

Clinical and Electrodiagnostic Features of Sciatic Neuropathies

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KEYWORDS

- Sciatic neuropathy • Common fibular division • Tibial division
- Electrodiagnostic testing

KEY POINTS

- Sciatic neuropathy is the second most common neuropathy of the lower extremity.
- Sciatic neuropathy often presents with foot drop, mimicking common fibular neuropathy.
- The hip is the most common site of sciatic nerve injury.
- Trauma and masses account for most of the pathology leading to sciatic neuropathy.
- Electrophysiologic studies can help localize the lesion.
- Neuroimaging can sometimes identify an abnormality in more severe cases.

INTRODUCTION

Sciatic neuropathy is the one of the most common neuropathies of the lower extremities, second only to common fibular (peroneal) neuropathy. One of the most common presentations of sciatic neuropathy is foot drop. Because ankle dorsiflexion weakness, with or without lower extremity sensory impairment, may also be associated with several other clinical syndromes, a careful evaluation is necessary before confirming a diagnosis of sciatic neuropathy. Electrodiagnostic testing is of great value in confirming the diagnosis of suspected sciatic neuropathy and assessing the potential for recovery of nerve function.

ANATOMY

The sciatic nerve is the longest and widest single nerve in the body, originating just distal to the lumbosacral plexus and extending distal branches into the feet. It is responsible for most of the function of the lower extremity. It is derived from several

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lumbosacral nerve roots and the lumbosacral plexus. A minor branch of L4 combines with the ventral ramus of L5 to form the lumbosacral cord or trunk. The lumbosacral trunk descends over the sacral ala and combines with the ventral rami of S1, S2, and S3 and a branch of S4 to form the sacral plexus.^{1,2} The sciatic nerve forms the sacral plexus apex, located anteriorly to the sacroiliac joint and the piriformis muscle (**Fig. 1**).³

The sciatic nerve comprises the lateral division, which eventually forms the common fibular nerve, and the medial division, which forms the tibial nerve, each separately encased from the outset.² After arising at the exit of the superior and inferior gluteal nerves, the sciatic nerve leaves the pelvis via the sciatic notch, typically through the greater sciatic foramen, and is accompanied by the posterior femoral cutaneous nerve, inferior gluteal artery, pudendal vessels and nerves (**Fig. 2**). There is a close relationship to the piriformis muscle, which also exits the pelvis via the greater sciatic notch. The sciatic nerve usually travels under the piriformis muscle, except in 10%–30% of

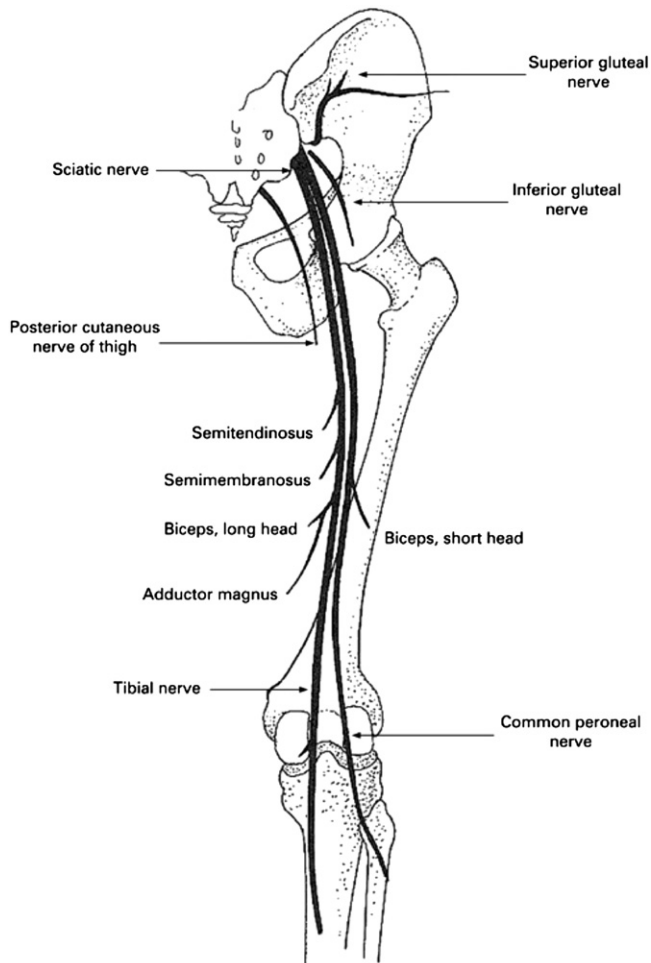


Fig. 1. The anatomy of the sciatic nerve from the gluteal region to the thigh. (From Stewart JD. Foot drop: where, why and what to do? *Pract Neurol* 2008;8:158–69; with permission.)

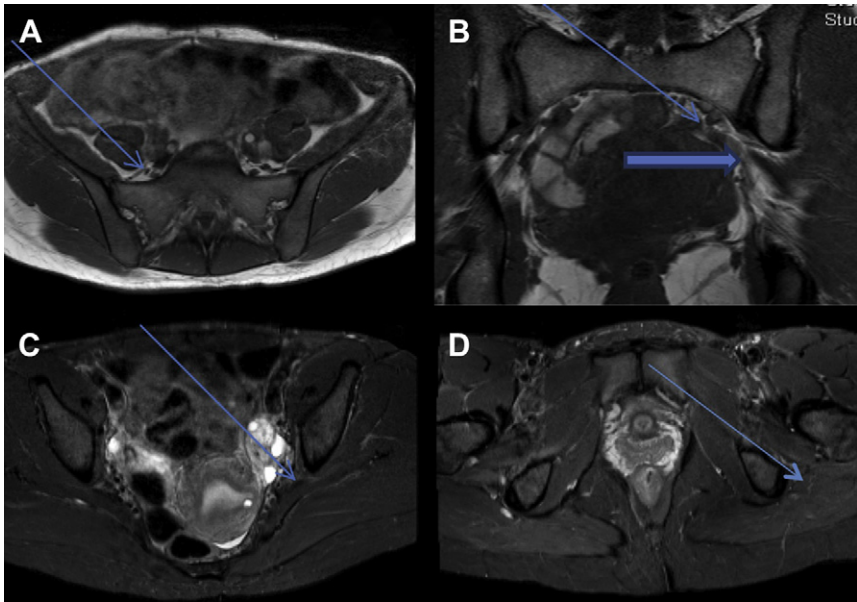


Fig. 2. The course of a normal sciatic nerve shown by MRI. (A) The lumbosacral trunk of the sciatic nerve (arrow) is shown lying posterior to the iliac vessels (axial T1). (B) The sacral plexus (thin arrow) and nerve at the notch sciatic notch (thick arrow) are denoted (coronal T1). (C) The sacral plexus (arrow) lies anterior to the piriformis muscle (axial T2). (D) The sciatic nerve (arrow) is situated between the ischial tuberosity and greater trochanter (axial T2).

cases, notably when the fibular division passes through or above the piriformis muscle.⁴

Distal to the piriformis muscle, the nerve is covered by the gluteus maximus muscle and soft tissue.¹ The nerve then travels halfway between the bony landmarks, greater trochanter laterally, and ischial tuberosity medially, and then descends into the sub-gluteal area. From there, it runs posteriorly in the midthigh, remaining dorsal to the adductor magnus and ventral to the long head of the biceps femoris. The tibial division of the sciatic nerve innervates the hamstring muscles (semimembranosus, semitendinosus, and long head of the biceps femoris) and the adductor magnus in the thigh. In the thigh, the common fibular division innervates the short head of the biceps femoris.

The nerve divides into the common fibular and tibial branches approximately 6 cm above the popliteal fossa crease.⁵⁻⁷ The tibial nerve then continues posteriorly in the midline to the calf, innervating the muscles of the posterior compartment of the lower leg and supplying sensation to the posterior calf and lateral foot by the sural nerve (which also has a limited contribution from the common fibular nerve), the sole of the foot by the medial and lateral plantar nerves, and the heel by the medial calcaneal nerve. After passing through the upper popliteal fossa, the common fibular nerve travels laterally and around the fibular head, dividing into the deep fibular and superficial fibular branches, which supply the muscles of the anterior and lateral compartments of the lower leg, respectively. The superficial fibular nerve also forms a sensory branch that supplies sensation to the anterolateral lower leg and dorsum of the foot while the deep fibular nerve supplies sensation to the webspace between the first and second toes.²

PRESENTATION

Sciatic neuropathy often presents with foot drop. Patients often experience abrupt pain radiating down the posterolateral limb, with weakness and numbness evolving more gradually.^{8–10} In sciatic neuropathy, the clinical findings are often more consistent with injury to the common fibular division rather than tibial division, sometimes mimicking a common fibular neuropathy at the knee. This finding is particularly true of more distal lesions, as they may not affect the flexors of the knee, or of less severe sciatic nerve injury. Because the common fibular division has fewer and larger fascicles and less supportive tissue compared with the tibial division, it is thought to be more vulnerable to compression. Also, the common fibular division is more taut, and secured at the sciatic notch and fibular neck, resulting in greater potential for stretch injury.²

In milder cases at the hip or thigh, the following features are typically noted:

- Foot drop that may mimic a common fibular neuropathy at the knee
- Weakness in knee flexion, ankle plantar flexion, ankle inversion
- Normal or decreased ankle jerk
- Pain and sensory loss in the foot and possibly the lateral shin

In severe lesions, these signs and symptoms are common:

- Weakness in ankle dorsiflexion and plantar flexion and toe extension and flexion
- Hamstring weakness
- Decreased ankle jerk
- Dysesthetic pain and numbness in the sole and dorsum of the foot and lateral lower leg

ELECTRODIAGNOSTIC STUDIES

Electrodiagnostic testing (EDx) is helpful in localizing the site of injury and the severity of the lesion. EDx studies are also useful for assessing both recovery and prognosis. Standard nerve conduction studies for evaluation of the sciatic nerve include testing the following:

- Ipsilateral common fibular and tibial motor nerve conduction and minimum F wave latencies
- Superficial fibular and sural sensory nerve conduction
- Comparison with the unaffected leg

Findings on motor nerve conduction studies most commonly include reduced fibular compound muscle action potential (CMAP) amplitudes often with a normal tibial CMAP amplitude. Given the depth and size of the sciatic nerve proximally, it is not possible to obtain reliable studies with more proximal stimulation using standard techniques. Tibial H reflex studies may be normal in fibular-predominant lesions, and therefore may not confirm the diagnosis or help localize the site of injury. In sensory nerve conduction studies, reduced superficial fibular and sural sensory nerve action potential amplitudes are seen in most cases (**Table 1**). Similar abnormalities are found in different age populations.^{11–13} In one study of patients with sciatic neuropathy, nerve action potential amplitudes were reduced in the fibular motor nerve of 80% of adults and 83% of children; tibial motor nerve of 52% of adults and 67% of children; sural sensory of 71% of adults and 79% of children; and superficial fibular sensory of 83% of adults and 60% of children. Minimum F wave latencies in adult patients with sciatic neuropathy were abnormally prolonged in 85% of fibular and in 57% of tibial nerve studies.¹¹

Table 1
Nerve conduction findings in sciatic neuropathy

Nerve	Latency	Amplitude	Velocity	F Wave
Common fibular motor				
Ankle	NL	Reduced		Abnl ^a
Below fibular head	NL	Reduced	NL	
Above fibular head	NL	Reduced	NL	
Tibial motor				
Ankle	NL	NL to reduced		NL/Abnl ^a
Knee	NL	NL to reduced	NL	
Sural sensory	NL	NL to reduced	NL	
Suprfl fibular sensory	NL	NL to reduced	NL	

Abbreviations: Abnl, abnormal; NL, normal; Suprfl, superficial.

^a F wave abnormalities include prolonged latency or decreased persistence.

To maximize the yield for identifying signs of active denervation, needle electromyography should generally be performed 3 to 4 weeks after onset of symptoms. Positive sharp waves and fibrillation potentials can be identified reliably only after this period, initially in muscles closer to the lesion and then in those more distal, with reinnervation occurring in a similar pattern. In sciatic neuropathy, any muscle in the foot and lower leg is likely to show denervation. Commonly tested muscles include the extensor digitorum brevis, tibialis anterior, tibialis posterior and gastrocnemius. In general, however, needle EMG abnormalities in sciatic neuropathy are more commonly seen in fibular-innervated muscles (94%–100% of patients) than tibial-innervated muscles (74%–84% of patients).¹¹ Muscles innervated by the sciatic nerve in the thigh that often show denervation, depending on the site of the lesion, include both the short and long heads of the biceps femoris, semimembranosus, and semitendinosus.

In addition to studying muscles innervated by the sciatic nerve, the examiner should also study muscles that share the same root innervation but are supplied by a nerve other than the sciatic, such as the gluteus medius or tensor fascia latae (superior gluteal nerve) and the gluteus maximus (inferior gluteal nerve) to exclude L5 or S1 radiculopathy and lumbosacral plexopathy. To rule out a length-dependent process, such as a peripheral neuropathy, the electromyographer should also compare distal with proximal muscles of the leg, including those muscles in the proximal leg not supplied by the sciatic nerve. For example, the examiner could compare the extensor hallucis longus muscle (supplied by the sciatic nerve) with the vastus medialis muscle (supplied by the femoral nerve). Paraspinal muscle abnormalities of the lumbosacral spine on needle examination support a diagnosis of lumbosacral radiculopathy rather than sciatic neuropathy or a superimposed additional site of neuropathic injury.

Performed together, nerve conduction studies and needle electromyography can detect subclinical involvement of the tibial nerve and thereby exclude a common fibular mononeuropathy, for example, by showing involvement in the gastrocnemius or hamstring muscles. It is important to recognize that more distal tibial-innervated muscles, such as the gastrocnemius, tibialis posterior, and flexor digitorum longus, are more likely to show denervation than the hamstring muscles in chronic lesions, because the hamstring muscles may have had adequate time to reinnervate.¹¹

IMAGING TECHNIQUES

Magnetic Resonance Imaging

Of all imaging techniques, magnetic resonance imaging (MRI) seems to be the best technique to identify sciatic nerve pathology. Depending on the severity of the injury, T2-weighted magnetic resonance images may show high signal intensity in the nerve fibers or increased nerve dimension, deformation of the nerve, or total loss of nerve integrity.¹⁴ Short tau inversion recovery (STIR) sequences of the nerve help identify the extent of a lesion, with a more diffuse area of high signal indicating an inflammatory etiology (**Fig. 3**). Chhabra and colleagues¹⁵ found higher nerve-to-vessel signal intensity ratios and higher incidences of T2 hyperintensity, nerve enlargement, and abnormal fascicular shape in affected nerves compared with normal nerves, with excellent interobserver and intraobserver reliability. Affected muscles showed more fatty infiltration, edema, and atrophy. However, findings on MRI may sometimes be difficult to interpret, because abnormal signal may occasionally be seen in normal individuals on both T2 and STIR imaging.

Other

Computerized axial tomography is helpful in finding lesions that impact the sciatic nerve involving bone (eg, sacrum fracture), vessel abnormalities (eg, aneurysm), or hematoma.³ Ultrasonographic studies have limited utility in the diagnosis of sciatic neuropathy and have been used more frequently for sciatic nerve blocks rather than to identify sciatic nerve pathology. However, a few recent studies have tried to use ultrasound scan to foster a greater understanding of the causes of sciatic nerve injury. For instance, Moayeri and Groen¹⁶ and Brull and colleagues,¹⁷ with the use of specific ultrasonographic settings, found more neural than nonneural tissue in the hip region than in the thigh, which might contribute to the greater proximal vulnerability of the sciatic nerve.

ETIOLOGIES

Different conditions can lead to sciatic neuropathy (**Table 2**). In one retrospective study of individuals referred for electrodiagnostic evaluation of sciatic neuropathy, hip trauma and surgery were the most common etiologies. Sciatic neuropathy has

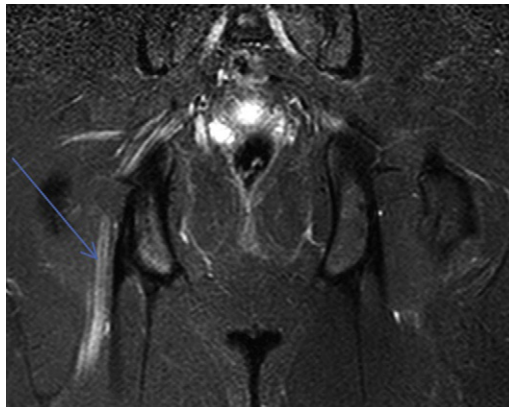


Fig. 3. Increased signal (*arrow*) is shown in an idiopathic right sciatic neuropathy in a proximal nerve segment. (coronal STIR).

Table 2
Etiologies of sciatic neuropathy

Infection	Abscess ^{3,20} ; tubo-ovarian, pelvic, psoas
Inflammation	Sacroiliitis ^{40,53,54}
Trauma	Intramuscular injection, ^{8,9,55,56} abdominal surgery, ⁵⁷ fracture, ⁵⁸ hematoma, ⁵⁹ open injury including gunshot or knife wound ^{21,60,61}
Tumor: intraneural	Schwannoma, ³ intraneural perineurioma, ^{24,26} neurofibroma, ²⁵ neurilemmoma, ²⁵ neurolymphomatosis, ^{3,24,27} malignant neurofibrosarcoma ^{25,62,63}
Compressive	Leiomyosarcoma, ²⁸ rhabdomyosarcoma, ²⁴ lipoma, ⁶⁴ metastasis ³
Vascular	Vasculitis, ²³ iliac artery occlusion ^{21–23} venous varix, ⁶⁵ arteriovenous malformation ^{20,66} ischemic, ⁶⁷ deep venous thrombosis ⁶⁸
Gynecologic	Endometriosis, ^{46,51} fibroids ⁶⁹
Other	Piriformis syndrome, ^{41,42} radiotherapy, ⁷⁰ hereditary neuropathy with liability to pressure palsy, ⁷¹ cryoglobulinemia ⁷²
Surgery, hip	Traction, ⁷³ arthroplasty debris ⁷⁴
Surgery, other	Compartment syndrome, ⁷⁵ anesthesia, ¹⁹ positioning ⁷⁶

Data from Ergun T, Lakadamyali H. CT and MRI in the evaluation of extraspinal sciatica. *Br J Radiol* 2010;83(993):791–803.

been reported in as many as 1%–3% of patients after total hip replacement surgery.^{18,19} The neuropathy is usually discovered immediately postoperatively and is typically a consequence of stretch injury. The next most common causes include external compression and open injuries, such as gunshot wounds and knife injury, and ischemia, which may occur secondary to vasculitis or atherothrombosis or after bypass surgery. Endometriosis, iatrogenic nerve injection, and compartment syndrome are also less commonly reported causes. Not infrequently, a cause cannot be identified (ie, idiopathic sciatic neuropathy).²⁰ An etiology for sciatic neuropathy was uncertain in 16% in the series reported by Yuen and colleagues²¹ of 73 patients. Vascular malformations may also uncommonly lead to sciatic neuropathy as a consequence of nerve compression.^{22,23}

Tumors involving the sciatic nerve are uncommon in adults, but in the pediatric population, sciatic neuropathy is often associated with a neoplasm. In a series of 7 pediatric cases, etiologies included nerve infiltration by an adjacent neoplasm (neuroblastoma, rhabdomyosarcoma, and leukemic and lymphomatous infiltration) as well as an intrinsic neurogenic tumor (perineurioma).²⁴ Although tumors are more common presentations in childhood, occurring often in the first decade, neurilemmomas, neurofibromas, neurofibrosarcomas, perineuriomas, lymphoma, leiomyosarcoma, and malignant peripheral nerve sheath tumors have been reported rarely in adults.^{25–29}

Certain etiologies for sciatic neuropathy often are associated with specific sites of injury to the sciatic nerve:

Hip

Given its proximal location to the hip joint and its long course, the sciatic nerve is predisposed to injury in the hip region. Partial entrapment of the sciatic nerve at the hip affects the lateral (fibular) division more commonly. Etiologies include hip arthroplasty, dislocation, and fracture in up to 30% of patients.^{10,30} Acute external compression can occur in patients in coma, with or without compartment syndrome, or after

prolonged sitting. Mass lesions are more common at this site than elsewhere, occurring in up to 14% of cases.^{19,31–35}

Gluteal

Gluteal compartment hemorrhage, misplaced intramuscular gluteal injections, and repeated injections leading to fibrosis can lead to sciatic nerve injury at this location.^{8,36–39} The sciatic nerve may also be at risk of compression based on its close association with the piriformis muscle at the level of the sciatic notch. Compression of the nerve by the piriformis muscle as a clinical syndrome was proposed by Freiberg and Vinke in 1934.⁴⁰ Robinson⁴¹ is credited with coining the term *piriformis syndrome*.⁴¹ Individuals with piriformis syndrome typically present with buttock tenderness and pain, which may radiate down the posterior thigh. Prolonged sitting, bending at the waist, and activities involving hip adduction and internal rotation exacerbate the pain. Paresthesias may occur in the buttocks and thigh, but weakness is not usually evident. The pain can be reproduced with palpation over the sciatic notch or with rectal or pelvic examination.⁴¹ Additionally, several maneuvers have been described to assess for piriformis syndrome in eliciting buttocks pain by either (1) passive stretching of the piriformis muscle (the Freiberg or flexion adduction internal rotation/FAIR maneuver) or (2) active contraction against resistance to the piriformis muscle (the Pace and Beatty maneuver).⁴² Although helpful, the Freiberg and Pace maneuvers only have a sensitivity of about 75% in confirmed cases.⁴¹ Potential causes of piriformis syndrome include hypertrophy of the piriformis muscle, which can cause impingement of the sciatic nerve at the greater sciatic foramen; inflammation of the piriformis muscle secondary to adjacent infectious or inflammatory processes; hematoma or fibrous adhesions of the piriformis muscle; and local ischemia of the nerve at this site.⁴³

Thigh

Causes of sciatic neuropathy at the thigh include external compression from coma and posterior thigh compartment syndrome. Prolonged positioning on a toilet seat, lotus position or craniotomy^{32,33,44,45} have been described as causes of sciatic neuropathy. Transient symptoms of sciatic neuropathy may also occur in endometriosis associated with menses.⁴⁶ Sciatic nerve injury can also occur at this site as a consequence of a gunshot wound or penetrating injury.²¹

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of sciatic neuropathy is largely limited to L5 or S1 radiculopathies, lumbosacral plexopathy, and common fibular neuropathy (**Table 3**). Additionally, distal lower extremity weakness mimicking sciatic neuropathy may be the presenting feature of motor neuron disease, distal myopathy or polyneuropathy. Some clinical findings may be helpful in distinguishing a sciatic nerve insult from these other nerve injuries. With L5 or S1 radiculopathies, patients typically describe low back pain radiating into the lateral (L5 distribution) or posterior (S1 distribution) leg. The pain in radiculopathy is better with standing and walking and worse with sitting. In contrast, sciatic nerve pain usually originates in the buttocks or more distally. Lumbosacral plexopathy often involves more weakness than sciatic neuropathy, including involvement of the gluteal muscles, and pain is usually more diffuse.⁴⁷ Unlike sciatic neuropathy, common fibular neuropathy should not affect knee flexion, and sensory symptoms in this neuropathy do not involve the posterior leg or plantar aspect of the foot.

Table 3
Differential diagnosis of sciatic neuropathy

	Sciatic Neuropathy	Common Fibular Neuropathy	LS Plexopathy	Radiculopathy	
				L5	S1
Pain	Foot + radiating posterolateral thigh	Rare	Posterolateral thigh	Low back to lateral thigh	Low back to posterior thigh
Weakness	Foot inversion, toe flexion, \pm knee flexion	Ankle dorsiflexion/foot eversion	Ankle dorsiflexion and plantar flexion	Ankle dorsiflexion \pm foot inversion	Ankle plantar flexion
Sensory	Upper 1/3 lateral leg/sole	Distal 2/3 lateral leg	\pm Antero- or posterolateral thigh and shin	\pm Lateral thigh across to medial foot	\pm Posterior thigh
Reflex	Depressed/absent - ankle	Normal	Norm/depressed at ankle	Normal	Depressed/absent - ankle
SLR	+	—	—	+	+
Common Fibular CMAP	Reduced	Reduced	\pm Reduced	\pm Reduced	Normal
Tibial CMAP	\pm Reduced	Normal	\pm Reduced	Normal	\pm Reduced
Sural SNAP	\pm Reduced	Normal	\pm Reduced	Normal	Normal
Sup. Fibular SNAP	Reduced	Reduced	Reduced	Normal	Normal

Abbreviations: SLR, straight leg raise sign; SNAP, sensory nerve action potential; Sup, superficial.

TREATMENT

Identification of the cause of sciatic neuropathy is vital in determining prognosis and therapy. For instance, in piriformis syndrome, conservative therapy is key, including stretching in flexion, adduction, and internal rotation of the hip joint, supine and standing.⁴⁷ Physical therapy and ankle foot orthotic for foot drop are commonly indicated. Steroid or botox injections have been used in cases of suspected piriformis syndrome.⁴⁸ Two randomized, controlled trials showed efficacy of botulinum toxin injection into the piriformis muscle compared with placebo.^{49,50} Pharmacologic suppression of the ovarian cycle may reverse sciatic neuropathy in endometriosis.⁵¹ Immunosuppressant therapy, such as prednisone, would be warranted as therapy for vasculitis affecting the sciatic nerve. Neuropathic pain is common in all causes of sciatic neuropathy and is generally treated with neuropathic pain medications such as tricyclic antidepressants like amitriptyline or nortriptyline; anticonvulsants, including gabapentin, pregabalin, or carbamazepine; and lidocaine patch. Surgery is indicated in compartment syndrome, hip fracture or dislocation, and tumors involving the sciatic nerve to prevent progressive symptoms.⁵²

Nerve grafting may play a role in treatment of severe sciatic nerve injuries from trauma but is a challenge in a nerve of this size and length. In a large retrospective review of 353 patients by Kim and colleagues,⁵² individuals with gluteal or thigh-level injury were examined to determine the degree of recovery after neurolysis if an intraoperative nerve action potential could be generated or after end-to-end suture repair or graft in severe sciatic neuropathy without an elicitable intraoperative nerve action potential. Outcomes varied depending on the mechanism of injury, with better responses to surgery noted in patients suffering from tibial division lesions than in those sustaining insults primarily to the common fibular division. Partial recovery ranged from 71%–96% of patients undergoing neurolysis, 30%–93% of those treated with end-to-end suture repair, and 24%–80% of those undergoing nerve grafts.

OUTCOME

In general, the prognosis for sciatic neuropathy is chiefly dependent on the severity of the lesion rather than the location. Yuen and colleagues²¹ found that most individuals with sciatic neuropathy had a good outcome at 3 years, whereas only 30% had a good or better recovery at 1 year. Good but incomplete recovery occurred primarily in those who did not show severe motor axonal loss on EDx study. In patients with an acute or subacute onset, a moderate or better recovery occurred in most patients: 30% recovered within 1 year, 50% within 2 years, and 75% within 3 years.²¹ Two factors predicted an earlier or better recovery: (1) presence of a common fibular compound muscle action potential recording from the extensor digitorum brevis, as all of these patients had moderate to excellent recovery by 3 years, and (2) an initial absence of paralysis of muscles controlling ankle plantar flexion and dorsiflexion.¹¹

SUMMARY

Sciatic neuropathy is a common cause of foot drop and the second most common neuropathy of the lower extremity. Sciatic neuropathy must be distinguished from other causes of foot drop, including common fibular neuropathy, lumbosacral plexopathy, and L5 radiculopathy. Less commonly, distal lower extremity weakness mimicking sciatic neuropathy may be the presenting feature of motor neuron disease, distal myopathy, or polyneuropathy. There are multiple potential sites of injury along the sciatic nerve, determined in part by the mechanism of insult, such as trauma, compression,

mass, inflammation, and vascular lesions. Diagnosis is augmented by careful EDx studies and imaging, which help to distinguish sciatic neuropathy from other neuropathic conditions. EDx may also help gauge recovery and aide in prognosis.

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