

# Entrapment Neuropathies of The Lower Extremities

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## 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 ekstremitte periferik sinirleri, traseleri boyunca anatomik olarak duyarlı buldukları bölgelerde sıkışabilir. Siniri direkt olarak etkileyen herhangi bir travma veya sinir çevresindeki bir inflamatuvar 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 parestezi; 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 ekstremitte, tuzak

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**P**atients 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. Laparoscopic 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 herniorrhaphy (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 persistent, 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-thigh.

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.

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