ACU Sağlık Bil Derg 2017(4):185-191

**DERLEME** / REVIEW **Fiziksel Tıp ve Rehabilitasyon** / Physical Medicine and Rehabilitation

# Entrapment Neuropathies of The Lower Extremities

**Meral Bayramoğlu**[1]

[1]Acıbadem Mehmet Ali Aydınlar University Faculty of Medicine, Department of Physical Medicine and Rehabilitation, Istanbul, Turkey

Meral Bayramoğlu, Prof. Dr.

**ABSTRACT**

**Peripheral nerves of the lower extremities might be compressed on their course where the anatomic configuration puts them in a vulnerable position. Neuropathic states can also be the result of any kind of trauma which directly injures the nerves or leads to a state of inflammation around the nerves. A wide variety of etiologies, as well as clinical presentations, may lead to diagnostic challenges for the clinician. The main symptom of a peripheral neuropathy is paresthesia. This could be accompanied by pain and numbness depending on the severity of the compression. The lumbosacral plexus, which arises from the ventral rami of the L1-S3 roots, serves the lower extremities. There are particular anatomic sites where the nerves are more vulnerable. A clear identification of the anatomic course, and motor and sensory distribution of each nerve arising from the lumbosacral plexus, is critical in localizing the injury and planning the optimal treatment. Electrodiagnostic studies help localize the site of the lesion, give a clue about the severity and potential recovery, and help differentiate any plexopathy and/or radiculopathy. Imaging studies, mostly magnetic imaging, can be ordered to help confirm the entrapment or exclude other pathologies. Most, but not all, of the cases can be treated by conservative measures. Common entrapments of the lower extremities, namely, meralgia paresthetica, femoral, obturator, sciatic, peroneal and tibial neuropathies will be discussed in this review.**

**Key words:** neuropathy, lower extremity, entrapment

**ALT EKSTREMİTELERİN TUZAK NÖROPATİLERİ**

**ÖZET**

**Alt ekstremite periferik sinirleri, traseleri boyunca anatomik olarak duyarlı bulundukları bölgelerde sıkışabilir. Siniri direkt olarak etkileyen herhangi bir travma veya sinir çevresindeki bir inflamatuar durum da nöropatiye neden olabilir. Hem etyolojinin, hem de klinik semptom ve bulguların çok çeşitli olabilmesi, klinisyenin tanıda zorluklarla karşılaşmasına neden olabilir. Bir periferik nöropatinin ana semptomu parestezidir; buna sıkışma şiddetinin derecesine bağlı olarak ağrı ve hissizlik eşlik edebilir. Alt ekstremitelerin innervasyonu L1-S3 köklerinin anterior ramuslarından oluşan lumbosakral pleksus tarafından sağlanır. Sinirlerin zedelenmeye daha duyarlı olduğu belirli anatomik bölgeler vardır. Hasar bölgesini saptamak ve uygun tedaviyi planlamak için lumbosakral pleksustan çıkan her sinirin motor ve duyu dağılımının net olarak ayırt edilmesi önemlidir. Lezyon bölgesinin saptanması, lezyonun şiddeti ve iyileşme potansiyeli ile ilgili ipuçları vermesi, pleksopati/radikülopati ayrımının yapılabilmesi açısından elektrodiagnostik çalışmalar oldukça faydalıdır. Görüntüleme yöntemlerinden özellikle manyetik rezonans görüntüleme, tuzak nöropatisi tanısının desteklenmesi veya diğer patolojilerin ekarte edilmesi için istenebilir. Tuzak nöropatilerin hepsi değilse de çoğu konservatif olarak tedavi edilebilmektedir. Bu derlemede alt ekstremitelerde sıkça görülen nöropatiler olan meralgia parestetika, femoral, obturator, siyatik, peroneal ve tibial nöropatilerden bahsedilecektir.**

**Anahtar sözcükler:** Nöropati, alt ekstremite, tuzak

**Correspondence:**
Prof. Dr. Meral Bayramoğlu
Acıbadem Mehmet Ali Aydınlar University Faculty of Medicine, Department of Physical Medicine and Rehabilitation, Istanbul, Turkey
**Phone:** +90 216 655 44 96
**E-mail:** meral.bayramoglu@acibadem.com.tr

**Received :** 13 January 2016
**Revised :** 13 January 2016
**Accepted :** 03 March 2016

Patients with peripheral nerve entrapments of the lower limbs may present with poorly localized symptoms and it is a challenge for the physician to distinguish the etiology of the symptoms in the leg and foot. There may be motor functional alterations like weakness, atrophy, sensory dysfunctions like pain and paresthesias, or autonomic dysfunction like ulceration of the involved skin area. The main causes of

the entrapment neuropathies are, mechanical compression mostly at fibrous/fibro-osseous tunnels and trauma of any type occurring through the course of the nerve. Some medical conditions where there is an accumulation of whole body fluid, the presence of mass occupying lesions such as tumor, scar or aberrant muscle, may also lead to compression of the peripheral nerves. A careful physical examination, aided by electrodiagnostic studies, and if necessary, imaging studies, will help the clinician find out the underlying etiology, and tailor the optimum treatment for the patient.

The purpose of this review is to present the etiology, various symptoms, diagnostics, other possible disease conditions, treatment options-rehabilitative/conservative or surgical-of lower extremity entrapment neuropathies. Of the published literature about lower extremity neuropathies, a few of them as old as published in 1982, have historical value and included because of that. Literature describing well-known etiologies, as well as case reports defining rather rare factors are selected.

## Meralgia paresthetica

Compression of the lateral femoral cutaneous nerve (LFCN) results in a condition known as meralgia paresthetica (MP), causing paresthesia, tingling, and/or burning sensation along the anterolateral aspect of the thigh. Severity of the symptoms can range from mildly uncomfortable to painfully disabling (1). Disabling pain in some patients may lead to severe limitations in activities of daily living although there is no muscle weakness. Early observations often related the condition to severe toxic disorders, such as lead poisoning, chronic alcoholism (2). Local mechanical factors have been blamed. Historically, the most common etiologic factor causing direct impingement of the nerve has been related to the tethering of a belt in an obese individual (3). Later in 2007, the same relation was supported in a study with young females wearing trendy low cut trousers (4). Surgical procedures such as inguinal hernia repair, iliac bone graft harvesting, hip surgery, or renal transplantation may also affect the nerve (5). In these conditions, neuropathy presentation may be masked at the beginning. Laparascopic cholecystectomy (6), laparoscopic myomectomy (7), aortic valve surgery (8), coronary artery bypass grafting (9) as well as gastric reduction surgery for morbid obesity (10–12) have been reported as iatrogenic causes of MP. Increased cases of injuries to the LFCN have been reported after laparoscopic inguinal hernia repairs by surgeons (13,14). The LFCN is a pure sensory nerve which originates from the L2–3 roots lateral to the psoas major muscle. It exits the pelvis by directly piercing the inguinal ligament or by running deep to that structure, making it prone to injury (15). There are various reports demonstrating that the LFCN may run many centimeters medial or lateral to the anterior superior iliac spine (16–21). Clinical symptoms with a characteristic distribution of pain in the anterolateral thigh, together with the absence of any motor or reflex abnormalities are usually enough to make the diagnosis. Imaging or nerve conduction studies are needed only in atypical cases. Especially in obese patients, it is difficult to confirm the LFCN neuropathy by nerve conduction studies. The use of EMG helps exclude other conditions, such as L2 radiculopathy, high lumbar plexopathy or femoral neuropathy.

Spontaneous improvement of the symptoms is the expected course of MP, so conservative measures should be the first choice. These include weight loss in obese patients, removal of extrinsic compressors such as tight trousers or belts, application of transcutaneous electrical nerve stimulation (TENS) and/or icing the area over the anterolateral thigh. The benefits of tricyclic antidepressants, antiarrhythmic agents, and anticonvulsants in patients with persistent symptoms have been documented (22). Capsaicin has been used to treat itch and surface hypersensitivity in MP (23). Local anesthetic and steroid infiltrations under ultrasound guidance can be helpful, and be repeated at intervals (24). Despite the measures taken, if the symptoms become intractable and disabling, surgical treatment becomes an option. Basic techniques are neurolysis of the constricting tissue, transposition of the LFCN, and transection with excision of a portion of the nerve. Once the diagnosis is made, MP usually responds favorably to treatment.

## Femoral neuropathy

Femoral nerve is the largest branch of the lumbar plexus, arising from L2-L4 roots. It travels down between the psoas and iliacus muscles, sends motor branches to these muscles, passes under the inguinal ligament lateral to the femoral artery and vein. In the thigh, it divides into several anterior and posterior branches. The muscular branches supply four heads of the quadriceps muscle, sartorius and pectineus. The sensory branches supply the anterior thigh and medial leg.

Isolated femoral neuropathy is uncommon and most commonly iatrogenic, within the retroperitoneal space or under the inguinal ligament (25). Pelvic and intraabdominal surgical procedures, as well as hip procedures may result in injuries of the femoral nerve. Femoral neuropathy has been reported after many operations like inguinal herniorraphy (26), spinal surgery (27,28), aortic clamping during vascular surgery (29), after operation for an acetabular fracture (30), as well as after radiotherapy to the inguinal area (31). Lithotomy position during vaginal hysterectomy, which is the most unphysiological position with exaggerated abduction and external rotation at the hip, as a cause of femoral neuropathy has been reported (32,33). This position leads to kinking and twisting of the nerve beneath the tough inguinal ligament. Hemorrhages in patients under anticoagulants (34,35) are also common causes for femoral neuropathy. As a rare injury mechanism of femoral nerve injury, use of tourniquet for surgery of a patellar fracture was reported (36).

The clinical picture is unilateral feeling of instability around the knee and weakness of knee extensors, sometimes atrophy of the quadriceps. The patellar reflex is usually absent and there is loss of sensation of the anteromedial thigh and medial leg. Neuropathic pain in the same area may be present. If the lesion is proximal to the inguinal ligament, hip flexion weakness may also be present due to the involvement of the iliopsoas.

Electrodiagnostic studies help localize the lesion site and assess the degree of axonal loss. They also help to rule out high lumbar plexopathy or radiculopathy. If the case is not bilateral, comparison with the normal side should be made to give an idea about the prognosis for recovery. Motor weakness persisting for less than 2 months is a good sign for almost total recovery. Weakness for more than 6 months usually indicates a permanent loss. Prevention, in cases of operations performed especially in the lithotomy position, is important. Early supportive treatment consisting of physiotherapy for both controlling the pain and supporting the knee is important. This could be performed by using transcutaneous electrical stimulation, together with active, active assistive or passive quadriceps exercises depending on the severity of motor weakness. Chronic neuropathic pain may respond to neuropathic pain medication, if persistant, percutaneous nerve stimulation may be an option (37). If direct nerve injury is suspected, or if there is an iliopsoas hematoma, surgical exploration is recommended by some authors (38,39).

Saphenous nerve is the posterior division and pure sensory branch of the femoral nerve which supplies the skin over the knee, medial lower leg and medial foot. It takes off from the femoral nerve near the inguinal ligament, proceeds through the subsartorial adductor canal, then pierces the subsartorial fascia about 10 cm proximal to the medial femoral condyle. It then descends along the medial surface of the tibia and medial malleolus. It can be affected at any site through this course. The most vulnerable part is the exit through the subsartorial fascia where it is quite superficial. Arthroscopic surgery (40–42), trauma (43–45), compression (46) are the causes of saphenous neuropathy. The symptoms vary from only mild sensory loss in the nerve's supplying area to severe neuropathic pain. No motor weakness is expected. Pure saphenous neuropathies are rarely reported and it is usually technically difficult to confirm the diagnosis with electrodiagnostic studies in clinically suspected cases. Needle examinations help rule out femoral neuropathy, L4 radiculopathy or lumbar plexopathy. If conservative measures like TENS, neuropathic medication fail, neurolysis may become an option for treatment.

## Obturator neuropathy

The obturator nerve arises from the anterior divisions of the ventral rami of L2-L4 nerve roots, descends through the psoas muscle, proceeds anterior to the sacroiliac joint, crosses the obturator canal, then divides into an anterior and a posterior branch. The anterior branch runs between the adductor longus and brevis, and gives motor supply to these muscles as well as to gracilis, and in some cases, the pectineus. Posterior branch provides motor innervation to obturator externus and a portion of adductor magnus. Sensory fibers from these branches innervate the hip joint, the medial knee joint, and the medial aspect of the mid-tigh.

Isolated obturator nerve injury is rare. Obturator neuropathy in athletes by fascial entrapment in the adductor compartment has been described (47). Lithotomy positioning as a cause of obturator nerve injury has also been reported (48). Trauma to the pelvic region as well as compression from tumor usually result in obturator nerve injury associated with other nerve injuries (49–52).

Most common presentation is sensory alteration in the groin or medial thigh. Patients may feel pain, sensory loss or paresthesias. Manoeuvres that stretch the nerve, like extension of the hip may exacerbate the pain. If the reason for nerve compression is trauma or surgery, pain symptoms may be masked. Medial thigh wasting can be observed. A circumducted gait may be observed due to weakness of adduction and internal rotation of the leg.

Needle EMG shows denervation of the short and long adductor muscles. Nerve conduction studies are not helpful. MRI may detect atrophy of the adductor brevis and longus and gracilis muscles, it is also useful to rule out intrapelvic mass lesions compressing the obturator nerve.

Pain medication together with physical therapy and rehabilitation measures consisting of both electrical stimulation and strengthening exercises help relieve the pain and improve strength. Surgical release has proved successful in resistant cases (47).

## Sciatic neuropathy

The sciatic nerve is the largest and the longest nerve in the body. It originates just distal to the lumbosacral plexus, extends to the feet, and it is responsible for most of the functions of the lower extremity. It arises from the ventral divisions of L2-S3, enters the gluteal region through the greater sciatic foramen. It is formed by a larger tibial and a smaller peroneal trunk in a common nerve sheath. There is a close relationship to the piriformis muscle which also exits the pelvis via the greater sciatic notch, as well as the posterior femoral cutaneous nerve, inferior gluteal artery, pudendal vessels and nerves. The sciatic nerve runs between the ischial tuberosity and the greater trochanter distal to the piriformis, in the proximal thigh, it is anterior to the adductor magnus and posterior to the hamstrings. Approximately 6 cm above the popliteal fossa, it divides into

common fibular (peroneal) and tibial branches. The common fibular nerve supplies the anterior and lateral compartments of the lower leg by the deep fibular and superficial fibular branches respectively. The superficial fibular nerve also has a sensory branch which supplies the anterolateral lower leg and dorsum of the foot. The deep fibular nerve has a sensory branch to supply the webspace between the first and second toes. The tibial nerve innervates the posterior calf muscles and supplies sensation to the posterior calf and lateral foot by the sural nerve, the sole of the foot by the medial and lateral plantar nerves, and the heel by the medial calcaneal nerve. There is no sensory branch arising from the sciatic nerve in the thigh. Posterior thigh sensation is supplied by the posterior femoral cutaneous nerve which directly arises from the lumbar plexus.

The sciatic nerve is more commonly injured in the pelvis or gluteal region than distally in the thigh. Fractures of the hip joint, posterior hip dislocations commonly affect the nerve. Total hip arthroplasty may cause entrapment in the retroacetabular region, an iatrogenic cause of neuropathy (53–56). Varicotic gluteal veins have been reported as etiologic factors for painful vascular compression of the sciatic nerve (57,58) where 3 patients had surgical release and one patient got relief from carbamazepine.

Piriformis syndrome is a condition where a hypertrophied piriformis muscle leads to a narrowing of the greater sciatic foramen resulting in sciatic nerve compression (59). Symptoms in piriformis syndrome are often nonspecific like a dull ache in the hip, going down the back of the thigh, worsened by prolonged sitting and sometimes reduced range of motion of the hip joint. It is usually considered as a diagnosis of exclusion.

More distally, the nerve may be trapped by a scar tissue formed because of a previous hamstring injury (60).

There are cases where a cause cannot be identified-idiopathic sciatic neuropathy (61). No etiologic factor could be identified in 16% of 73 patients in a study (62).

Patients with sciatic neuropathy present with pain radiating down the posterolateral limb, weakness and numbness follow the pain. The common fibular division is more vulnerable to compression because of its fewer and larger fascicles and less supportive tissue compared with the tibial division. Its more superficial location, a smaller blood supply, and its fixation at two points-the sciatic foramen and the fibular head-also make it more vulnerable. The findings sometimes mimic a common fibular neuropathy at the knee because foot drop can be seen in both conditions due to denervation of anterolateral leg muscles. Weakness of hip extension and knee flexion because of hamstring involvement, and weakness of muscles in the peroneal and tibial nerve distribution may be seen in varying degrees. Weakness of the hamstrings and plantar flexors of the ankle may be hard to detect clinically, as they are very powerful muscles innervated by the tibial nerve, whereas less powerful dorsiflexors and evertors of the ankle which are innervated by the peroneal division may be more easily detected, if they are weak. Achilles reflex is diminished or absent, while patellar reflex is preserved. Sensory findings range from pain and sensory loss in the foot and possibly the lateral shin to dysesthetic pain and numbness in the sole and dorsum of the foot and lateral lower leg.

Electrodiagnostic studies are useful for both localizing the site and severity of the lesion and assessing recovery and prognosis. There will be reduced amplitudes of the superficial peroneal sensory and sural sensory responses in nerve conduction studies. Tibial and peroneal motor responses will also be low. The needle examination is helpful in localizing the lesion. Denervation will be seen in peroneal and tibial innervated, and more proximal hamstring muscles. A more proximal plexus injury or L5-S1 radiculopathy should be excluded by examination of other gluteal muscles and paraspinal muscles.

Treatment is planned according to the etiology of the sciatic neuropathy. Neuropathic pain is found in almost all cases and neuropathic pain medications are widely used for symptomatic relief of pain. Neuropathy because of hip fracture or dislocation needs surgical treatment. In piriformis syndrome, treatment of choice is conservative, physical measures, stretching of the hip joint. Steroid or botulinum toxin injections have been used in piriformis syndrome (63,64). Orthotics for foot drop are commonly used to improve both safety and speed of ambulation.

## Fibular (peroneal) neuropathy

The common fibular nerve is formed by the L4-S1 nerve roots. Together with the tibial nerve, it runs in the posterior thigh as a part of the sciatic nerve. Proximal to the popliteal fossa, they are separated, and fibular nerve gives off a muscular branch to the short head of biceps femoris, which is the only muscle that it innervates proximal to the knee. Within the popliteal fossa, it gives off the lateral cutaneous nerve of the thigh, supplying sensation of the skin of lateral upper leg. It wraps around the fibular head and neck, where it separates into the superficial and deep fibular nerves. This is the site where the nerve is very vulnerable to trauma. The superficial fibular nerve innervates the ankle everters and gives sensory branches to the lateral lower leg and foot. The deep fibular nerve enters the anterior leg compartment and innervates the ankle dorsiflexors and toe extensors. Its terminal branch supplies sensory innervation to the first web space.

The superficial position of the common fibular nerve at the fibular neck makes it susceptible to injury, thus, peroneal (fibular) mononeuropathy is the most common mononeuropathy in

the lower extremity. Direct blows or lacerations to, or fracture and dislocation of the fibular head and neck may lead to nerve injury. People who squat or kneel for extended periods of time, or who cross the knees for prolonged periods might have neuropathy because of external compression. Casts and braces, as well as improper positioning during surgery can also cause direct compression. Popliteal lipoma (65), baker cyst, neuroma, callus formation from fibular fractures can cause internal compression of the nerve. Previous patellectomy, ankle sprain, manipulation for hallux valgus were reported as etiologies for injury (66). Compression secondary to popliteal venous aneurysms (67), tibiofibular ganglion cysts (68), lipoma (69), severe burn injury (70) have been reported to lead to fibular nerve injury, but they are rare. Anterior compartment syndrome can lead to isolated injury to the deep branch by pressure effects.

Patients with fibular nerve lesions will present with complete or partial foot drop. Pain is not a common symptom, it usually depends on the type of injury (ie, chronic compression will not cause pain, whereas traumatic wounds have pain). Typical gait pattern is described as steppage gait where the patient tries to hyperflex the knee and hip to clear the drop foot from ground. Patients may have relative sparing of the ankle everters, so dorsiflexion strength should be tested with the foot in inversion to isolate the tibialis anterior. Patients may have hypoesthesia of the anterolateral surface of the lower leg and dorsum of the foot. A positive Tinel sign at the fibular head may be found. Reflexes are spared.

Electrodiagnostic studies are helpful in localizing the lesion. Sensory and motor conduction studies should be performed also on the contralateral side for comparison. Needle electromyography helps to rule out other possible causes of foot drop, and gives information about the severity of the axonal loss.

Treatment is usually to avoid the precipitating factors. Conservative measures are generally indicated for about 3–4 months where spontaneous resolution is expected. Electrical stimulation and active/active assistive/passive exercises of the peroneal muscles help during this period. Ankle foot orthoses provide improved gait in patients with foot drop. When conservative measures fail to restore function, surgical decompression of the nerve is indicated. In cases of entrapment of the nerve by ganglion cysts or other space occupying lesions compressing the nerve, early surgical treatment will be a better treatment choice than conservative management. Although rare, if pain is present, neuropathic pain medication such as tricyclic antidepressants or gabapentin can be used.

## Peroneal neuropathy at the ankle

Deep peroneal nerve, branching 1 cm proximal to the ankle joint has one mixed branch to innervate the extensor digitorum brevis (EDB) and provide sensation to the lateral tarsal joints, and another branch consisting of only sensory fibers, providing cutaneous innervation to the first dorsal web space. This branch passes under the inferior extensor retinaculum together with the dorsalis pedis artery and vein, and the tendons of extensor hallucis longus (EHL), extensor digitorum longus (EDL), tibialis anterior and peroneus tertius. Compression at this site may be acute, as in any trauma involving the anatomic region, or it may be a chronic process such as tight shoe laces, talonavicular osteophytosis, any space occupying lesion, and pes cavus (71). This type of nerve compression is also called anterior tarsal tunnel syndrome. Typical presentation is pain on the dorsum of the foot and numbness in the first dorsal web. Electrodiagnostic studies help diagnosis. General physiotherapy measures are used for treatment.

## Tibial neuropathy

Composed of L4, L5, S1, and S2 nerve roots, tibial nerve is a part of sciatic nerve. It supplies all hamstring muscles except the short head of the biceps femoris, also provides partial innervation to the adductor magnus. In the popliteal fossa, a medial sural cutaneous branch exits to form the sural nerve together with the lateral sural cutaneous branch of the fibular nerve. In the leg, it supplies the posterior compartment of the lower leg. Passing posterior to the medial malleolus through the tarsal tunnel, it divides into the terminal branches, the calcaneal, medial and lateral plantar nerves. When compared with the more superficially located fibular nerve, tibial nerve injuries are rare due to the nerve's deep course within the posterior leg. Proximally, in the popliteal fossa, space occupying lesions like Baker cysts (72) may be the cause of injury. Compression by the arch of soleus has been reported (73). Patients with proximal tibial lesions present with varying degrees of weakness of plantar flexion and inversion of the ankle, loss of toe flexion, and calf atrophy. Toe raises may be hard to perform, together with sensory loss in the sole of the foot and the posterolateral lower leg and foot. Achilles reflex is absent.

Distally, the tibial nerve is compressed under the flexor retinaculum, where it passes posterior to the medial malleolus and medial to the talus and calcaneus, and it is referred to as tarsal tunnel syndrome. Within the tarsal tunnel, the nerve divides into the three terminal branches, medial and lateral plantar nerves and medial calcaneal nerve in most patients (74). Space occupying lesions such as ganglion cysts (75), exostosis, accessory musculature within the canal are some possible factors (76). Other etiologic factors are ankle trauma and, fibrosis of the flexor retinaculum (77). In some cases, it is idiopathic.

The symptoms of tarsal tunnel syndrome are numbness of the foot radiating to the first 3 toes, burning sensation of various parts of the sole of the foot depending on the site of the entrapment. Ankle pain may be present if the tibial nerve is compressed more proximally. The objective physical findings

are few. With careful examination and comparison with the other foot, weakness or atrophy of the intrinsic foot muscles may be noticed. There may be hypoesthesia on the medial side of the sole, with sparing of the heel because of its calcaneal supply. Tenderness behind the medial malleolus may be seen. Tinel sign over the tarsal tunnel is positive.

Tarsal tunnel syndrome should be differentiated from other neuropathic processes like S1 radiculopathy, small fiber peripheral neuropathy, as well as arthritis, plantar fasciitis and vascular disease. The most sensitive diagnostic test is the slowing of sensory conduction of the nerve in the tarsal tunnel, but this is rarely seen. Absence of medial and lateral plantar sensory nerve action potentials is not specific for this condition as it may also be a finding of sensory polyneuropathies. Mimicking conditions are excluded by nerve conduction studies (78). Imaging studies help exclude structural or space occupying lesions.

Activity and biomechanical modification, together with or without physical therapy measures consisting of pain relieving electrical currents, whirlpool, non-steroidal anti-inflammatory and neuropathic pain medication are the first choices of treatment in patients without a space occupying lesion. If conservative measures fail, surgical decompression of the tarsal tunnel is performed.

## Conclusion

Entrapment neuropathies of the lower extremities may lead to difficulties in activities of daily living because of significant interference on mobility. An understanding of the pathways of lower extremity nerves and the areas of vulnerability for entrapment or trauma is needed to identify the cause of the injury. Electrodiagnostic tests and imaging studies not only confirm and localize the site of the injury, its severity and/or chronicity, they also help exclude other causes of the clinical condition. Treatment is planned according to the etiology of the condition. Unless the cause is a space occupying lesion, physical therapy and rehabilitation measures together with pain medication are the first treatment choice in most of the cases. Surgical decompression is indicated in resistant symptoms.

## References


1. Harney D, Patijn J. Meralgia paresthetica: diagnosis and management strategies. Pain Med 2007;8:669–77. doi: 10.1111/j.1526-4637.2006.00227.x
2. Grossman MG, Ducey SA, Nadler SS, Levy AS. Meralgia paresthetica: diagnosis and treatment. J Am Acad Orthop Surg 2001;9:336–44.
3. Boyce JR. Meralgia paresthetica and tight trousers. JAMA 1984;251:1553.
4. Moucharafieh R, Wehbe J, Maalouf G. Meralgia paresthetica: a result of tight new trendy low cut trousers ('taille basse'). Int J Surg 2008;6:164–8. doi: 10.1016/j.ijsu.2007.04.003
5. Shapiro BE, Preston DC. Entrapment and compressive neuropathies. Med Clin North Am 2009;93:285–315. doi: 10.1016/j.mcna.2008.09.009
6. Yamout B, Tayyim A, Farhat W. Meralgia paresthetica as a complication of laparoscopic cholecystectomy. Clin Neurol Neurosurg 1994;96:143–4.
7. Hutchins FL Jr, Huggins J, Delaney ML. Laparoscopic myomectomy –an unusual cause of meralgia paresthetica. J Am Assoc Gynecol Laparosc 1998;5:309–11.
8. Antunes PE, Antunes MJ. Meralgia paresthetica after aortic valve surgery. J Heart Valve Dis 1997;6:589–90.
9. Parsonnet V, Karasakalides A, Gielchinsky I, Hochberg M, Hussain SM. Meralgia paresthtetica after coronary bypass surgery. J Thorac Cardiovasc Surg 1991;101:219–21.
10. Abarbanel JM, Berginer VM, Osimani A, Solomom H, Charuzi I. Neurologic complications after gastric restriction surgery for morbid obesity. Neurology 1987;37:196–200.
11. Grace DM. Meralgia paresthetica after gastroplasty for morbid obesity. Can J Surg 1987;30:64–5.
12. Macgregor AM, Thoburn EK. Meralgia paresthetica following bariatric surgery. Obes Surg 1999;9:364–8. doi: 10.1381/096089299765552945
13. Seid AS, Amos E. Entrapment neuropathy in laparoscopic herniorraphy. Surg Endosc 1994;8:1050–3.
14. Sampath P, Yeo CJ, Capmbell JN. Nerve injury associated with laparoscopic inguinal herniorraphy. Surgery 1995;118:829–33.
15. Aszmann OC, Dellon ES, Dellon AL. Anatomical course of the lateral femoral cutaneous nerve and its susceptibility to compression and injury. Plast Reconstr Surg 1997;100:600–4.
16. Williams PH, Trzil KP. Management of meralgia paresthetica. J Neurosurg 1991;74:76–80. doi: 10.3171/jns.1991.74.1.0076
17. Dibenedetto LM, Lei Q, Gilroy AM, Hermey DC, Marks SC Jr, Page DW. Variations in the inferior pelvic pathway of the lateral femoral cutaneous nerve: implications for laparoscopic hernia repair. Clin Anat 1996;9:232–6. doi: 10.1002/(SICI)1098-2353(1996)9:4<232::AID-CA3>3.0.CO;2-G
18. Sürücü HS, Tanyeli E, Sargon MF, Karahan ST. An anatomic study of the lateral femoral cutaneous nerve. Surg Radiol Anat 1997;19:307–10.
19. Dias Filho LC, Valença MM, Guimarães Filho FA, Medeiros RC, Silva RA, Morais MG, et al. Lateral femoral cutaneous neuralgia: an anatomical insight. Clin Anat 2003;16:309–16. doi: 10.1002/ca.10106
20. Grothaus MC, Holt M, Mekhail AO, Ebraheim NA, Yeasting RA. Lateral femoral cutaneous nerve: an anatomic study. Clin Orthop Relat Res 2005;(437):164–8.
21. Ropars M, Morandi X, Huten D, Thomazeau H, Berton E, Darnault P. Anatomical study of the lateral femoral cutaneous nerve with special reference to minimally invasive anterior approach for total hip replacement. Surg Radiol Anat 2009;31:199–204. doi: 10.1007/s00276-008-0433-3
22. Massey EW. Sensory mononeuropathies. Semin Neurol 1998;18:177–83. doi: 10.1055/s-2008-1040871
23. Puig L, Alegre M, de Moragas JM. Treatment of meralgia paraesthetica with topical capsaicin. Dermatology 1995;191:73–4.
24. Hurdle MF, Weingarten TN, Crisostomo RA, Psimos C, Smith J. Ultrasound-guided blockade of the lateral femoral cutaneous nerve: technical description and review of 10 cases. Arch Phys Med Rehabil 2007;88:1362–4. doi: 10.1016/j.apmr.2007.07.013
25. Kim DH, Murovic JA, Tiel RL, Kline DG. Intrapelvic and thigh level femoral nerve lesions: management and outcomes in 119 surgically treated cases. J Neurosurg 2004;100:989–96. doi: 10.3171/jns.2004.100.6.0989
26. Lange B, Langer C, Markus PM, Becker H. Paralysis of the femoral nerve following totally extraperitoneal laparoscopic inguinal hernia repair. Surg Endosc 2003;17:1157. doi: 10.1007/s00464-002-4270-x
27. Naroji S, Belin LJ, Maltenfort MG, Vaccaro AR, Schwartz D, Harrop JS, et al. Vulnerability of the femoral nerve during complex anterior and posterior spinal surgery. J Spinal Cord Med 2009;32:432–5.
28. Papastefanou SL, Stevens K, Mullholland RC. Femoral nerve palsy. An unusual complication of anterior lumbar interbody fusion. Spine (Phila Pa 1976) 1994;19:2842–4.
29. Boontje AH, Haaxma R. Femoral neuropathy as a complication of aortic surgery. J Cardiovasc Surg (Torino) 1987;28:286–9.

30. Gruson KI, Moed BR. Injury of the femoral nerve associated with acetabular fracture. J Bone Joint Surg Am 2003;85-A:428–31.
31. Laurent LE. Femoral nerve compression syndrome with paresis of the quadriceps muscle caused by radiotherapy for malignant tumors. A report of four cases. Acta Orthop Scand 1975;46:804–8.
32. Baxi A, Kaushal M, Kadi P, Baxi DA. Femoral Neuropathy: A curse of vaginal hysterectomy. Journal of Gynecologic Surgery 2010;26:171–4. doi: 10.1089=gyn.2009.0056
33. Bohrer JC, Walters MD, Park A, Polston D, Barber MD. Pelvic nerve injury following gynecologic surgery: A prospective cohort study. Am J Obstet Gynocol 2009;201:531–7. doi: 10.1016/j.ajog.2009.07.023
34. Tosun A, İnal E, Keleş I, Tulmaç M, Tosun Ö, Aydın G, et al. Conservative treatment of femoral neuropathy following retroperitoneal hemorrhage: a case report and review of the literature. Blood Coagul Fibrinolysis 2014;25:769–72. doi: 10.1097/MBC.0000000000000110
35. Ducic I, Delton L, Larson EE. Treatment concepts for idiopathic and iatrogenic femoral nerve mononeuropathy. Ann Plast Surg 2005;55:397–401.
36. Mingo-Robinet J, Castaneda-Cabrero C, Alvares V, Alonso-Cortes JML, Monge-Casres E. Tourniquet-related iatrogenic femoral nerve palsy after knee surgery: Case report and review of the literature. Case Reports in Orthopedics 2013;1–4. doi: 10.1155/2013/368290
37. Narouze ZN, Zakari A, Vydyanathan A. Ultrasound-guided placement of permanent percutaneous femoral nerve stimulator leads for the treatment of intractable femoral neuropathy. Pain Physician 2009;12:E305–8.
38. Garvin KL, Backstein D, Pellegrini VD Jr, Kim RH, Lewallen DG. Dealing with complications. J Bone Joint Surg Am 2009;91 Suppl 5:18–21. doi: 10.2106/JBJS.I.00347
39. Parmer SS, Carpenter JP, Fairman RM, Velazquez OC, Mitchell ME. Femoral neuropathy following retroperitoneal hemorrhage: case series and review of the literature. Ann Vasc Surg 2006;20:536–40. doi: 10.1007/s10016-006-9059-2
40. Mochida H, Kikuchi S. Injury to infrapatellar branch of saphenous nerve in arthroscopic knee surgery. Clin Orthop Relat Res 1995;(320):88–94.
41. Tennent TD, Birch NC, Holmes MJ, Birch R, Goddard NJ. Knee pain and the infrapatellar branch of the saphenous nerve. J R Soc Med 1998;91:573–5.
42. Bertram C, Porsch M, Hackenbroch MH, Terhaag D. Saphenous neuralgia after arthroscopically assisted anterior cruciate ligament reconstruction with a semitendinosus and gracilis tendon graft. Arthroscopy 2000;16:763–6. doi: 10.1053/jars.2000.4820
43. Lippitt AB. Neuropathy of the saphenous nerve as a cause of knee pain. Bull Hosp Jt Dis 1993;52:31–3.
44. Schwartzman RJ, Maleki J. Postinjury neuropathic pain syndromes. Med Clin North Am 1999;83:597–626.
45. Pendergrass PT, Moore JH. Saphenous neuropathy following medial knee trauma. J Orthop Sports Phys Ther 2004;34:328–34. doi: 10.2519/jospt.2004.1269
46. Busis NA. Femoral and obturator neuropathies. Neurol Clin 1999;17:633–53.
47. Bradshaw C, McCrory P, Bell S, Brukner P. Obturator neuropathy: a cause of chronic groin pain in athletes. Am J Sports Med 1997;25:402–8. doi: 10.1177/036354659702500322
48. Wagner MA, Warner DO, Harper CM, Schroeder DR, Maxson PM. Lower extremity neuropathies associated with lithotomy positions. Anesthesiology 2000;93:938–42.
49. Pavlov H, Nelson TL, Warren RF, Torg JS, Burstein AH. Stress fractures of the pubic ramus. A report of twelve cases. J Bone Joint Surg Am 1982;64:1020–5.
50. Schmalzried TP, Amstutz HC, Dorey FJ. Nerve palsy associated with total hip replacement. Risk factors and prognosis. J Bone Joint Surg Am 1991;73:1074–80.
51. Finan MA, Fiorica JV, Hoffman MS, Barton DP, Gleeson N, Roberts WS, et al. Massive pelvic hemorrhage during gynecologic cancer surgery: "pack and go back". Gynecol Oncol 1996;62:390–5. doi: 10.1006/gyno.1996.0254
52. Bischoff C, Schönle PW. Obturator nerve injuries during intra-abdominal surgery. Clin Neurol Neurosurg 1991;93:73–6.
53. Brown GD, Swanson EA, Nercessian OA. Neurologic injuries after total hip arthroplasty. Am J Orthop (Belle Mead NJ) 2008;37:191–7.
54. Feinberg J, Sethi S. Sciatic neuropathy: case report and discussion of the literature on postoperative sciatic neuropathy and sciatic nerve tumors. HSS J 2006;2:181–7. doi: 10.1007/s11420-006-9018-z
55. Farrell C, Springer BD, Haidukewych GJ, Morrey BF. Motor nerve palsy following primary total hip arthroplasty. J Bone Joint Surg Am 2005;87:2619–25. doi: 10.2106/JBJS.C.01564
56. Sosna A, Pokorny D, Jahoda D. Sciatic nerve palsy after total hip replacement. J Bone Joint Surg Br 2005;87:1140–1. doi: 10.1302/0301-620X.87B8.16550
57. Bendszus M, Rieckmann P, Perez J, Koltzenburg M, Reiners K, Solymosi L. Painful vascular compression syndrome of the sciatic nerve caused by gluteal varicosities. Neurology 2003;61:985–7.
58. Hu MH, Wu KW, Jian YM, Wang CT, Yang SH. Vascular compression syndrome of sciatic nerve caused by gluteal varicosities. Ann Vasc Surg 2010;24:1134.e1–4. doi: 10.1016/j.avsg.2010.02.043
59. Papadopoulos EC, Khan SN. Piriformis syndrome and low back pain: a new classification and review of the literature. Orthop Clin North Am 2004;35:65–71. doi: 10.1016/S0030-5898(03)00105-6
60. Lohrer H, Nauck T, Konerding MA. Nerve entrapment after hamstring injury. Clin J Sport Med 2012;22:443–5. doi: 10.1097/JSM.0b013e318257d76c
61. Goh KH, Tan CB, Tjia HT. Sciatic neuropathies –a retrospective review of electrodiagnostic features in 29 patients. Ann Acad Med Singapore 1996;25:566–9.
62. Yuen EC, Olney RK, So YT. Sciatic neuropathy: clinical and prognostic features in 73 patients. Neurology 1994;4:1669–74.
63. Childers MK, Wilson DJ, Gnatz SM, Conway RR, Sherman AK. Botulinum toxin type A use in piriformis muscle syndrome: a pilot study. Am J Phys Med Rehabil 2002;81:751–9. doi: 10.1097/01.PHM.0000027426.98000.57
64. Fishman LM, Anderson C, Rosner B. BOTOX and physical therapy in the treatment of piriformis syndrome. Am J Phys Med Rehabil 2002;81:936–42. doi: 10.1097/01.PHM.0000034956.35609.5E
65. Vasudevan JM, Freedman MK, Beredjiklian PK, DeLuca PF, Nazarian LN. Common peroneal entrapment neuropathy secondary to a popliteal lipoma: ultrasound superior to magnetic resonance imaging for diagnosis. PM R 2011;3:274–9. doi: 10.1016/j.pmrj.2010.09.014
66. Kernohan J, Levack B, Wilson JN. Entrapment of the superficial peroneal nerve. Three case reports. J Bone Joint Surg Br 1985;67:60–1.
67. Jang SH, Lee H, Han SH. Common peroneal nerve compression by a popliteal venous aneurysm. Am J Phys Med Rehabil 2009;88:947–50. doi: 10.1097/PHM.0b013e3181b331d8
68. Farjoodi P, Johnson TS. Proximal tibiofibular ganglion cyst causing peroneal neuropraxia. Curr Orthop Pract 2010;21:209–12.
69. Hsu YC, Shih YY, Gao HW, Huang GS. Subcutaneous lipoma compressing the common peroneal nerve and causing palsy: sonographic diagnosis. J Clin Ultrasound 2009;38:97–9. doi: 10.1002/jcu.20643
70. Bozkurt A, Grieb G, O'Dey Dm, Fuchs P, Waberski T, Krombach G, et al. Common peroneal nerve compression and heterotopic ossification resulting from severe burn injury: a case report. J Bone Joint Surg Am 2010;92:978–83. doi: 10.2106/JBJS.I.00187
71. Liu Z, Zhou J, Zhao L. Anterior tarsal tunnel syndrome. J Bone Joint Surg Br 1991;73:470–3.
72. Kashani SR, Moon AH, Gaunt WD. Tibial nerve entrapment by a Baker cyst: case report. Arch Phys Med Rehabil 1985;66:49–51.
73. Iida T, Kobayashi M. Tibial nerve entrapment at the tendinous arch of the soleus: a case report. Clin Orthop Relat Res 1997;(334):265–9.
74. Singh G, Kumar VP. Neuroanatomical basis for the tarsal tunnel syndrome. Foot Ankle Int 2012;33:513–8. doi: 10.3113/FAI.2012.0513
75. Takakura Y, Kitada C, Sugimoto K, Tanaka Y, Tamai S. Tarsal tunnel syndrome. Causes and results of operative treatment. J Bone Joint Surg Br 1991;73:125–8.
76. Frey C, Kerr R. Magnetic resonance imaging and the evaluation of tarsal tunnel syndrome. Foot Ankle 1993;14:159–64.
77. Baba H, Wada M, Annen S, Azuchi M, Imura S, Tomita K. The tarsal tunnel syndrome: evaluation of surgical results using multivariate analysis. Int Orthop 1997;21:67–71. doi: 10.1007/s002640050122
78. Toussaint CP, Perry EC 3rd, Pisansky MT, Anderson DE. What's new in the diagnosis and treatment of peripheral nerve entrapment neuropathies. Neurol Clin 2010;28:979–1004. doi: 10.1016/j.ncl.2010.03.017