Electrodiagnostic Testing in Lumbosacral Plexopathies

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KEYWORDS

• EMG • Lumbosacral plexopathy • Lumbosacral • Radiculoplexus neuropathy
• Lumbar plexopathy • Diabetic lumbosacral radiculoplexus neuropathy
• Diabetic amyotrophy

KEY POINTS

- Pure lumbosacral plexopathies are rare.
- Lumbosacral radiculoplexus neuropathies are more common than pure lumbosacral plexopathies.
- Inflammatory diabetic and nondiabetic lumbosacral radiculoplexus neuropathies are among the most common causes of lumbosacral plexopathies and are pathologically due to ischemic injury and microvasculitis.
- Lumbosacral plexopathies often do not occur alone but are found in association with thoracic and cervical radiculoplexus neuropathies.

INTRODUCTION

The lumbosacral roots and the lower extremity peripheral nerves are commonly involved in peripheral nervous system diseases (radiculopathies, length-dependent peripheral neuropathies). The lumbosacral plexus (composed of both the upper lumbar plexus and lower lumbosacral plexus) as a primary target for peripheral nervous system disease is less common. Despite this lesser frequency, the scope of processes that may be implicated in lumbosacral plexopathies is vast, ranging from compressive causes (ie, hematoma) and neoplastic diseases[1,2] to inflammatory conditions secondary to systemic disease (ie, diabetic radiculoplexus neuropathies [DRPN]).[1,2]

In all cases of lumbosacral plexopathy, electrodiagnostic studies incorporating nerve conduction studies and needle electromyography (EMG) can be helpful for localization and characterization of the underlying process. Localization to the lumbar

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plexus as opposed to nerve roots or individual roots is important, because diagnostic implications, evaluation, and treatment options may be different. Disorders that clinically present as a lumbosacral plexopathy often are not isolated to the lumbosacral plexus electrophysiologically and can extend into the roots (paraspinal involvement) as well as peripheral nerves. The concept of a radiculoplexus neuropathy (a process involving roots, plexus, and peripheral nerves) may therefore perhaps be more fitting when considering an electrodiagnostic approach to evaluating these patients.

This article focuses on nerve conduction studies and needle EMG in the diagnosis of lumbosacral plexopathies. In some clinical scenarios, there may be usefulness in other electrodiagnostic testing, including autonomic and quantitative sensation testing, to more firmly establish a diagnosis of lumbosacral plexopathy.

ANATOMY

Knowledge of the lumbosacral plexus anatomy is critical in assessment of the patient and planning a comprehensive electromyographic evaluation. Although often considered one entity, the lumbosacral plexus can be divided into 2 parts anatomically: the "upper" the lumbar plexus; and the "lower" the lumbosacral plexus.

Lumbar Plexus

The lumbar plexus lies within the psoas muscle and comprises the anterior rami of the T12 to L4 nerve roots (**Fig. 1**). Many of these muscles and nerves cannot be tested by standard EMG techniques because they are deep in the abdomen or are small cutaneous nerve branches. The 6 major branches of the lumbar plexus include[3]:

- Iliohypogastric nerve (T12/L1), which supplies the transverse and internal oblique muscles as well as sensation to the low abdomen.
- Ilioinguinal nerve (L1), a sensory branch to the inguinal region that also provides sensation to a small area of medial thigh and upper scrotum/labia sensation.
- Genitofemoral nerve (L1, L2) provides sensation to the skin of the femoral triangle. In men, this nerve provides muscular innervation to the cremasteric muscle and sensory innervation to the lower scrotum. In women, this nerve provides lower labial sensation.
- Lateral femoral cutaneous nerve (L2,L3) is a sensory branch to the lateral thigh. This sensory nerve, unlike those listed earlier, can be tested by nerve conduction techniques, although reliability of the results is questionable.
- Obturator nerve (L2, L3, L4 anterior rami) provides motor innervation to the thigh adductors and a small area of sensation to the medial thigh.
- Femoral nerve (L2, L3, L4 anterior rami, posterior divisions) is the largest branch of the lumbar plexus whose motor component can be tested by nerve conduction techniques. This branch supplies motor innervation to the iliopsoas, sartorius, and quadriceps muscles, and divides to form the saphenous nerve, which supplies sensation to the medial lower leg.

Unlike the brachial plexus, there are no subcomponents (trunks, cords) in the lumbar plexus (see **Fig. 1**).

Lumbosacral Plexus

The lumbosacral plexus primarily originates from the ventral rami L4 to S3 nerve roots (see **Fig. 1**).

- The lumbosacral trunk (also called the furcal nerve) has an L4 component that joins the L5 nerve root to form the lumbosacral trunk. This nerve then joins the

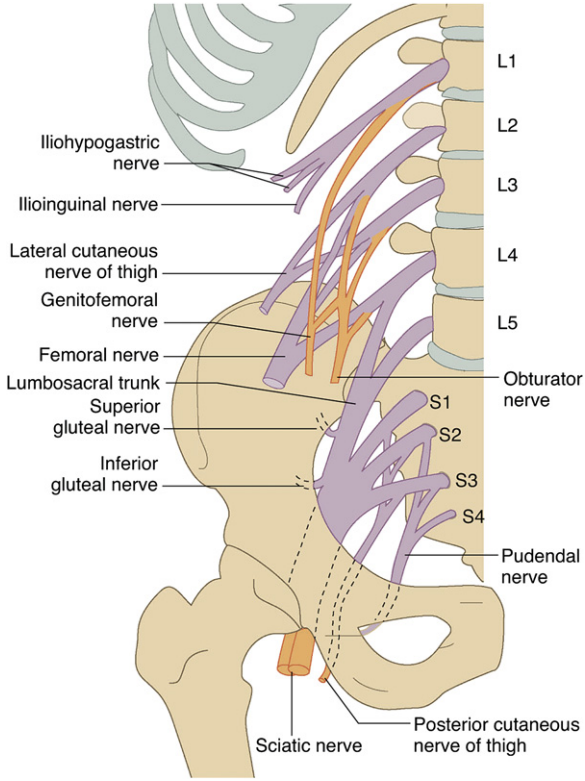

Fig. 1. Anatomy of the lumbosacral plexus. (Elsevier illustration from www.elsevierimages.com. © Elsevier Inc. All rights reserved.)

sacral plexus within the pelvic outlet. This structure is commonly affected in postpartum lumbosacral plexopathy, thought to be secondary to compression from the fetal head.[4]

- Superior gluteal nerve (L4, L5, S1) supplies motor innervation to the tensor fascia lata, gluteus medius, and gluteus minimus muscles.
- Inferior gluteal nerve (L5, S1, S2) provides motor innervation to the gluteus maximus muscle.
- Sciatic trunk/nerve (L5–S3) provides most motor innervation to the muscles of the posterior thigh and then into the leg via its 2 branches (common peroneal and tibial nerves).
- The pudendal nerve is formed from the anterior S2, S3, and S4 roots.

The tibial portion of the sciatic nerve innervates all the muscles of the posterior thigh except the short head of the biceps femoris, which is innervated by the peroneal division of the sciatic nerve or the common peroneal nerve branch of the sciatic nerve. This point is important when performing needle electromyogram studies for localization purposes.

HISTORY AND PHYSICAL EXAMINATION

When presented with a patient with lower limb symptoms, careful history and examination are imperative. Many different processes can cause lower extremity

plexopathy,[2] and the responses to a few questions can be important in narrowing a differential diagnose and planning a subsequent electrodiagnostic study:

Onset

Did the process begin acutely (hours to days), subacutely (days to weeks), or is it chronic (months to years)? Most lumbosacral plexopathies have an acute to subacute onset, which is helpful in identifying the process.

Progression

Are the symptoms and findings worsening, stable, or improving? The disease course is a helpful feature to consider when thinking about the cause of a lumbosacral plexopathy as well as predicting prognosis. For instance, a slow, progressive course may point to a malignant cause, whereas a relapsing course may favor an inflammatory cause.

Extent

Is the process unilateral or bilateral at onset? We, and others, have described inflammatory plexopathies that are generally unilateral and focal in onset but become bilateral and widespread with time.[5] At clinical presentation, the disease may be bilateral but it should not be assumed that symptoms were bilateral at onset. This point should be clarified when taking a medical history. Also, it is important to confirm that the symptoms are confined to the lower limb, because involvement of the upper limb may make a structural process less likely, and raise concern for a more diffuse, possibly inflammatory, process affecting cervical, thoracic, as well as lumbosacral segments.

Pain, Sensation, and other Temporally Associated Symptoms

Associated pain and sensation changes are important clues in lumbosacral plexopathies. If the weakness seems to be confined to the plexus distribution, but the sensory loss seems to follow a more dermatomal distribution, this may increase the extent of needle examination and encourage imaging and further work-up, because concern for a primary root level process (ie, radiculopathy) may be heightened. Other clinical points to consider are associated systemic features that may support an inflammatory immune disorder such as sarcoidosis. Weight loss, in addition to constitutional symptoms such as fever and night sweats, may support the diagnosis of neoplastic infiltration or a paraneoplastic cause. A rash associated with the neuropathic symptoms may occur in the context of certain vasculitides.

ELECTROPHYSIOLOGIC EVALUATION

Nerve Conduction Studies

The electrophysiologic presence of lumbosacral plexopathy can be defined when there is evidence for electrophysiologic abnormalities in the distribution of at least 2 different peripheral nerves in at least 2 different nerve root distributions. Sparing of paraspinals on needle examination is also helpful in localizing a pure lumbosacral plexopathy.[6] However, most of these conditions are not pure and involve paraspinal denervation, hence our preferred term: radiculoplexus neuropathies.

Several sensory and motor nerve conduction studies are helpful in the diagnosis of a lumbosacral plexopathy (**Box 1**, **Table 1**). Several of these nerve conduction studies are not performed on routine lower limb studies and can be considered when evaluating for a lumbosacral plexopathy, especially if clinically involvement of the upper lumbar plexus is suspected. In many cases, if symptoms are unilateral, bilateral

Box 1
A nerve conduction study protocol for lumbosacral plexopathy

If the lumbar plexus is the most likely site of a lesion, then test:

1. Peroneal motor nerve conduction with F-wave study
2. If the peroneal compound muscle action potential (CMAP) amplitude is low or the conduction velocity is reduced then:

   Consider testing the opposite side

   Consider recording the peroneal motor nerve over the tibialis anterior muscle
3. Tibial motor nerve conduction with F-wave study
4. Femoral motor nerve conduction study with side-to-side comparisons (may require needle stimulation)
5. Sural sensory nerve conduction
6. Superficial peroneal sensory nerve conduction
7. Saphenous sensory nerve conduction
8. Lateral femoral cutaneous sensory nerve conduction

If the lumbosacral plexus is the most likely site of a lesion, then test:

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2. If the peroneal CMAP amplitude is low or the conduction velocity is reduced then:

   Consider testing the opposite side

   Consider recording the peroneal motor nerve over the tibialis anterior muscle
3. Tibial motor nerve conduction with F-wave study
4. Sural sensory nerve conduction

   Superficial peroneal sensory nerve conduction

In both, it is likely necessary to compare abnormalities or borderline abnormalities with the contralateral limb.

If symptoms are bilateral, consider studying the upper limb to assess for a polyradiculoneuropathy or peripheral neuropathy.

studies can be helpful in determining a relative reduction in the size of the motor or sensory response (looking for axonal loss). Our laboratory uses a 50% difference from side to side as representing a significant and potentially pathologic difference.[7]

Sensory Nerve Conduction Studies

In cases of lumbosacral plexopathy, the sensory studies are likely to be most helpful for localization. Recall that, in most spinal segments, the dorsal root ganglion lies lateral to and outside the intervertebral foramen (**Fig. 2**). In electrodiagnostics, this feature is important because it helps assist in localization of a preganglionic process (ie, radiculopathy), versus a postganglionic process (ie, plexopathy or mononeuropathy). Therefore, a key point to remember is that, in most cases of lumbosacral plexopathy, reduced sensory nerve action potential amplitudes imply a postganglionic process and can help to exclude a radiculopathy as the main cause for clinical symptoms. The saphenous and lateral femoral cutaneous sensory nerve conductions in the upper lumbar plexus (see **Table 1**) are less technically reliable, whereas the sensory

Table 1
Useful nerve conduction studies in the evaluation of lumbar and lumbosacral plexopathies

	Stimulation Site	Recording Site	Pitfalls
Motor Nerves			
Lumbar Plexus			
Femoral (L2, L3, L4)	Femoral nerve in inguinal region	Quadriceps	Nerve is deep; often requires needle stimulation Proximity to femoral artery
Lumbosacral Plexus			
Peroneal (L4, L5)	Ankle and popliteal fossa	Extensor digitorum brevis (L5) Anterior tibialis (L4, L5)	Can be impaired in length-dependent neuropathies
Tibial (L5, S1, S2)	Ankle and popliteal fossa	Abductor hallucis (L5, S1)	Can be impaired in length-dependent neuropathies
Sensory Nerves			
Lumbar Plexus			
Saphenous (L3, L4)	Medial leg	Medial leg	Technically difficult
Lateral femoral cutaneous (L3, L4)	Lateral thigh	Lateral thigh	Technically difficult, deep, requires side-to-side comparison
Lumbosacral Plexus			
Superficial peroneal (L5)	Anterior shin	Distal anterior shin	Can be impaired in length-dependent neuropathies
Sural (S1, S2)	Ankle, midcalf and high calf	Ankle	Can be impaired in length-dependent neuropathies

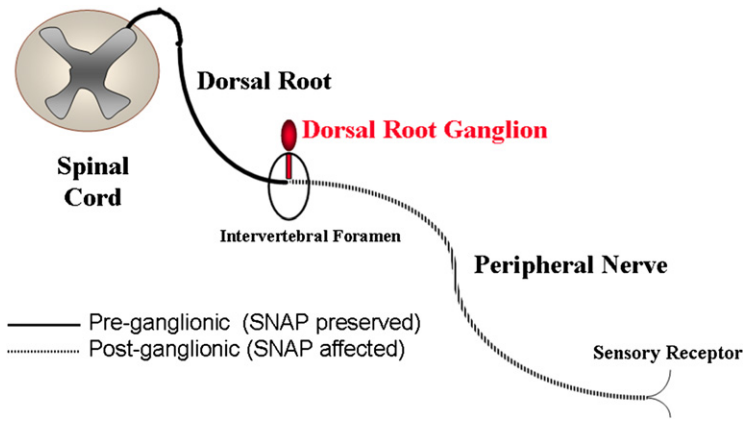

Fig. 2. Anatomy of the dorsal root ganglion. SNAP, sensory nerve action potential.

studies in the lumbosacral plexus (superficial peroneal, sural, and medial plantar) are more reliable and therefore more useful.

For example, the finding of the unilateral absence of superficial peroneal sensory and sural sensory nerve action potentials (SNAPs) suggests a lesion at or proximal to the sciatic nerve and lumbosacral plexus but distal to the dorsal root ganglion. When axonal loss is significant, a reduced or absent SNAP should be seen by 10 days after the inciting event. It is of note that although quite helpful, normal SNAPs do not completely exclude a plexopathy. This can be because the lesion may be proximal to the DRG (**Fig. 2**), there may be fascicular predilection for the motor fibers, or a conduction block. Additionally, in some lumbosacral plexopathies, the sensory responses are preserved because they primarily involve the upper lumbar plexus where the sensory studies are less reliable. An alternative explanation is the possible patchy involvement of nerves which spares sensory fascicles. It is dues to this latter situation that diabetic radiculoplexus neuropathy has also been called a "diabetic polyradiculopathy" (given the preservation of sensory responses in many cases).[8] As a result, if an electromyographer finds evidence for a polyradiculopathy on an electrodiagnostic study, an inflammatory plexopathy could still be considered in the differential diagnosis of that patient, depending on the clinical presentation.

Motor Nerve Conduction Studies

The femoral nerve is the only motor nerve conduction study likely to give meaningful information about the possibility of a pure lumbar plexopathy. As seen in **Table 1**, the most common method is to stimulate the nerve high in the inguinal region and record from a quadriceps muscle. Because of significant overlying connective tissue, needle stimulation is often necessary and may be contraindicated in some patients on anticoagulation given the proximity to the femoral artery, but the emphasis on this may be overstated.[9] In this case, side-to-side comparisons may not be as relevant as when using surface electrode recordings.

Peroneal and tibial motor studies are helpful in assessing axonal loss in the lumbosacral plexus; however, in elderly patients, these responses may be diminished or absent because of old age. Also, because the common recording sites are intrinsic foot muscles, patients with superimposed length-dependent peripheral neuropathies may have a reduction in CMAP amplitudes caused by their neuropathy and not by a lumbosacral plexopathy. In these cases, symmetric reductions are expected.

Needle Examination

The needle electromyographic examination may be the most important component of the electrodiagnostic evaluation of lumbosacral plexopathies, both for localization as well as determination of the severity of disease. The importance of a good examination and history cannot be overstated in deciding which muscles to test.

In suspected lumbosacral plexopathies, the planned needle EMG examination should be widespread and cover L2 to S1 innervated muscles as well as muscles innervated by the same root but different peripheral nerves to determine the extent of the abnormalities. Needle examination of the lumbosacral paraspinal muscles should also be performed (**Box 2**).[6,10] It is important that needle EMG evaluation of proximal L2/L3 muscles be conducted, because many providers do not routinely evaluate these muscles.

In lumbosacral plexopathies, one often sees a diffuse reduction of compound muscle and SNAP amplitudes, and needle examination shows neurogenic abnormalities in distal upper and lower limb muscles innervated by multiple lumbosacral roots.

Box 2
A needle examination protocol for lumbosacral plexopathy

To validate a diagnosis of lumbosacral plexopathy, involvement of at least 2 different muscles innervated by 2 different peripheral nerves must be shown:

1. Anterior tibialis (peroneal, L4/5)
2. Posterior tibialis/flexor digitorum longus (tibial, L5)
3. Medial gastrocnemius (tibial, S1/2)
4. Biceps femoris (sciatic, L5/S1)
5. Vastus medialis (femoral, L3/4)
6. Adductor longus (obturator, L2/3)
7. Tensor fascia latae/gluteus medius (inferior gluteal nerve, L5)
8. Gluteus maximums (superior gluteal nerve, S1/2)
9. Low lumbar paraspinals

If a sacral-predominant plexopathy is strongly suspected, consider also testing the anal sphincter and soleus muscles.

The needle examination abnormalities may be more severe in distal versus proximal muscles, with more fibrillation potentials. In these cases, it is tempting to interpret the findings as multiple lumbosacral radiculopathies superimposed on a length-dependent peripheral neuropathy. Although this interpretation at face value is not incorrect, it implies two pathophysiologically and temporally distinct processes. This pattern may be better characterized as a radiculoplexus neuropathy, in the appropriate clinical context.

Causes and Pathogenesis

As with other conditions of peripheral nerves, there are many potential causes of a lumbosacral plexopathy, including inflammatory, neoplastic, structural, and mechanical causes (**Box 3**).[11–15] Probably the most common cause of lumbosacral plexopathy that may mimic a lumbosacral radiculopathy electrophysiologically is diabetic lumbosacral radiculoplexus neuropathy (DLRPN). In these patients, careful history usually reveals an acute to subacute, asymmetrical, focal onset of lower limb pain followed by multifocal weakness, often associated with weight loss.[12] The natural history and underlying pathophysiologic mechanism of DLRPN has long been debated with many different names reflecting multiple views, including diabetic myelopathy,[16] diabetic amyotrophy,[17] Bruns-Garland syndrome,[18] diabetic mononeuritis multiplex,[19] proximal diabetic neuropathy,[20] diabetic lumbosacral plexopathy,[21] diabetic polyradiculopathy,[8] and multifocal diabetic neuropathy.[22] We use the term DLRPN because it emphasizes the frequent association of this syndrome with diabetes mellitus and its sites of involvement (roots, plexus, and nerves). We have studied these patients, as well as a cohort of patients with a similar syndrome without diabetes (lumbosacral radiculoplexus neuropathy [LRPN]) and found that clinically, electrophysiologically, and pathologically they are indistinguishable. Most of these patients have a monophasic course.

Electrophysiologic testing of these patients often shows axonal loss on routine lower limb motor nerve conduction studies, as shown by a reduction in CMAP amplitudes, as well as mild slowing of conduction velocity. This slowing is typically more in

Box 3
Causes of lumbosacral plexopathy

Inflammatory/immune
- Diabetic lumbosacral radiculoplexus neuropathy (microvasculitis)
- Nondiabetic lumbosacral radiculoplexus neuropathy (microvasculitis)
- Sarcoidosis
- Postsurgical inflammatory neuropathy

Neoplastic
- Metastatic diseases
  - Prostate
  - Cervix
  - Colorectum
  - Bladder
- Primary nerve sheath tumor
  - Benign schwannoma
  - Malignant nerve sheath tumor
- Perineurioma
- Lymphoma
- Paraneoplastic
- Amyloidosis

Compressive
- Retroperitoneal hematoma
- Retroperitoneal abscess

Stretch
- After hip or knee surgery

Trauma

Iatrogenic
- Injections
- Strict glucose in new diabetics

Pregnancy and parturition

Idiopathic

keeping with an axonal process as opposed to a demyelinating process, which typically translates to slowing that is greater than 70% of the upper limit of normal when the CMAP amplitude is greater than 50% of normal.[7] SNAP amplitudes are usually also reduced, supporting a postganglionic process.

As previously mentioned, needle EMG examination is often the most useful component of electrodiagnostic testing because of the lack of reliable nerve conduction study of the lumbar plexus. Needle EMG findings shows may be interpreted as multiple lumbosacral radiculopathies superimposed on a length-dependent peripheral neuropathy. This is because inflammatory cases of lumbosacral plexopathy such as

DLRPN (as opposed to a structural cause such as neoplastic infiltration), the pathologic process often involves the roots, plexus, and peripheral nerves simultaneously. Fibrillation potentials and long duration, high amplitude motor unit action potentials are commonly found extending from the lumbar paraspinals to distal leg muscles involving multiple myotomes. Extensive and often contralateral nerve conduction and needle EMG studies are often required to document the extent disease.

Additional testing in these patients can also be useful to help secure a diagnosis including quantitative sensation testing performed using CASE IV[23–25] and quantitative autonomic testing (a measure of postganglionic sudomotor, adrenergic, and cardiovagal function).[22] In a recent study of 17 DLRPN patients who underwent CASE IV testing, panmodality (small-fiber and large-fiber sensation) abnormalities were identified. Fourteen patients underwent autonomic testing (8 had clinical symptoms) all with abnormalities, 8 of which were severe.[5] Given these findings, additional testing of small and autonomic fibers is warranted when there is suspicion of a lumbosacral plexopathy, because these abnormalities would not be expected in radiculopathies, or other inflammatory polyradiculopathies.[26]

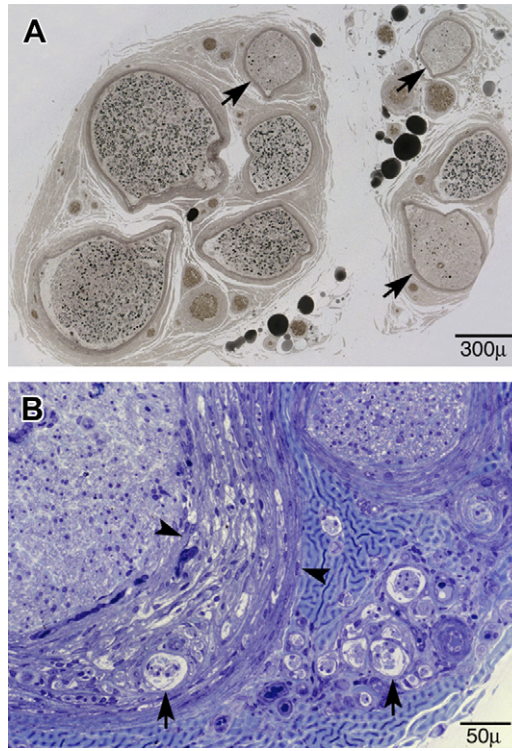

Fig. 3. Transverse semithin epoxy sections of sural nerves from patients with DLRPN stained with paraphenylenediamine (*A*) And methylene blue (*B*) Showing (1) multifocal fiber loss in which 3 fascicles (*A*, *arrows*) have almost no fibers, and the adjacent fibers are less affected; and (2) abortive repair with injury neuroma (*B*, *arrows*) and thickened perineurium (*B*, *between arrowheads*). These findings are typical for ischemic injury and repair and were commonly seen in both DLRPN an LRPN. (*From* Dyck PJB. Radiculoplexus neuropathies: diabetic and nondiabetic varieties. In: Dyck PJ, Thomas PK, eds. Peripheral Neuropathy. 4th ed. Vol. 2. Philadelphia: Elsevier; 2004; with permission.)

In the past, it has been suggested that the pathophysiologic basis of disease in diabetic patients who present with rapid asymmetrical plexopathies is caused by ischemic injury, which we and others have confirmed in DLRPN.[5,22,24] In our prospective series of 33 patients with DLRPN, distal cutaneous nerve biopsy samples in affected patients showed characteristic ischemic findings of multifocal fiber loss, perineurial degeneration or thickening, neovascularization, and abortive regeneration of nerve fibers forming microfasciculi (ie, an injury neuroma) (**Fig. 3**). We were able to compare these nerve samples with nerves of patients with diabetic peripheral neuropathy (DPN), and those with DLRPN showed significantly more ischemic changes. Axonal enlargements were also noted on transverse nerve sections; these were similar to enlargements described by others in experimental ischemia and were probably caused by accumulated organelles.[12,27,28] Teased nerve fiber evaluation showed increased rates of axonal degeneration and empty nerve strands compared with normal controls as well as DPN. In our recent prospective series, there were inflammatory infiltrates in all nerve biopsy samples. Inflammation involving the vessel walls suggesting microvasculitis was seen in half of the cases and, in several, diagnostic changes confirming microvasculitis were noted (**Fig. 4**).[15]

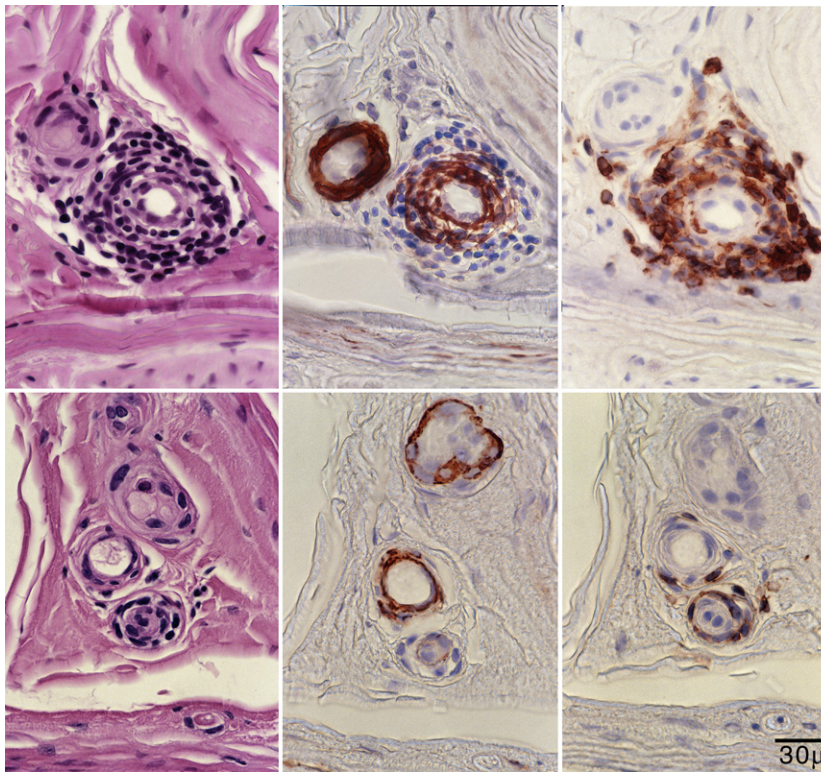

Fig. 4. Serial skip paraffin sections from a patient with painful DLRPN. Left panels are stained with hematoxylin and eosin, middle panels are stained with anti–smooth muscle actin, and the right panels reacted with leukocyte common antigen (CD45). The upper row shows fragmentation of the tunica media of the microvessel with mononuclear cells that show focal microvasculitis seen in DLRPN. (*From* Dyck PJB. Radiculoplexus neuropathies: diabetic and nondiabetic varieties. In: Dyck PJ, Thomas PK, eds. Peripheral Neuropathy. 4th ed. Vol. 2. Philadelphia: Elsevier; 2004; with permission.)

Symptomatic treatment in the form of narcotics in combination with neuropathic agents for pain as well as physical therapy should be considered at recognition of the condition. Patients need to be reassured that these inflammatory DRPN almost always seem to be monophasic in nature and, although complete resolution may not be seen, with time, meaningful improvement can usually be expected.

PITFALLS

Although electrodiagnostic testing is essential in the evaluation of a patient with a suspected lumbosacral plexopathy, it has limitations. For instance, in the case of a focal process such as a benign, slow-growing mass, the needle EMG findings may be limited to the distribution of only 1 nerve or 1 nerve root distribution, thereby failing to meet the definition of plexopathy (ie, the involvement of 2 different nerves and nerve roots in the lower extremity). However, if a high index of suspicion still exists, magnetic resonance imaging may be helpful to confirm a focal lesion. If the pathologic process is a lumbar radiculoplexus neuropathy, as discussed earlier, paraspinal denervation and long duration, high amplitude motor unit potentials on needle EMG are common.[5,13,29] The diagnosis of lumbosacral radiculoplexus neuropathy should also always be considered in patients presenting with what seems to be multiple radiculopathies superimposed on a length-dependent peripheral neuropathy, especially with a history of unilateral or asymmetrical onset of a painful, acute to subacute process associated with weight loss and autonomic derangements.

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