness. Surgical intervention might be needed in cases where the symptoms persist after conservative treatments [5,9,14]. Marker-Based Techniques, The conventional way merely depends on anatomical markers. The needle is placed 2.5 cm medial to the ASIS caudally to the inguinal ligament. The exit point is marked by a “loss of resistance” or a “pop” sensation in the fascial lining or by infiltration in a fan-wise fashion [8,22].

The accuracy of marker-based methods varies, but a study shows a low success rate of around 40%. The low success rate of these blocks might be caused by the wide anatomical variations of the LFCN course, as well as the lack of foreseeable association between LFCN and palpable bone markers or vascular structures [23]. Research that correlates needle placement using traditional marking techniques in both cadaveric dissection and nerve localization with transdermal stimulation in volunteers did not show a good correlation (with only 5% and 0% respectively). Additionally, in 35% of subjects that received LFCN block, improper LFCN localization led to accidental femoral nerve block. Using a nerve stimulator that can induce paresthesia of the anterolateral part of the thigh, can increase the success rate as high as 85% although it may cause discomfort to the patients [9,19].

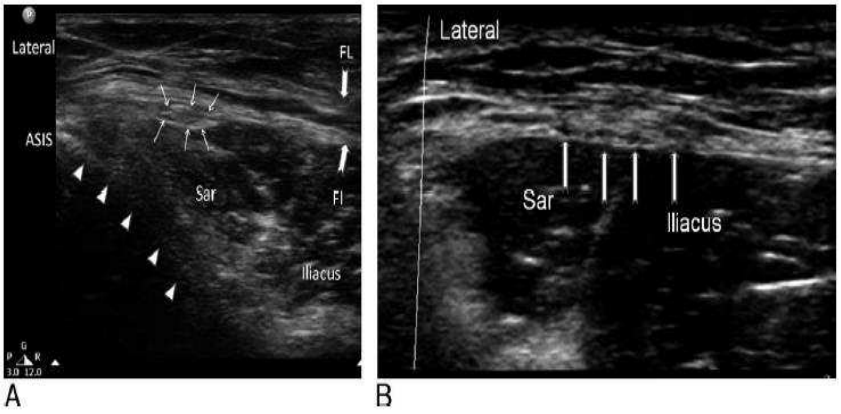

Figure 4. Ultrasonogram shows the lateral femoral cutaneous nerve (LFCN) (www.usra.ca). An LFCN is marked with a line arrow. The thick arrow marks the fascia (FL stands for fascia lata, and FI stands for iliac fascia). The solid arrow shows the ilium (Sar stands for sartorius muscle) B, LFCN has bifurcated into smaller nerves and is shown as a hypoechoic structure (solid arrow line)[9].

5.2.2 Ultrasound-Guided Injection Technique

Ultrasound-guided LFCN block injection is very well known. One study showed that the

identification of LFCN by ultrasound in both volunteers and cadavers had great accuracy. From cadavers, we obtain the accuracy of needle-LFCN was 84% in ultrasound guidance compared to 5% for the marker-based technique [8]. In addition, the success rates of needle-LFCN contact with ultrasound guidance in volunteers were as impressive as nerve stimulators: 80% incorrect identification of LFCN sites under the skin [13].

In another case series, 10 patients with an average body mass index of 31 kg / m^2 were observed. It is reported that ultrasound-guided LFCN was visualized and the sensory block was successful in all patients. This technique did not exacerbate nearby nerve blockade and no patient complained of paresthesia due to direct needle contact with LFCN [21].

While the patient is lying supine, we marked the area of the inguinal ligament and ASIS. ASIS is visualized as a hyperechoic structure with a posterior acoustic image using high frequency (6-13 MHz) linear array transducers. The ultrasonic probe is put on top of the ASIS across the long axis of the inguinal ligament and then moved distally. An inverted triangular structure describes the sartorius muscle. The focus is on the direction of the probe relative to the neural passage. The LFCN will be shown in the short-axis as one or more hyper or hypoechoic structures (Figure 3). If the nerve cannot be identified, search the LFCN in the area between the sartorius and tensor fascia lata. After the LFCN is identified, the needle is inserted into the field with an ultrasound probe (Figure 4). Alternatively, the needle can be removed from the field using a nerve stimulation needle to confirm needle placement [8,9,24].

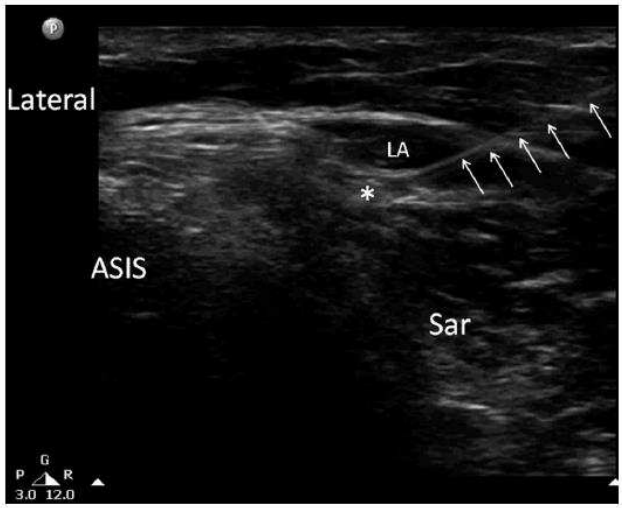

Figure 5. Post injection ultrasonogram; the needle is marked with a line arrow line. LA denotes local anesthesia. *LFCN. (www.usra.ca) [9].

In situations where the LFCN cannot be found using those techniques, 2 other techniques can be tried. The first method is by injecting 5% dextrose as a hydrodissection to the area between the fascia above

the iliacus muscle and fascia lata. Another method that can be used is to find the surrounding nerves percutaneously to a transdermal nerve stimulator.

5.3 Surgical Management

Surgery should be performed when all non-operative therapies fail to manage the condition effectively. Literature on surgical management for MP is rare. This could be because only 25% of neurosurgeons perform fewer than 10 of this procedure per year, and the rest 70% do not perform these procedures. Three kinds of surgical intervention have been explained earlier: neurolysis of the narrowed tissue near the nerve with or without LFCN transposition and transection with LFCN partial excision [15,18].

Ivins, 2017, in his study involved 14 adult patients with a 3-6 year follow-up, who were first treated using conservative management; however, almost half of the patients needed operative intervention. Out of four cases of decompression, three of them have an excellent initial result, such as rapid symptoms relief. However, 2 to 24 months later after follow-up, patients underwent relapse and need re-exploration with resection which results in long-term recovery. Furthermore, two other patients had similar results after being performed the primary resection. Van Eerten analyzed neurolysis and transection of 21 unsuccessful conservatively treated patients and discovered a result that transection (9 out of 11 patients with total symptoms improvement) was better than neurolysis (only 3 out of 10 patients). However, one thing that should be taken into consideration is that in this research the procedure is performed by different neurosurgeons [2,9,20,22].

According to research conducted by Ivins et al in 2017, The indication for operative intervention was classified into three groups based on the symptoms that persist after the conservative treatment :

1. Simple decompression should be performed on both adults having symptoms for less than 1 year and all pediatric patients.
2. Resection should be considered in patients from group 1 with persistent and recurrent symptoms.
3. Primary resection is considered in adults with persisting symptoms for more than 1 year.

Another fascinating case of a 40-year-old woman with MP after resection of a malignant right inguinal tumor who were performed new procedures such as neurolysis and the use of prophylactic methods of inferior epigastric perforator in adipose flap wrapping to prevent entrapment of the nerve [1,9,10,25].

6. Conclusion

MP is a syndrome characterized by tingling, numbness, and pain due to the trapping of LFCN. The diagnosis of MP is generally established based on clinical symptoms of paresthesia and pain in the anterolateral thigh. The clinician must understand the shape of the anatomical variation and the various

modifiable factors. Electrophysiological testing and nerve block are crucial when the diagnosis is still uncertain. Most patients respond well conservatively such as nerve blocks.

Recent ultrasound-guided injection technique has improved the accuracy of nerve blockade. Neurologists are needed in the management of MP especially in treating the pain and performing LFCN blocks. LFCN block using locally given anesthesia and steroids acts both as therapeutic and diagnostic roles. Surgery should be considered in patients that show refractory to these treatments.

Acknowledgements

None

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