Meralgia Paresthetica—A Common Cause of Thigh Pain

Meralgia paresthetica is caused by impingement of the lateral femoral cutaneous nerve. Careful history can help identify this mononeurapathy and lead to successful treatment.
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Meralgia paresthetica (MP), a common condition seen by both primary care physicians and specialists, is easily misdiagnosed because it can mimic other disease processes. Often, merely the awareness of the condition and knowledge of a few key differentiating factors helps the clinician quickly recognize this disorder.

MP is a mononeuropathy caused by impingement of the lateral femoral cutaneous nerve (LFCN), which supplies sensation to the lateral aspect of the thigh. When impingement or entrapment of the nerve occurs, the patient can experience numbness, burning, stabbing, and aching along the well-delineated path of the LFCN, from the front of the thigh to just above the knee (Figure 1).

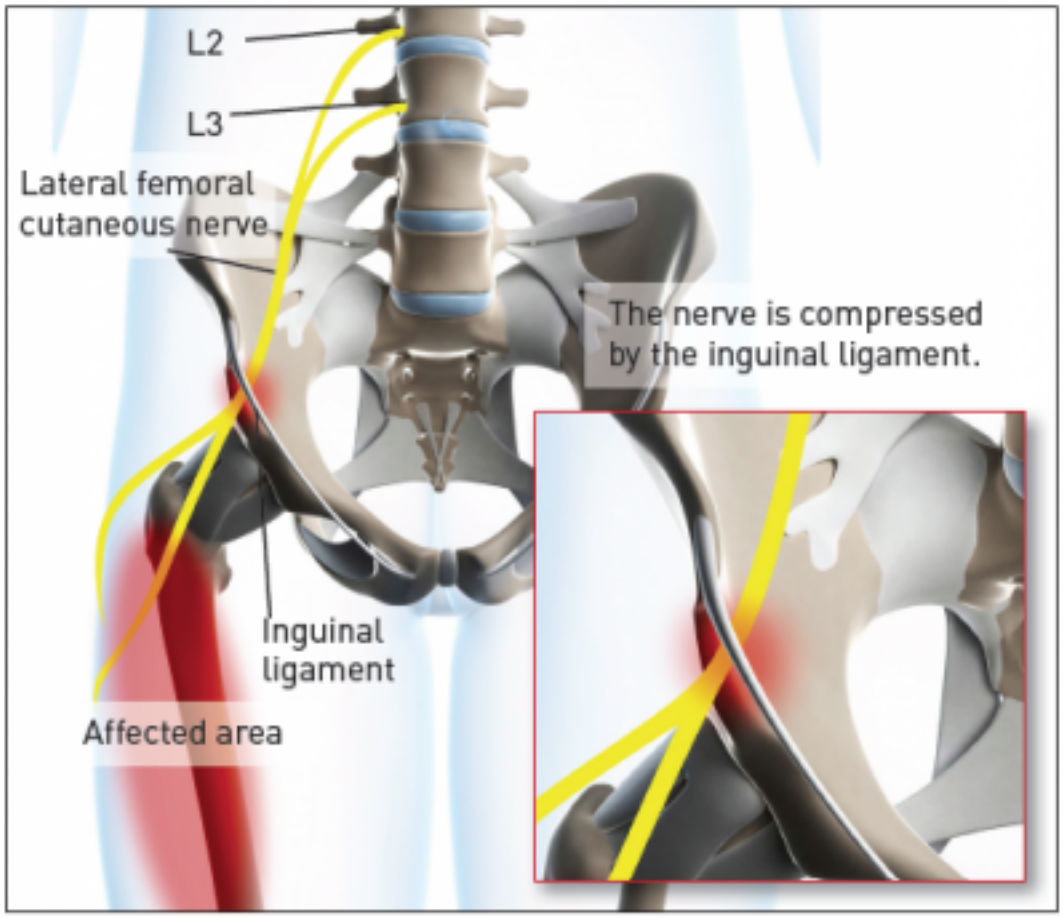

Figure 1. Distribution of symptoms in meralgia paresthetica.

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The most common cause of impingement of the LFCN is entrapment of the nerve under the inguinal ligament (Figure 2), which can occur spontaneously or develop after an injury.[1] Causes of LFCN nerve entrapment can be divided into 3 categories: mechanical, metabolic, and iatrogenic. Pregnancy (or any condition that increases abdominal pressure), obesity, wearing tight clothing/belts in the waist area, different leg lengths, and pubic symphysis (pelvic girdle) dysfunction are common mechanical issues leading to MP.[2] It also has been recognized that carrying items such as a wallet or a cell phone in the front and side pockets of pants can cause unintentional compression of the nerve. Metabolic causes include neuropathy (from diabetes or alcoholism), hypothyroidism, and lead poisoning. Clinicians should ask patients about occupational and living conditions that may have exposed them to lead paint. Prolonged traction during spine surgery or injury to the nerve during retroperitoneal dissection are common iatrogenic causes; therefore, a surgical history is important to consider.[3]

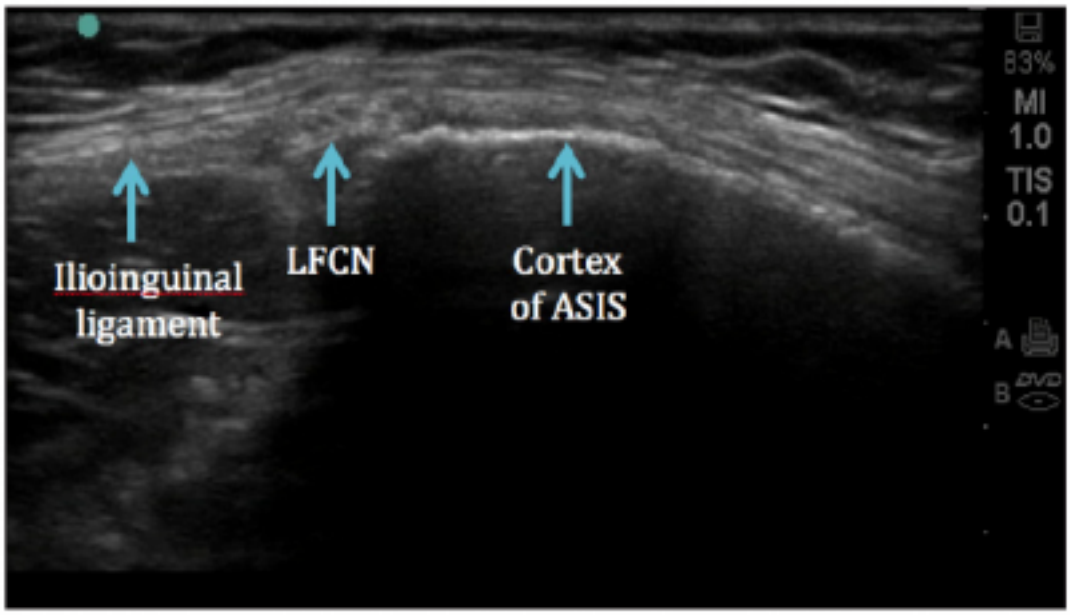

Figure 2. Entrapment of the lateral femoral cutaneous nerve between the ilioinguinal ligament and anterior superior iliac spine.
ASIS, anterior superior iliac spine; **LFCN**, lateral femoral cutaneous nerve
Image courtesy of Dr. Joseph Ruane.

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Differential Diagnosis

It is imperative to differentiate MP symptoms from other causes of pain and nerve discomfort that can have similar clinical presentations. Included in the differential are:

- Spinal nerve radiculopathy at L1-L3[4]
- Malignancy or metastasis to the iliac crest[5]
- Uterine fibroids or pelvic mass that compress the nerve[6]
- Avulsion fracture of the anterior superior iliac spine (ASIS)[7,8]
- Chronic appendicitis[9]

Hip pain that presents with neurological, urogenital, or gastrointestinal symptoms should prompt a more

focused evaluation to rule out MP.[10] Although symptoms may vary between patients, the differential diagnosis of MP is limited, and it usually can be distinguished through clinical evaluation and diagnostic workup.

Clinical Evaluation

The patient's medical history can elicit key information to direct the clinician to the diagnosis of MP. For example, a thorough discussion of the type of clothing the patient wears (tight), use of thick belts, or work apparel that can lead to pinching at the ASIS can elicit whether symptoms are worse during their use. Another clue is if symptoms worsen with prolonged standing and hip extension and improve with sitting.[11]

The pelvic compression test is a diagnostic tool that involves placing the patient in the lateral recumbent position while an external downward force is applied with the examiner's hands over the lateral aspect of the ASIS. The pressure is held for 45 seconds to determine if the patient's symptoms improved. Symptom amelioration is considered a positive test result and helps to rule out lumbosacral radicular pain.[12] In a study conducted by Nouraei et al, 19 out of 20 patients who had abnormal nerve conduction studies also had a positive pelvic compression test.12 Based on these results, the pelvic compression test has a sensitivity of diagnosing MP of 95%. The same study found that a negative pelvic compression test was found in 14 out of 15 patients, yielding specificity of 93.3%. The course of the LFCN, from the abdomen to the thigh, can be variable, with 5 separate anatomic subtypes described.[13]

Sensory testing with light touch or monofilament testing can be performed with the patient in the supine position to determine the presence of numbness or paresthesia along the LFCN distribution. The LFCN is a sensory nerve only, so the patient will not have any resulting motor weakness in true MP. The Tinel's sign also can be used to elicit tenderness and sensitivity over the lateral aspect of the inguinal ligament just medial to the ASIS (where the LFCN typically runs).[14] In the LFCN distribution, often there is a small area of hair loss on the thigh due to persistent rubbing of the area by the patient.[10]

A diminished or absent cremasteric reflex (in male patients) can indicate L1 nerve damage, which would eliminate MP as a possible diagnosis. Manual muscle testing of myotomes L2-L5 is critical, because again, LFCN pathology will not cause motor dysfunction in any of lower extremity muscles. The patellar tendon reflex will be normal, and a straight leg test also should be performed to rule out lumbar radiculopathy.[10] If a motor or sphincter dysfunction is present on exam, the patient's symptoms likely are secondary to a spinal cord or nerve root lesion.

Diagnostic Evaluation

Plain radiographs (x-rays) of the pelvis and hip (AP and frog leg views) should be obtained first to rule out osteoarthritis of the hip joint or bone metastasis to the ileum.[5] Magnetic resonance imaging or computed tomography can rule out disc herniation, nerve lesions, annular tears, or other spinal pathology that may be causing radiculopathy. A pelvic ultrasound should be obtained in women of childbearing age with a history of prolonged menstrual bleeding to rule out uterine fibroids.[6] A detailed history also will indicate a history of alcoholism or lead exposure.

During the initial work-up, the clinician should order blood work to test for thyroid function, vitamin B12 and folate levels, and serum lead levels because these are common causes of MP.[3] A complete blood count may be ordered to evaluate for macrocytic anemia and diabetes in patients with neuropathy symptoms.

A nerve test (blockade) may be both diagnostic and therapeutic in patients suspected of having MP. Using a nerve stimulator, the LFCN can be located and injected with a local anesthetic. Relief of the numbness and pain confirms the diagnosis of MP. Newer methods of conducting nerve blocks include

landmark-based and ultrasound-guided techniques.[14] The landmark approach can present a challenge because the normal course of the LFCN and anatomy can vary in patients. This method involves insertion of a needle, 2.5 cm medial to the ASIS and caudal to the inguinal ligament. Lidocaine can be injected when a “loss of resistance” or “pop” is felt as the needle goes through the fascial layer.[3,8,15]

Since the landmark approach essentially is a blind approach, ultrasound-guided techniques greatly improve the success rates for contacting the LFCN (84% vs 5%, respectively).[16] If ultrasound cannot identify the LFCN, an injection of 5% dextrose can be used to dissect between the fascia lata and the iliacus muscle, decompressing the trapped nerve.

A study by Dureja et al examined the benefits of using regular steroid and bupivacaine injections (minimum of 5 total injections), with the use of diphenylhydantoin as a possible treatment of MP. These regular injections are thought to disrupt the afferent efferent loop at the spinal level, thereby reducing the pain caused by irritation of the LFCN.[14] In combination with injections, patients can also be prescribed tricyclic antidepressants and anticonvulsants to help treat neuropathic pain.[17]

The diagnostic capability of nerve conduction studies, however, has been questioned. A study by Seror et al demonstrated that somatosensory-evoked potential (SSEP) studies of the LFCN were not sensitive or specific for the diagnosis of MP. In this study, SSEP shows sensitivity of 5% and specificity of 95%, whereas thigh stimulation had a sensitivity of 52% and specificity of 76%. Following this study, it is generally accepted that SSEP testing is not effective in the diagnosis of MP.[18]

Neurodiagnostic testing is highly dependent on the skill of the user to obtain an accurate result. Two techniques for evaluating nerve conduction have been described: stimulating the LFCN as it exits the pelvis near the ASIS and recording potentials distally, or stimulating distally and recording proximally in the region of the ASIS. The responses are typically of small amplitude; therefore interpretation is highly user-dependent. It is important to study both sides to compare amplitudes, and the study can be difficult to perform in an overweight individuals.[13]

Treatment Options

The treatment of MP firsts consists of removing any underlying cause of the impingement (tight pants, belts, wallets, cellphone). This is followed by conservative treatment with oral anti-inflammatory medications. Typically, 85% of patients will have improved symptoms with conservative treatment alone.[19] Patients who are obese should be counseled to lose weight, which can aid in resolution of their symptoms.

Physical therapy should be considered as a non-surgical modality to treat MP, although there is no current data that supports its effectiveness.[20] Manual therapeutic techniques, including Active Release Technique and myofascial therapy for the rectus femoris and iliopsoas muscles, have been suggested as viable options, but most of the evidence for these techniques come from individual case studies.[20] Other useful therapies include moist heat, transcutaneous electrical nerve stimulation (TENS), phonophoresis, and use of soft tissue techniques that can improve pain and increase range of motion.[21]

Use of non-steroidal anti-inflammatory medications (NSAIDs) generally is accepted among the literature as part of the initial treatment, although there does not appear to be a consensus about any particular NSAID or duration of treatment. It is important to keep in mind contraindications to NSAID use, including, but not limited to, gastrointestinal ulcers, kidney impairment, and bleeding disorders. Oral steroids also can be considered as a conservative option for management of inflammation, although it appears that there is no current research to support this.

Injection therapies using either the landmark-based or ultrasound-guided techniques should be attempted prior to surgical intervention. Symptoms that are unyielding or disabling can be referred for earlier surgical evaluation.[13]

Surgical management to correct the impingement typically is reserved for patients who continue to have symptoms despite treatment with conservative measures. Three approaches to surgical treatment of MP include: neurolysis of constricting tissue, neurolysis and displacement of LFCN, and excision of part of the LFCN.[13] A study by Son et al examined the effectiveness of neurolysis as a means of treating persistent MP. The study found the technique provided complete relief in 81.8% of patients and partial relief in 18.2% of patients.22 LFCN decompression can be performed surgically with a 2- to 3-cm incision inferior to the ASIS along the line of the inguinal ligament. Careful blunt dissection of the fascial planes can identify the LFCN and allow the surgeon to follow it towards the inguinal ligament. At the point where the LFCN meets the inguinal ligament, the ligament is divided to decompress the nerve.[12] Surgical management typically is the last option for treatment of MP; again conservative measures should be performed first in the treatment plan.

Summary

In our experience, the prevalence of MP has been increasing, perhaps because of the demographic shift in risk factors including obesity, jobs that require prolonged sitting, and fashion trends. The well-demarcated zone of pain and paresthesias that characterizes MP is the primary clue that raises a clinician's index of suspicion in identifying this condition. Patient education is paramount because modifying risk factors are essential to resolving nerve irritation. Ultrasound has become a valuable tool for identifying nerve entrapment and accurately guiding therapeutic injections. Hydrodissection of tissues is an evolving option, poorly described in the literature but practiced by many versed in the technique. Advanced imaging and neurodiagnostic testing often is not necessary when the history and clinical evaluation are consistent for MP.

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