Meralgia Paresthetica

What an Anesthesiologist Needs to Know

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Abstract: Meralgia paresthetica (MP) is an entrapment pain syndrome of the lateral femoral cutaneous nerve (LFCN) of thigh. Diagnosis is principally made on clinical ground with pain and paresthesia of the anterolateral thigh. Electrophysiological test and nerve block play important roles when the diagnosis is uncertain. Clinicians should be aware of the various etiological factors that can be potentially modified or treated. Most of the patients respond to conservative management including nerve block. Surgical options should be considered in patients refractory to those treatment options. Anesthesiologists are commonly involved in the management of MP because of their expertise in pain management and performance of the LFCN block. Blockade of the LFCN with local anesthetics and steroid serves both the diagnostic and therapeutic role.

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Meralgia paresthetica (MP) is a painful mononeuropathy of the lateral femoral cutaneous nerve (LFCN), presenting as unpleasant paresthesia, pain, and numbness in the anterolateral aspect of the thigh.[1,2] Anesthesiologists are commonly involved in the management of this group of patients because of their expertise in the interventional procedures and pain medicine. The objective of this article was to provide a narrative review of the history, epidemiology, etiology, anatomy, clinical presentation, and the management of this pain syndrome, with emphasis on the roles and techniques of blockade of the LFCN.

First described by Bernhardt[3] in 1878, the symptom complex initially comprised of pain, numbness, tingling, and paresthesia in the anterolateral thigh that was not associated with a surgical procedure. However, Roth was credited for using the term "meralgia paresthetica,"[4] which was derived from the Greek words "meros" and "algos," meaning thigh and pain. Hager[5] was the first to associate MP with compression of the LFCN.

EPIDEMIOLOGY

The incidence of the disease is approximately 4.3 per 10,000 person years based on a large case-control study in general practice setting. Spontaneous MP can occur in any age group but is most prevalent in the age group 41 to 60 years.[2] There was no consensus to sex predominance. A previous study noted a higher incidence in men.[6] However, a recent large cohort study suggested a higher female preponderance (5.0 versus 3.2 per 10,000 person years).[1] Familial trait of the disease has been noted in a family with MP in 4 generations, suggesting the possibility of autosomal dominant trait inheritance.[7] Pregnancy and carpel tunnel syndrome are 2 conditions associated with increased occurrence of MP.[8] The disease also affects pediatric population. In 1 case series, one third of the pediatric patients treated for pelvic osteoid osteoma developed MP.[9]

ETIOLOGY

Many etiological factors can account for the presence of MP and can be categorized as mechanical, metabolic, and iatrogenic factors (Table 1).[10–34] In most of the patients with MP, there is no attributable cause.

Compressive effect from uterine fibroid[10] or tumor in iliac crest[11] has been implicated as one of the causes of MP. The risk of MP is higher in obese and pregnant patients or those with conditions associated with an increase in intra-abdominal pressure and the protrusion of the abdomen.[12] This may be related to the close relationship between the LFCN and the iliac fascia, and thus, the protruding anterior abdominal wall will result in the traction of the iliac fascia and the LFCN.[35] Wearing of belts, corset, and tight low-waist trousers like hip huggers can also result in direct pressure on the nerve.[13,14] This is of particular significance when the LFCN runs an aberrant course above and lateral to the inguinal ligament.[38]

Meralgia paresthetica has been reported after a variety of orthopedic surgeries.[18–21] Because the nerve courses around the iliac bone near the anterior superior iliac spine (ASIS), the LFCN is at risk after surgery in the vicinity such as iliac crest bone grafting,[18] pelvic fixation, and pelvic osteotomy.[19] Meralgia paresthetica is a well-known complication after spine surgery. Mirovsky and Neuwirth[21] noted a 20% complication rate in patients undergoing spine procedures. The mechanisms for the LFCN injury include bone grafting from the ASIS, pressure on the nerve while on prone position, and retraction of the psoas muscle in retroperitoneal approaches (the LFCN travels lateral to the psoas muscle in pelvis).

Meralgia paresthetica has also been reported after a large variety of nonorthopedic surgeries such as laparoscopic cholescystectomy, laparoscopic myomectomy, coronary bypass grafting, aortic valve surgeries, bariatric surgery for the morbid obesity patient, cesarean delivery, and laparoscopic hernia repair.[22–34]

ANATOMY OF THE LATERAL FEMORAL CUTANEOUS NERVE

The LFCN is a pure sensory nerve arising from the second and third lumbar nerve roots.[35,36] Emerging from the lateral border of psoas major muscle, the LFCN runs across the iliacus muscle traveling toward the ASIS (Figs. 1 and 2). The nerve travels under the inguinal ligament medial and close to the ASIS. On entering the anterior compartment of the thigh region, this nerve runs in a lateral and caudad direction and divides into

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TABLE 1. Etiology of Meralgia Paresthetica

Mechanical causes
Pelvic tumor, eg, fibroid[10] or iliac bone tumor[11]
Increased abdominal pressure,[12] eg, pregnancy, obesity, ascites
Braces/corsets[13]
Low cut trousers[14]
Trauma: pelvic crush injury[15]
Metabolic causes
Lead poisoning[16]
Alcoholism[16]
Diabetes mellitus[17]
Iatrogenic cause: orthopedic surgeries
Iliac crest bone grafting[18]
Pelvic fixation or osteotomy[19]
Hip arthroplasty or fracture reduction/fixation[20]
Spine surgery[21]
Iatrogenic cause: nonorthopedic surgeries
Laparoscopic surgeries, eg, myomectomy,[22] hernia repair,[23–28] cholescystomy[29]
Coronary bypass grafting[30]
Aortic valve surgery[31]
Bariatric surgery[32,33]
Cesarean delivery[34]

anterior and posterior branches. The diameter of LFCN at the level of inguinal ligament is 3.2 ± 0.7 mm.[35]

The course of the LFCN, particularly for the course where it approaches and exits the inguinal ligament, is very variable and is summarized as below.

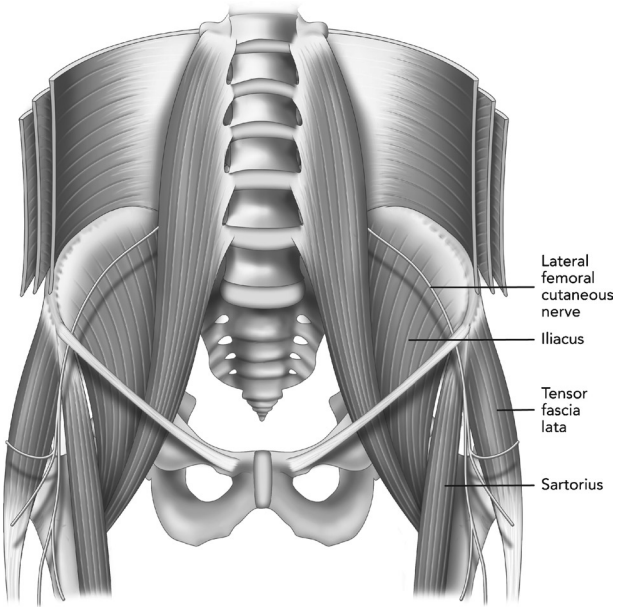

FIGURE 1. Schematic diagram showing the pathway of a typical course of LFCN. Note that the nerve courses beneath the inguinal ligament and runs superficially to the sartorius muscle and then in between this muscle and tensor fascia lata muscle. Reproduced with permission from Ultrasound for Regional Anesthesia (USRA; www.usra.ca).

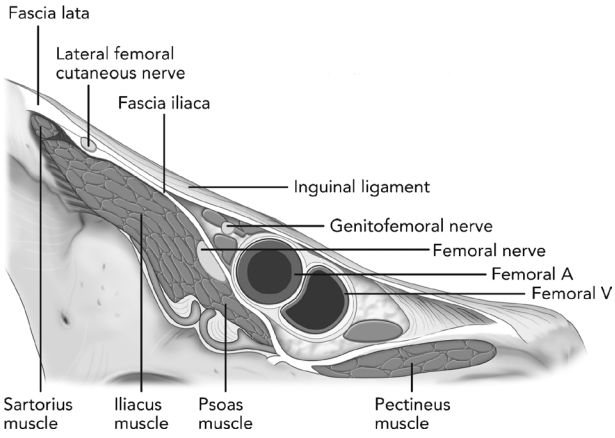

FIGURE 2. Nerves at the inguinal area. Reproduced with permission from USRA (wwww.usra.ca).

1. Although the LFCN mostly presents as a single branch just distal to the inguinal ligament,[37] it can divide before crossing the inguinal ligament in up to 28% of the cases (range, 0–5 nerve branches).[38]
2. Although a recent cadaveric and volunteer study of LFCN suggested the mean distance of the nerve to be approximately 29 mm medial to the ASIS,[39] this distance varies between 6 and 73 mm.[38]
3. While the nerve courses medial to the ASIS most of the time, it can pass over or even posterior to the ASIS in 4% to 29% of the cadavers.[34,40,41]
4. Although the LFCN commonly enters the thigh superficial to the sartorius muscle beneath the fascia lata, the LFCN passes through the muscle itself in 22% of cases.[35] The LFCN crosses the lateral border of the sartorius muscle in distance ranging from 22 to 113 mm inferior to the ASIS.[38]
5. The LFCN may run under, through or above the inguinal ligament.[35–37]

EVALUATION

Clinical Evaluation

The most common presentation is a burning and tingling sensation on the anterior and lateral aspects of the thigh as far as the knee.[42] Numbness is a late sign and is rarely the only presentation, the presence of which theoretically disqualifies the use of the term of "meralgia." Bilateral presentation is uncommon (20%).[43] Pain or unpleasant tingling sensation can be aggravated by standing and hip extension and relieved by sitting.[42–44] Physical examination may reveal tenderness over the lateral aspect of the inguinal ligament with Tinel sign elicited at the site of entrapment. Hypoesthesia over the area innervated by the LFCN is commonly found with or without allodynia. Clues suggestive of the etiology may be revealed during the examination, such as the surgical scars or raised intra-abdominal pressure (Table 1).

In addition, clinicians evaluating patient with potential diagnosis of MP should look for other underlying causes and the important negative clinical features (Table 1). The former include the mechanical or metabolic causes which can be modified by lifestyle (abstinence of tight outfit) or investigated with the appropriate laboratory tests (blood glucose, HbA_{1c}, and lead assay). Because the LFCN is a sensory nerve, the presence of dermatomal sensory loss and motor and sphincter dysfunctions should alert the clinicians to the spinal etiology.[45–47] In that case,

spine imaging (magnetic resonance imaging) should be arranged. The presence of red flags such as weight loss and appetite change and acute onset of severe pain on pressure may suggest metastasis at the iliac crest[11] or avulsion fracture of ASIS,[48] respectively.

The diagnosis of MP is mainly on clinical ground.[42] In the situation of uncertainty, both electrophysiological test and diagnostic nerve block can be useful.

Electrophysiological Tests

When the diagnosis is still uncertain after history and physical examination (eg, atypical sensory distribution of presentation), sensory nerve conduction test can be very useful.[49–51] The method involves either by stimulating the LFCN as it exits the pelvis near ASIS and recording potentials distally or by stimulating distally along the course of the nerve and recording proximally in the region of ASIS.[52] One limitation of the nerve conduction test is that it evaluates mainly large myelinated axons. Because neuropathic pain in the entrapment syndrome is often due to the disease of small myelinated Aδ and C fibers, nerve conduction test can be normal in patients whose condition principally affects the small fibers.[53,54] A recent study confirmed the contribution of the small fiber in pain in patients with MP.[55]

Another method of investigation is somatosensory evoked potential (SSEP) after thigh stimulation. However, the role of SSEP is not as well accepted as sensory nerve conduction test. Seror[51] found SSEP only useful in obese individuals or patients with very serious nerve damage, thus SSEP is not recommended as routine investigation tool.[56]

Diagnostic Injection With Local Anesthetics and Steroid

Although the use of the LFCN block has not been compared with other diagnostic tests, it can be a useful diagnostic tool to ascertain the diagnosis after initial evaluation.[16,42] The role of this in the management of MP will further be discussed in the management section. Because anesthesiologists are commonly involved in the management of this group of patients because of their expertise in the diagnostic and therapeutic blocks, the details of the techniques will be described here.

TECHNIQUES FOR LFCN BLOCK

In general, the LFCN block is performed with either landmark-based or imaging-guided techniques. The former can be assisted with nerve stimulator and the latter mainly refers to the use of ultrasound.

Landmark-Based Technique

The traditional approach is solely based on anatomic landmark. The needle is inserted at a point 2.5 cm medial to ASIS just caudal to the inguinal ligament. The end point is determined by a "loss of resistance" or "pop" sensation through the fascial layer[57–59] or by a fan-wise infiltration.[60,61]

The success of the landmark-based method is variable, but a well-designed study suggested the success rate was as low as 40%.[57] The low success rate of the block can be attributed to the wide anatomic variability of the course of the LFCN, as well as to the lack of any predictable relationship of the LFCN to palpable vascular structures or bony landmarks.[62] A study correlating the needle placement by the classic landmark technique with both the cadaver dissection and localization of the nerve with transdermal nerve stimulation in volunteers showed a very poor correlation (5% and 0% respectively).[63] In addition, imprecise localization of the LFCN can result in unintentional femoral nerve block, which occurred in 35% of subjects receiving LFCN block.[57] With the use of nerve stimulator eliciting paresthesia at the anterolateral thigh, the block success rate was raised to 85% but may be at the expense of increase patient discomfort.

Ultrasound Guided Technique

Literature Review

The technique of application of ultrasound to the LFCN blockade has been well published.[39,62–65] One of these was a validation study that demonstrated greater accuracy in identifying the LFCN with ultrasound in both cadavers and volunteers.[63] In cadavers, the success rate of needle contact with the LFCN was 84% under ultrasound guidance as opposed to 5% with landmark-based technique. In the same study, the success rate with ultrasound guidance was equally impressive in human volunteers with the LFCN identified by percutaneous nerve stimulator: 80% versus 0% in the correct identification of the location of the LFCN underneath the skin.

In another small case series of 10 patients with a mean body mass index of 31 kg/m^2, the authors reported that the LFCN could be visualized by ultrasound in all patients and that sensory block was successful in all cases.[62] The technique was not complicated by coincidental blockade of any nearby nerves nor did any patients complain of paresthesia from the needle coming into direct contact with the LFCN.

Perils in Sonoanatomy

The LFCN is a small peripheral nerve and the course of this is highly variable. To locate the nerve successfully with

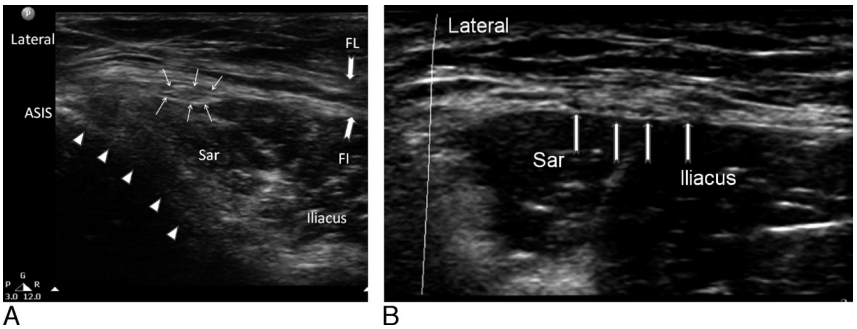

FIGURE 3. Ultrasonogram showing the lateral femoral cutaneous nerve (LFCN). Reproduced with permission from USRA (www.usra.ca). A, The LFCN is indicated by line arrows. The fascia is indicated by bold arrows (FI indicates fascia iliaca; FL, fascia lata); the ilium is indicated by solid arrows (ASIS indicates anterior superior iliac spine; Sar, sartorius muscle). B, The LFCN has already branched into smaller nerves and appears as hypoechoic structures (solid line arrows).

ultrasound scanning, one needs to be aware of a few important principles:

(1) A sound knowledge of anatomy of the course and orientation of the LFCN as well as the structures around the LFCN helps to appreciate the location of the nerve.[39]
(2) The nerve is better appreciated with dynamic scanning or sweeping view because of the size of the nerve and its proximity with the fascia layers.[39,62]
(3) The LFCN nerve may appear as hyperechoic, hypoechoic, or mixed structure, depending on the course of the nerve itself (under or through the inguinal ligament or over the iliac crest), the special tissue architecture in the corresponding area (surrounded by fatty tissue in the plane between sartorius and tensor fascia lata muscle), and the frequency of the transducer used (higher frequency probe likely produces artifacts).[39,62,63,65]
(4) The LFCN in a patient with severe or advanced symptoms of MP is likely to be swollen or enlarged (pseudoneuroma) and is likely to be picked up ultrasonography.[35]
(5) The common locations that the LFCN can be found are usually in the infrainguinal region, either superficial to the sartorius muscle or between sartorius and tensor of fascia lata muscles. In the latter case, it is a fat-filled space.

Ultrasound-Guided Injection Technique

With the patient in the supine position, the ASIS and the inguinal ligament are marked on the skin. Using a high-frequency linear array transducer (6–13 MHz), the ASIS is visualized as a hyperechoic structure with posterior acoustic shadowing. The ultrasound probe is placed over the ASIS initially with the long-axis view of inguinal ligament and is then moved distally. The sartorius muscle will be seen as an inverted triangular structure. Attention is paid to the orientation of the probe relative to the course of the nerve. The LFCN will appear as one or more hyperechoic or hypoechoic structures in the short-axis view (Fig. 3). If the nerve cannot be identified, the alternative is to look for the LFCN in the plane between the tensor fascia lata and the sartorius muscle. Once the LFCN has been identified, a needle is advanced in plane with the ultrasound probe (Fig. 4). Alternatively, the needle can be advanced out-of-plane using a nerve-stimulating needle to confirm placement.

In the situation when the LFCN cannot be identified by the above methods, 2 methods can be tried. One is to inject dextrose 5% solution to hydrodissect the plane between the fascia lata and the fascia over the iliacus muscle. The other is to locate the proximity of the nerve percutaneously with a transdermal nerve stimulator.[63]

MANAGEMENT OF MP

Conservative Treatment

Most of the patients will respond to the conservative therapy.[66,67] These include mainly lifestyle modification, pharmacological therapy, and injections.

Lifestyle modification includes weight reduction and avoidance of tight low-cut pants. Obesity doubles the risk of MP,[68] probably due to the increase in intra-abdominal pressure.[36] Tight-fitting low-cut pants with the waistline resting at hip level may cause MP,[14] especially in a thin person with an anomalous pathway for the LFCN.[2]

Nonsteroidal anti-inflammatory agents are usually used as the initial pharmacological treatment.[16,69] The use of tricyclic antidepressants and gabapentinoids for neuropathic pain has been well documented.[70]

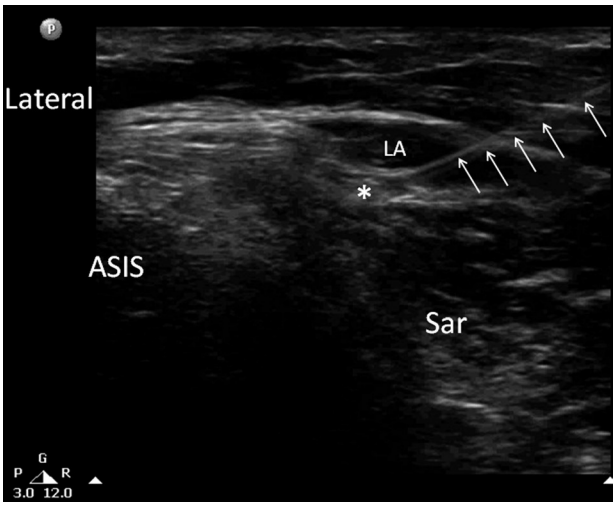

FIGURE 4. Postinjection ultrasonogram; the needle is indicated by line arrows. LA indicates local anesthetic. *LFCN. Reproduced with permission from USRA (www.usra.ca).

Blockade of the LFCN with local anesthetics and steroid is usually offered to patients as part of the multimodal therapy with pharmacological treatment and lifestyle modification.[16,42,67,69] Although the optimal frequency and dose has not been well examined, the procedure may be repeated for up to 3 to 5 times if required.[67] Different studies showed excellent results from the LFCN block. Relief of symptoms can be achieved in 66% to 91%.[6,12,67,71] In addition to the anti-inflammatory properties, steroids also possess membrane-stabilizing properties by suppressing the transmission in unmyelinated C fiber and inhibiting ectopic discharge from the experimentally created neuroma.[72,73] It has been demonstrated that the partial loss of function in small fibers may also account for the painful symptoms of patients with MP, especially in those with longer disease duration.[55] Pulsed radiofrequency lesioning of the nerve for treatment of intractable MP had been reported,[12,74] but a large-scale study is required to determine its effectiveness. If symptoms persist despite the conservative measures, surgical interventions may be required.

Surgical Treatment

Surgical intervention should be considered when nonoperative treatment fails and symptoms become intractable and disabling.[16,42] The literature on the surgical treatment for MP is lacking. It is not surprising as a survey of the neurosurgeons in United States revealed that 70% of neurosurgeons do not perform any LFCN procedures and that only 25% did less than 10 such procedures per year.[75] Three types of surgical intervention have been described: neurolysis of the constricting tissue around the nerve with or without transposition of the LFCN and transection with excision of a portion of LFCN. The technical details of the surgical treatment are beyond the scope of this review but described elsewhere.[16]

CONCLUSIONS

Meralgia paresthetica is an entrapment pain syndrome of the LFCN of thigh. Diagnosis is mainly made on clinical ground with pain and paresthesia of the anterolateral thigh. Electrophysiological test and nerve block play important roles when the diagnosis is uncertain. Clinicians should be aware of the multiple etiological factors that can be potentially modified or treated. Most of the patients respond to conservative measures including

nerve block. Surgical options should be considered in patient refractory to those treatment options.

Anesthesiologists are commonly involved in the management of MP because of their expertise in pain management and the performance of the LFCN block. Blockade of the LFCN with local anesthetics and steroid serves both the diagnostic and therapeutic role. The practitioner should be familiar with the variation of anatomy and the limitation of the landmark-based technique. Ultrasound-guided injection technique has recently been described and offers an improvement in the accuracy of the nerve blockade.

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