FEMORAL AND OBTURATOR NEUROPATHIES

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Femoral and obturator neuropathies are relatively uncommon clinical syndromes with characteristic clinical features. This article is an update of excellent discussions of the causes, diagnosis, and treatment of these disorders that have appeared in books devoted to entrapment neuropathies published over the last several years.[34,111,153]

FEMORAL NEUROPATHIES

Femoral neuropathies can be divided into syndromes in which both motor and sensory involvement is commonly present and into purely sensory syndromes that involve the saphenous nerve, which is the distal sensory continuation of the femoral nerve. These two sets of disorders will be discussed separately.

Anatomy

The femoral nerve originates from the posterior divisions of the ventral rami of the L2, L3, and L4 spinal nerves in the lumbar plexus within the psoas major muscle. After the nerve emerges from the lateral border of the psoas it lies in the groove between the psoas and the iliacus muscles. The femoral nerve approaches the external iliac artery, which is antero-

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medial, as the two structures descend to leave the pelvis, and gives off some twigs to innervate the iliacus and the psoas muscles (the psoas is also innervated by branches derived from L2 and L3 spinal nerves in the lumbar plexus[111,153]). The femoral nerve, the psoas and the iliacus muscles, and the iliolumbar vessels occupy a tight compartment bounded by the iliac fascia. The femoral nerve subsequently passes under the inguinal ligament, where it gives off a branch to the pectineus muscle, and enters the femoral triangle lateral to and separated from the femoral artery by some psoas fibers. Approximately 4 cm distal to the ligament, the nerve divides into anterior and posterior divisions. The anterior division innervates the sartorius muscle and forms the medial and intermediate femoral cutaneous nerves innervating the anterior surface of the thigh as far as the knee. The posterior division divides into the saphenous nerve, which innervates the skin of the medial and the anterior surfaces of the knee and the medial surface of the lower leg, medial malleolus, and a small portion of the medial arch of the foot and great toe; and motor branches to the quadriceps (rectus femoris, vastus lateralis, vastus intermedius, and vastus medialis).[34,153,156]

Authorities differ on whether the clinically significant innervation of the iliacus and the psoas muscles derives from the beginning of the femoral nerve[34,111,156] or from the fibers arising proximal to the origin of this nerve.[153] Depending on the viewpoint, therefore, weakness of hip flexion caused by dysfunction of these muscles is included in or excluded from the clinical spectrum of femoral neuropathy.

Clinical Presentation

Symptoms

Patients complain that the ipsilateral knee is weak or that it buckles on walking, causing frequent falls. Thigh atrophy can be noted. Numbness and paresthesias may be noted in the anterior thigh or the medial calf, medial foot, and great toe. Sensory symptoms may be mild or absent. If present, pain can be felt in the iliac fossa, inguinal region, anterior thigh, or medial calf.[34,153]

Signs

A severe femoral nerve lesion produces wasting and weakness of the quadriceps muscles, absence of the knee reflex, and sensory impairment over the anterior thigh and the medial calf, the medial foot, and the great toe. On even ground the patient may be able to walk by keeping the knee hyperextended, but the patient cannot use the involved leg to step up when climbing stairs; the involved leg has to lead when descending stairs.

In partial lesions of the femoral nerve, various combinations of motor and sensory loss in part or all of the femoral distribution can be seen. A diminished or absent knee jerk is an objective clinical sign consistent with, but not diagnostic of, femoral neuropathy.[34,153]

In patients who have suspected femoral neuropathy, the iliopsoas muscle and the hip adductors must be examined carefully. Weakness of the iliopsoas indicates involvement of the origin of the femoral nerve (according to some authorities, see above), lumbar plexus, or the L2 or L3 nerve roots. Weakness of the hip adductors, innervated by L2-L4, lumbar plexus, and obturator nerve, indicates a lumbar radiculopathy or plexopathy or lesions of both femoral and obturator nerves.[34,153]

An intact iliopsoas muscle masks any hip flexion weakness caused by rectus femoris and sartorius dysfunction. Similarly, any knee flexion weakness caused by sartorius involvement is obscured by strong knee flexors innervated by other nerves. Other hip adductors compensate for pectineus weakness.

A mass, which may be tender, in the inguinal area suggests retroperitoneal hematoma or abscess formation. Sometimes the leg is held flexed at the hip. Ecchymosis in the inguinal area, around the flank or in the upper thigh, is a sign of retroperitoneal hematoma. Anemia and shock can accompany massive retroperitoneal hematomas.[25]

Electrophysiology

Electrodiagnostic studies are an important means of diagnosing femoral neuropathies, following them over time, and establishing a prognosis.[34,78,85] Needle electromyography (EMG) of the quadriceps muscles can demonstrate lower motor neuron pathology in a femoral nerve distribution. Needle EMG of lumbar paraspinal, hip adductor, and other lower extremity muscles can establish whether there is involvement outside of the femoral nerve distribution, which suggests pathology of lumbar roots, plexus, or multiple nerves. EMG abnormalities in the iliopsoas muscle indicate involvement of the origin of the femoral nerve (according to some authorities, see above), lumbar plexus, or the L2 or L3 nerve roots.

Femoral motor-nerve-conduction studies, carried out by surface or needle stimulation techniques,[27,48] can establish side-to-side latency and amplitude differences, and, in some cases, can demonstrate slowing of femoral nerve conduction across the inguinal ligament.[34] These tests are generally considered to be less useful than needle EMG studies; however, because femoral motor nerve conduction studies can estimate axon loss in the involved leg—the only prognostic factor in recovery from femoral neuropathy[85] (see below)—these studies may be more useful than previously thought.

Saphenous nerve conduction studies, although technically challenging, are useful for confirming damage to those sensory fascicles of the femoral nerve and for localizing the site of femoral nerve distribution lesions.[40,155,166] Lesions proximal to the dorsal root ganglia, such as L4 radiculopathies, have normal saphenous sensory nerve action potential amplitudes. Lesions at or distal to the dorsal root ganglia, such as lumbar plexopathies or femoral neuropathies, classically have reduced saphenous sensory nerve action potential amplitudes.[78] The recently described tech-

nique of medial femoral cutaneous nerve conduction may also be useful in lesion localization.[89] Saphenous somatosensory-evoked potential studies can document dysfunction of these nerve fibers but cannot precisely localize femoral nerve lesions.[158,159,164]

Other Diagnostic Tests

Abdominal and pelvic computerized tomographic (CT) scanning, ultrasound, and magnetic resonance (MR) imaging are excellent means of detecting masses, such as retroperitoneal hematomas and tumors, which can damage the femoral nerve.[5,25,41,63,90,99,129,147,152] Lumbar spine imaging studies (CT scanning, MR imaging, and myelography) may be necessary to rule out lumbar radiculopathy. Investigations for diabetes mellitus (fasting blood glucose, glucose tolerance test) and vasculitic disorders (anti-nuclear antibody [ANA], sedimentation rate, and so forth) should be undertaken in patients in whom there is no obvious trauma or mass involving the femoral nerve.

Differential Diagnosis

Differential diagnostic possibilities of knee extensor weakness theoretically include upper motor neuron disorders in the brain and the spinal cord[115]; lumbar radiculopathies; lumbar plexopathies; neuromuscular junction pathology; and quadriceps muscle, hip, or knee disorders.[34,153] Sensory involvement rules out primary disorders of neuromuscular junction, muscle, or joints. The presence of a Babinski sign indicates an upper motor neuron lesion.

The motor, sensory, and reflex findings in L4 radiculopathy, lumbar plexopathy, and femoral neuropathy are superficially similar. However, L4 radiculopathy has a different distribution of sensory loss (it involves the medial calf but not the anterior thigh) and can be associated with some weakness or denervation on EMG of hip adductors, gluteus medius, and tibialis anterior and tibialis posterior muscles.[34,153] Lumbar plexopathy can be associated with hip flexor and adductor weakness in addition to knee extensor weakness and with sensory loss in the femoral, obturator, and lateral femoral cutaneous nerve distributions.[153]

The symptoms, signs, and diagnostic test results should enable the clinician to sort through these possibilities by determining the presence or absence of sensory, motor, or reflex dysfunction outside of the femoral nerve distribution.

Origin

Iatrogenic

Many of the causes of femoral neuropathy are iatrogenic. The femoral nerve is vulnerable to injury during surgical procedures involving the

abdomen, pelvis, inguinal area, and hip.[168] These injuries are often secondary to nerve compression by self-retaining retractors.[66,165] The lateral blade of the self-retaining retractor can either compress or impinge upon the intrapelvic portion of the femoral nerve.[16] When retracting in the deep pelvis, consideration should be given to using small, well-padded retractor blades and to repositioning these blades regularly.[36]

After hip arthroplasties the incidence of femoral neuropathy ranges from 0.1% to 2.3%.[57,137,148,172] The mechanism of injury can be pressure from retractors; thermal injury; or entrapment by bone cement, laceration, iliacus hematoma complicating postoperative anticoagulation, or postoperative scar formation.[69,120,168,172] In a series of 2713 hip arthroplasties, 15 cases of postoperative femoral neuropathy were identified. The risk was significantly higher in revisions—especially when the acetabular component was exchanged—than in primary arthroplasties.[116] A postoperative femoral neuropathy was diagnosed in 8 patients in a prospective series of 1000 consecutive total hip arthroplasty patients for an overall prevalence of 0.8%. The overall prevalence of nerve palsy with the posterior approach was 0.6% and was 1.0% with the lateral transtrochanteric approach. There were no statistical differences between the two approaches in both primary or revision surgeries. It is the anatomic variations and complexity of the reconstruction that are associated with nerve injury and not the surgical approach per se.[114]

Two prospective studies found incidences of 7.45% and 11.6% of femoral neuropathy after abdominal hysterectomy.[53,86] Avoiding self-retaining retractors reduced the incidence of femoral neuropathy by 93%.[53] Microsurgical fallopian tuboplasty can also be complicated by femoral neuropathy.[62,161]

Obstetric and gynecologic procedures performed in the lithotomy position, such as vaginal hysterectomies or prolonged labor, have resulted in unilateral or bilateral femoral neuropathies. The mechanism is presumably microvascular or a local mechanical injury of the femoral nerve, which is compressed beneath the inguinal ligament in a sustained posture with the hip joint in extreme abduction and external rotation.[33,51,54,65,66,68,74,92,149,162] Even uncomplicated deliveries and minor pelvic procedures, such as laparoscopy, are associated with femoral neuropathies.[4,121]

Renal transplantation, during which the donor kidney is implanted retroperitoneally in the iliac fossa, is associated with femoral nerve injury.[66,101,125,150,163,168] Nerve compression by retractors or a hematoma can cause the neuropathy.[125,150] In other cases, nerve ischemia, possibly caused by a steal phenomenon, after the anastomosis of the graft renal artery to the internal iliac artery, may contribute to femoral nerve injury.[71] Recovery of the nerve is usual.

Urological procedures have been linked with femoral neuropathy. Such procedures include radical cystectomy,[61] transurethral resection of the bladder with exploration and biopsy of a tumor mass,[23] percutaneous nephrolithotomy of a pelvic kidney,[106] radical cystoprostatectomy and continent urinary diversion,[18] and psoas hitch vesicopexy.[83]

Femoral neuropathy may complicate inguinal or femoral herniorrhaphy, including laparoscopic hernia repairs.[79,96,111,124,128,139,156] The nerve may be cut, or trapped by a suture or staples used to affix prosthetic mesh[77,138,140] or by scar tissue.[80]

Femoral artery surgery, femoral arterial puncture and catheterization, balloon angioplasty, and inguinal lymph node resection may be complicated by femoral neuropathy.[8,9,34,144] One percent of 100 consecutive patients who required the placement of an intra-aortic balloon pump (IABP) developed femoral neuropathy.[93] Femoral neuropathy has been reported after vascular access cannulation for hemodialysis[73] and infusions of chemotherapeutic agents into the femoral artery.[21] The neuropathy may follow external pressure to the femoral artery from a clamping device applied after femoral artery catheterization.[98] Cardiac surgery patients may develop asymptomatic ischemia of the femoral nerve or quadriceps muscles if they have compromised femoral artery blood flow and another cause of tissue hypoxia.[100]

Maintaining the hip in extension during the course of anterior fusion of a previously failed posterior fusion was another causative factor of femoral nerve injury.[117] Percutaneous vertebroplasty[29] and the free vascularized iliac crest tissue transfer[45] have been associated with femoral neuropathy.

Nonoperative Trauma

Femoral neuropathy may result from injuries caused by bullet or stab wounds in the groin, by blunt trauma, and by fractures of the hip or pelvis. Because the femoral nerve divides into several motor and sensory branches after passing underneath the inguinal ligament, severe femoral nerve injuries are more likely to result from lesions proximal to this ligament rather than in the thigh itself.[34] Gymnasts or dancers have developed acute femoral nerve injuries.[17,59,105,133] Stretching of the nerve from prolonged hyperextension of the leg or compression of the nerve under the inguinal ligament during hip flexion[95,131,145] may cause the neuropathy, and iliacus compartment hematomas may play a role in some situations.

Hematoma

Femoral neuropathy can be caused by hematoma formation in the iliacus compartment beneath the iliacus fascia.* Iliacus hematomas are a complication of anticoagulant therapy, hemophilia and other coagulopathies, and traumatic avulsion of the iliacus muscle.[50,59] They sometimes occur spontaneously.[52,94] Psoas muscle hematomas usually produce lumbar plexopathy, but occasionally, only the femoral nerve is affected.[99]

Spontaneous iliacus or iliopsoas hematoma can cause femoral neuropathy in patients who have unstable coronary syndromes and who are

*3, 11, 15, 25, 26, 31, 52, 55, 63, 84, 94, 122, 126, 129, 147, 151, 174

receiving intravenous heparin in therapeutic doses.[134] In a large retrospective review of femoral artery catheterizations, retroperitoneal hematoma developed in 0.5%, with the highest frequency after coronary artery stenting (3%).[76] Femoral neuropathy occurred in approximately 36% of these patients or approximately 0.2% of all femoral artery catheterizations.[20,75,76,107,169] Catheter-directed urokinase thrombolysis can result in retroperitoneal hematoma and femoral neuropathy.[136]

Infection

Iliacus pyomyositis has presented with femoral neuropathy.[24] Abscesses may develop primarily or from secondary infection of an iliacus hematoma.[3,109]

Other Mass Lesions

Primary tumors of the iliopsoas muscle, the ilium,[142,152] or of the nerve itself[5,39] may cause femoral neuropathy. Metastatic tumors can also involve the femoral nerve. For example, six patients who had recurrent appendix, colon, or rectal cancer presented with femoral neuropathy.[49] Aneurysms of the internal iliac artery[67,97] and chronic contained rupture of an abdominal aortic aneurysm causing femoral neuropathy have been reported.[7,35,127] Four patients who developed a profunda femoral artery pseudoaneurysm after cardiac catheterization or percutaneous transluminal coronary angioplasty had femoral neuropathy.[70]

Diabetes Mellitus

Diabetic radiculopathy or plexopathy, often superficially resembles femoral neuropathy.[19,28,46,56] However, in most cases, involvement of spinal roots, lumbosacral plexus, or other peripheral nerves is also found on careful clinical or electrodiagnostic evaluation.

Other Causes

There are a variety of other reported causes of femoral neuropathy. These include mononeuropathy multiplex syndromes,[6] inguinal irradiation,[87] and pigmented villonodular synovitis of the hip.[1] Femoral neuropathy has been associated with pregnancy[81] and endometriosis.[175] One patient had femoral neuropathy that was associated with a recent Epstein–Barr virus (EBV) infection.[143]

Idiopathic

There were idiopathic femoral neuropathies reported in the series of Biemond.[10] Many of these patients were elderly and had the sudden onset of femoral neuropathy that was painful, followed by some recovery. Stew-

art[153] and Dawson et al[34] have suggested that some of these patients may have actually had diabetic lumbar plexopathy or femoral neuropathy.

Practical Management

Regardless of the cause, patients who have femoral neuropathies may require pain management, including analgesics, tricyclic antidepressants (such as amitriptyline), or anticonvulsants (such as phenytoin, carbamazepine, or gabapentin). Nerve blocks may be useful for pain control. Physical therapy is indicated to increase strength and to maintain mobility and ambulation.[60] Bracing or assistive devices such as crutches may be needed.[34]

Exploration of the femoral nerve should be undertaken if complete disruption or inadvertent stapling or suturing of the nerve is suspected. Usually, however, the patient is observed for at least several weeks to see if spontaneous recovery takes place. If recovery does not occur, surgical exploration should be considered if the deficit is significant.[153]

The management of retroperitoneal hematomas should be individualized. Anticoagulation must be reversed with vitamin K, fresh frozen plasma, or both. Fluid and blood replacement are sometimes necessary. Supportive care is often associated with satisfactory recovery.[26,55,174] In some cases, however, prompt surgical evacuation of the hematoma is indicated.[15,26] Percutaneous drainage of the hematoma may be successful.[103] Drainage is the best treatment for an iliacus abscess.[3,109]

Prognosis

In iatrogenic femoral neuropathies, recovery is generally said to be fairly common, occurring within a few weeks to a few months.[4,20,25,53,68,153] However, this assessment may be too optimistic in some situations.[85,116] The degree of recovery is reported to be better when induced by the lithotomy position[4] than when associated with hip surgery or inguinal procedures.[69,137,168]

Kuntzer et al[85] studied the clinical and the prognostic features of 31 patients who had femoral neuropathy. A six-point clinical grading scale was set up based on the Medical Research Council (MRC) rating of strength and functional disability. Ten patients (31%) had excellent recovery (complete), 11 (34%) had satisfactory recovery (moderate improvement—two or more grades), and 10 (31%) had poor recovery (improvement by fewer than two grades) over a mean follow-up period of 37.2 months. Regardless of the cause of femoral neuropathy, functional improvement was seen in 2 out of 3 patients within 2 years. No further recovery was noted after 2 years.

The percentage of axon loss derived by comparison of vastus medialis compound muscle action potential amplitude on the affected and the unaffected sides after stimulation of the femoral nerves was the only signifi-

cant variable. The best prognosis was seen in patients who had an estimated axon loss of less than or equal to 50%, with all patients fulfilling this criterion showing improvement within 1 year. Fewer than 50% of the patients who had axon loss greater than 50% showed improvement. Patients who had greater degrees of axon loss took longer to improve. A model of estimated improvement showed that for axon loss not exceeding 50%, nearly 100% of patients may expect to achieve satisfactory or excellent outcomes, but with higher degrees of axon loss, the success rate drops drastically. One plausible interpretation of these data is that patients who have lesions consisting of a conduction block (neurapraxia) proximal to the point of femoral nerve stimulation did better than patients who had axonal injury (axonotmesis or neurotmesis).[34,111,153,156]

SAPHENOUS NEUROPATHIES

Anatomy

The distal sensory continuation of the femoral nerve is the saphenous nerve. It descends within the quadriceps muscles in the subsartorial (Hunter's) canal lateral to the femoral artery, gives off an infrapatellar branch that innervates the skin over the anterior surface of the patella, then pierces a fascial layer between the sartorius and the gracilis muscles to emerge from the canal and become subcutaneous approximately 10 cm proximal to the knee. The nerve crosses the pes anserine bursa at the upper medial end of the tibia and descends along its medial aspect. The saphenous nerve and vein are close to one another along most of their courses in the medial calf and are often closely bound together, especially in the distal third of the leg. At the lower third of the leg, the saphenous nerve divides into two main branches. One continues along the medial border of the tibia to reach and end at the ankle. The other passes anteriorly with the vein across the medial surface of the tibia and in front of the medial malleolus to reach the foot along the medial side of which it continues to the ball of the great toe. The saphenous nerve innervates the skin of the medial and the anterior surfaces of the knee and the medial surface of the lower leg, medial malleolus, and a small portion of the medial arch of the foot and the great toe.[14,153,156]

Clinical Presentation

Symptoms

Patients who have saphenous neuropathy complain of sensory loss or paresthesias in the medial calf of varying degrees of severity that can extend to the medial foot and the great toe.[156] Radiating pain medially or distally to the knee or in the medial calf, the medial foot, and the great toe may also be present.[14,111,153]

Signs

Sensory loss in the medial calf, the medial foot, and the great toe may be present. A small area of sensory loss just below the knee is noted in injury to the infrapatellar branch of the saphenous nerve. The cause may be evident on inspection, for example, a surgical scar along the course of the nerve from harvesting the saphenous vein during cardiac bypass surgery. The site of injury may be marked by a tender neuroma or by Tinel's sign.[14,111,153]

Electrophysiology

Saphenous nerve conduction studies can be used to investigate lesions of this nerve.[40,155,166] Because the saphenous nerve is difficult to study, it is often more useful to compare the saphenous sensory nerve latency and amplitude of the involved leg with those of the contralateral leg rather than with published normal values. A low saphenous sensory nerve action potential amplitude (less than 50% of the uninvolved side) is strong evidence that the lesion is at or distal to the dorsal root ganglia.[78] Saphenous nerve somatosensory-evoked potential studies have been described[158,159,164] but are less useful clinically. Needle electromyographic studies of lumbar paraspinal muscles and proximal lower extremity muscles, including iliopsoas, quadriceps, hip adductor, and tibialis anterior muscles, are indicated when a lumbar radiculopathy or plexopathy or a partial femoral nerve lesion is in the differential diagnosis.[78]

Other Diagnostic Tests

CT scanning, MR imaging, and ultrasound examinations are useful when lesions involving the lumbar roots or the lumbar plexus need to be ruled out. Possible intraneural masses can be investigated by MR imaging with gadolinium enhancement.[153]

Differential Diagnosis

The main differential diagnoses are L4 radiculopathy and a partial femoral neuropathy.[153] Motor or reflex involvement rules out a pure saphenous nerve lesion. Weakness of the quadriceps muscles only or a depressed knee reflex is evidence in favor of a femoral neuropathy. If there is also weakness of the hip adductors and the tibialis anterior or the tibialis posterior muscles, the diagnosis is most likely a lumbar radiculopathy.

Origin

Saphenous neuropathies can occur anywhere along the course of the nerve. Most appear to be because of some sort of injury, although entrapments caused by anatomic factors have been described. Since the femoral artery travels with the saphenous nerve in the subsartorial canal in the thigh, the saphenous nerve is often damaged during arterial surgery such as femoral-popliteal bypass and femoral thrombectomy.[2,72,130,141] Compression can occur from fibrous bands and from branches of the femoral vessels.[112,159] Neurilemoma of the saphenous nerve presenting as pain in the knee has been reported.[38]

Knee pain caused by saphenous nerve entrapment where it pierces a fascial layer to leave the subsartorial canal has been reported.[58,82,108,110,173] Kopell and Thompson[82] described two patients who had sudden-onset knee pain who had paresthesias and sensory abnormalities in an incomplete saphenous nerve distribution. The patients improved after surgical decompression. The saphenous nerve was constricted at its point of emergence through the fascia. Stewart[53] has voiced the opinion that other reports of saphenous nerve entrapment where it leaves the subsartorial canal are less convincing. He points out that although the patients had pain in the lower part of the thigh and leg that was aggravated by walking, there were no paresthesias or sensory loss in a saphenous nerve distribution that unequivocally demonstrated saphenous nerve involvement.

The saphenous nerve can be damaged at the knee at the time of surgery, for example, during meniscectomy or arthroscopy.[104,132,141,157] External compression of the nerve can occur from knee-supporting stirrups. Surfers can compress their saphenous nerves by gripping the surfboard between their knees.[42] Saphenous nerve entrapment can be caused by pes anserine bursitis that mimics a stress fracture of the tibia.[64] The infrapatellar branch of the saphenous nerve may be damaged during knee operations, such as arthroscopy, by direct blunt trauma or accidental lacerations.[67,157,171] Although these injuries most commonly produce minor sensory symptoms, painful neuromas sometimes occur.[123] Wartenberg[171] called the spontaneous onset of paresthesias in the distribution of the saphenous nerve gonyalgia paresthetica. He thought that this condition could arise owing to entrapment of the saphenous nerve where it pierces the sartorius tendon. Alternatively, the nerve may be compressed when the knees press against each other, because the nerve crosses the medial epicondyle of the femur in some people.[171] Nerve compression while working in a kneeling position is also possible and may be in the differential diagnosis of housemaid's knees.[153]

The saphenous nerve may be injured during varicose vein operations in the lower leg.[47] Harvesting the saphenous vein for use during coronary artery bypass surgery[22,113] may cause saphenous nerve damage in 3% of patients.[88] Saphenous vein cannulation at the ankle may also injure the saphenous nerve.[108]

Practical Management

Pharmacologic pain management as outlined above for femoral neuropathy may be indicated. Local anesthetic and corticosteroid injections may be indicated for compression in the subsartorial canal. For severe and persistent neuropathic symptoms, surgical release of the nerve at its exit from the subsartorial canal can be carried out.[153]

Prognosis

New studies are always being conducted. However, when this article was written, no studies detailing the prognosis of saphenous neuropathy were found in a MEDLINE database search.

OBTURATOR NEUROPATHIES

Anatomy

The obturator nerve originates from the ventral divisions of the ventral rami of the L2, L3, and L4 spinal nerves within the psoas major muscle; this is in contrast to the femoral nerve, which is formed by fibers from the posterior divisions of the ventral rami of the same roots. The course of the obturator nerve in the pelvis is more medial than that of the femoral nerve.[34] The obturator nerve descends through the psoas muscle to emerge from its medial border at the pelvic brim. The nerve then curves downward and forward around the wall of the pelvic cavity and travels through the obturator foramen in which it divides into anterior and posterior branches: the anterior branch enters the thigh over the obturator externus and the posterior through the fibers of that muscle. The anterior branch innervates the adductor longus, gracilis, and adductor brevis muscles and gives off sensory fibers that innervate the medial aspect of the midthigh, sometimes extending to, and just below, the knee. The posterior division innervates the obturator externus and adductor magnus (which is also innervated by the sciatic nerve) muscles and, occasionally, the adductor brevis.[153,156]

Clinical Presentation

Symptoms

Some patients who have obturator neuropathy note sensory alteration in the medial thigh.[34,153] Symptoms can include paresthesias, sensory loss, or pain. Sometimes the pain and paresthesias can extend from hip to knee, along the medial aspect of the thigh.[156] Maneuvers that stretch the nerve such as extension or lateral leg movement can increase the pain.

Sensory complaints can extend to the medial calf, although the obturator nerve only rarely supplies sensation distal to the knee.[34,156] Patients may complain of trouble walking or leg weakness because they cannot normally adduct the ipsilateral hip.[153]

Signs

On examination, the hip adductors on the affected side are weak. Obturator externus (lateral rotation of the thigh) and gracilis (flexion and internal rotation of the leg) dysfunction is adequately compensated for by muscles innervated by other nerves.[156] Medial thigh wasting may be observed.[156] During ambulation, the hip is abnormally abducted, resulting in a circumducting, wide-based gait.[34,111] There can be an area of sensory loss or alteration in the mid and the lower thirds of the medial thigh, sometimes extending to below the knee.[34,153] Ipsilateral loss of the hip adductor tendon reflex can suggest obturator neuropathy. However, because it is not always present in healthy people, this reflex must be easily elicitable in the contralateral (asymptomatic) leg for the finding to be useful.[153]

Electrophysiology

Obturator neuropathy can be confirmed by needle EMG. Findings should be consistent with acute or chronic denervation in the hip adductors, but not in other lower extremity muscles such as the iliopsoas or quadriceps muscles.[78]

Other Diagnostic Tests

Additional diagnostic testing is sometimes needed in evaluating patients with obturator neuropathy. Examination of the pelvic or rectal areas, or examinations with CT, MR, or ultrasound imaging studies are indicated for suspected intrapelvic mass lesions entrapping the obturator nerve.[34,153]

Differential Diagnosis

Weakness of the hip adductor muscles or medial thigh sensory alteration can also be caused by lumbar radiculopathy or plexopathy. In those cases, weakness, sensory loss, and reflex loss should usually extend beyond an obturator nerve distribution.[34,153]

Origin

Traumatic injuries restricted to the obturator nerve are relatively uncommon. In addition to damaging the obturator nerve, pelvic fractures, gunshot wounds, and other traumatic conditions usually also injure other important neural structures.[34,118,154] The obturator nerve can be injured during pelvic or hip surgery secondary to stretch, compression from a retractor, encasement by cement, or thermal injury by cement or electrocautery.[12,32,102,119,137,146,172] Because the obturator nerve injury can follow laparoscopic pelvic lymphadenectomy, it is recommended that unequivocal visualization of the nerve be achieved during electrocautery because of the risk of nerve injury from thermal conduction through apposed tissues.[44] Massive pelvic hemorrhage during gynecologic cancer surgery has been reported to cause obturator neuropathy.[43] Malignant tumors in the pelvis may compress or invade the obturator nerve,[156] as can an aneurysm of the hypogastric artery.[34] Obturator neuropathy after cardiac catheterization caused by retroperitoneal hematoma formation has been reported.[75] Entrapment of the nerve within the obturator canal by obturator hernias has been reported.[110] Endometriosis may damage the nerve in the obturator canal or in the pelvis.[13,128] Compression of the obturator nerve can occur during pregnancy and delivery. The injury may be caused by the fetal head, the application of forceps, the trauma or hematoma caused by cesarean section, or the improper positioning in leg holders.[37,91,170]

Bradshaw et al[14] reported 32 athletes who had obturator neuropathy caused by a fascial entrapment of the obturator nerve where it enters the thigh. They described a characteristic clinical pattern of exercise-induced medial thigh pain commencing in the region of the adductor muscle origin and radiating distally along the medial thigh. Needle EMG demonstrated denervation of the adductor muscles. The role of conservative treatment in the management of this condition was not described in detail. Surgical neurolysis cured the problem, with athletes returning to competition within several weeks of treatment. At surgery, entrapment of the obturator nerve by a thick fascia overlying the short adductor muscle was found. Confirmation of this postulated entrapment neuropathy by others is awaited.

There are other case reports describing idiopathic obturator neuropathies.[135] One reported of a newborn infant[30] who had neuropathy, possibly secondary to prolonged abnormal leg position before birth.

Practical Management

Pharmacologic management of pain and physical therapy to improve strength and to preserve mobility and ambulation may be indicated. Nerve blocks may be useful for pain control. Surgical exploration may be indicated for prolonged or severe lesions, if it is thought that the nerve

may be encased in cement (after a hip arthroplasty) or impinged upon somewhere along its course.[14,34,153]

Prognosis

As stated above, new studies are always being conducted; however, when this article was written, no studies detailing the prognosis of obturator neuropathy were found in a MEDLINE database search.

SUMMARY

Femoral, saphenous, and obturator neuropathies have diverse causes, many of which are iatrogenic. They have overlapping but distinct clinical features. Electrodiagnostic testing results can distinguish between these disorders and others in the differential diagnosis. Imaging studies may demonstrate the origin of the neuropathy in some cases. Conservative treatment is usually sufficient, but occasionally surgical exploration of the affected nerve is indicated. The prognosis of femoral neuropathy has been studied, but no systematic studies of the prognosis of saphenous or obturator neuropathies have as yet been published.

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