

Role of electromyography and ultrasonography in the diagnosis of double crush lumbar radiculopathy and common fibular injury: illustrative cases

Lisa B. E. Shields, MD,¹ Vasudeva G. Iyer, MD,² John E. Harpring, MD,¹ Abigail J. Rao, MD,¹ Yi Ping Zhang, MD,¹ and Christopher B. Shields, MD^{1,3}

¹Norton Neuroscience Institute, Norton Healthcare, Louisville, Kentucky; ²Neurodiagnostic Center of Louisville, Louisville, Kentucky; and ³Department of Neurological Surgery, University of Louisville School of Medicine, Louisville, Kentucky

BACKGROUND Double crush syndrome consists of two compression sites along a peripheral nerve and is rare in the lower extremities. Electrodiagnostic and ultrasound (US) studies may be helpful in evaluating foot drop involving overlapping pathologies.

OBSERVATIONS Case 1 involved a man who presented with left dorsiflexor weakness and left foot numbness. Electromyography (EMG) revealed a left common fibular nerve entrapment neuropathy and left L5 radiculopathy. US and magnetic resonance imaging (MRI) revealed a large cystic lesion of the left common fibular nerve treated by cyst removal. The left foot drop persisted postoperatively. Lumbar computed tomography myelography revealed severe left foraminal stenosis at L5–S1. Multilevel lumbar laminectomies and facetectomies with an L5–S1 fusion were performed. Within 1 month postoperatively, the left foot drop had improved. Case 2 involved a man who developed a right foot drop caused by right lumbar foraminal stenosis at L4–5 and L5–S1. EMG and US of the right common fibular neuropathy showed large fascicles involving the right common fibular nerve. MRI revealed a hyperintense signal of the right common fibular nerve. Spontaneous improvement occurred within 6 months without surgery.

LESSONS Spine surgeons should recognize double crush in the lower extremities. EMG and US are valuable in detecting peripheral nerve abnormalities, especially in cases with overlapping lumbar pathology.

<https://thejns.org/doi/abs/10.3171/CASE21566>

KEYWORDS neurosurgery; double crush; lumbar; common peroneal nerve; common fibular nerve; electromyography; nerve conduction study; ultrasound

Double crush syndrome refers to two compression sites along a peripheral nerve.¹ Initially described by Upton and McComas in 1973, double crush syndrome is thought to be due to asymptomatic compression at one site that predisposes a peripheral nerve to increased susceptibility to impairment at another anatomical location.^{1,2} This condition results in impaired axoplasmic flow along the nerve that causes axons at a different site to be more vulnerable to compression syndromes. Double crush syndrome may be considered when a patient's condition fails to improve after decompression at one site due to continued compression at another site along a peripheral nerve. Most commonly described in the setting of concurrent cervical radiculopathy and carpal tunnel syndrome,^{1,3–6} double crush syndrome may also

develop in association with underlying systemic diseases such as diabetes mellitus, drug-induced neuropathy, vascular and thyroid diseases, excessive use of alcohol, rheumatoid arthritis, chemotherapeutic agents, and obesity.^{1,5–7} Double crush syndrome involving lumbar radiculopathy and the fibular nerve has rarely been reported.^{8–10}

Both of these conditions may cause weakness in the form of foot drop.

Four mechanisms have been postulated to contribute to the underlying development of double crush syndrome: (1) loss of axonal transport, (2) focal immune response inflammation of the dorsal root ganglia, (3) ion channel dysregulation, and (4) neuroma in continuity.¹¹ Nutrient flow is impaired in both the antegrade and retrograde directions along the axon.¹ The same pathophysiology may be applied to any nerve.

ABBREVIATIONS BMI = body mass index; CT = computed tomography; EDB = extensor digitorum brevis; EMG = electromyography; MRI = magnetic resonance imaging; NCV = nerve conduction velocity; PL = peroneus longus; TA = tibialis anterior; TFL = tensor fascia lata; TP = tibialis posterior; US = ultrasound.

INCLUDE WHEN CITING Published April 18, 2022; DOI: 10.3171/CASE21566.

SUBMITTED October 1, 2021. **ACCEPTED** March 9, 2022.

© 2022 The authors, CC BY-NC-ND 4.0 (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

We present two cases of double crush lumbar radiculopathy and common fibular nerve entrapment. The importance of electromyography and nerve conduction velocity (EMG/NCV) and ultrasound (US) studies in confirming this condition is discussed. The radiological findings, treatment, and prognosis are also highlighted.

Illustrative Cases

Case 1

History and Radiological Imaging

A 52-year-old man (body mass index [BMI] 32.77 kg/m²) was involved in a motor vehicle accident 4 months after lumbar spine surgery that precipitated a recurrence of his low back pain, left leg numbness along the L5 and S1 distribution, and left foot drop. Lumbar magnetic resonance imaging (MRI) without gadolinium contrast showed moderate stenosis with severe facet hypertrophy at L3–4, moderate stenosis at L4–5, and a severe collapsed degenerated disc, modic changes, and severe facet hypertrophy and foraminal stenosis at L5–S1 on the left with L5 nerve root compression. Lumbar computed tomography (CT) myelography confirmed the presence of severe left foraminal stenosis at L5–S1 (Fig. 1A–C).

Physical Examination

The dorsiflexors and evertors of the left ankle were weak (0/5 strength), and there was mild weakness (4/5) of the invertor of the left ankle. Decreased pinprick sensation was noted over the dorsum of the left foot and the lateral aspect of the left leg, suggesting lumbar radiculopathy. Palpation of the common fibular nerve at the left fibular neck revealed a mass with an inconsistent Tinel sign.

EMG/NCV of the Legs and US of the Left Common Fibular Nerve

Stimulation of the left common fibular nerve did not evoke compound muscle action potentials over the extensor digitorum brevis (EDB), the tibialis anterior (TA), or the peroneus longus (PL). No sensory potentials could be recorded over the superficial fibular nerve on the left. Needle EMG revealed denervation of the left TA and PL. There were increased polyphasic units of the tibialis posterior (TP) and tensor fascia lata (TFL). US showed a large cystic lesion in the vicinity of the left common fibular nerve (Supplementary Fig. 1).

These findings indicated the presence of severe left common fibular nerve neuropathy causing total denervation of the muscle innervated by the superficial and deep fibular branches. The increase in polyphasic units without fibrillations in the left TP and TFL indicated L5 radiculopathy.

Left Knee MRI and Common Fibular Nerve Surgery

A left knee MRI scan without gadolinium contrast revealed a multilocular intraneural ganglion cyst measuring 7.0 × 4.0 × 1.5 cm in close contact with and surrounding the common fibular nerve above, at, and below the left fibular head (Fig. 2A–F). The ganglion cysts arose from the anterior aspect of the proximal tibiofibular joint. Proximal to these cysts, the common fibular nerve was abnormally enlarged and hyperintense, compatible with neuropathy/neuritis. There was muscle atrophy and an abnormal T2 hyperintensity within the muscles of the anterior compartment of the left lower leg, compatible with denervation. The patient underwent dissection of the left common fibular nerve with drainage of multiple large intraneural ganglion cysts (Fig. 3A and B).

Follow-Up

The patient experienced minimal improvement of the left foot drop. Ten weeks after the common fibular nerve surgery, left foot dorsiflexion strength was grade 1/5, and left extensor hallucis longus strength was grade 0/5. Because the left foot drop persisted, the patient underwent bilateral L3–S1 laminectomies and left L4–5 and L5–S1 medial facetectomies and foraminotomies as well as L5–S1 fusion. Within 1 month after the lumbar spine surgery, both his left foot drop and gait showed significant improvement.

Case 2

History and Radiological Imaging

A 62-year-old man (BMI 23.39 kg/m²) reported a 3-month history of right foot drop. He denied numbness or pain distal to the right knee, although he had a 20-year history of low back pain. The patient was treated with an ankle and foot orthosis brace and physical therapy, which led to gradual improvement of his strength. The patient also underwent a suboccipital craniotomy for resection of a left intraparenchymal cerebellar tumor (metastatic pulmonary adenocarcinoma) 4 months before the onset of the right foot drop. The patient completed immunotherapy and total brain radiation therapy.

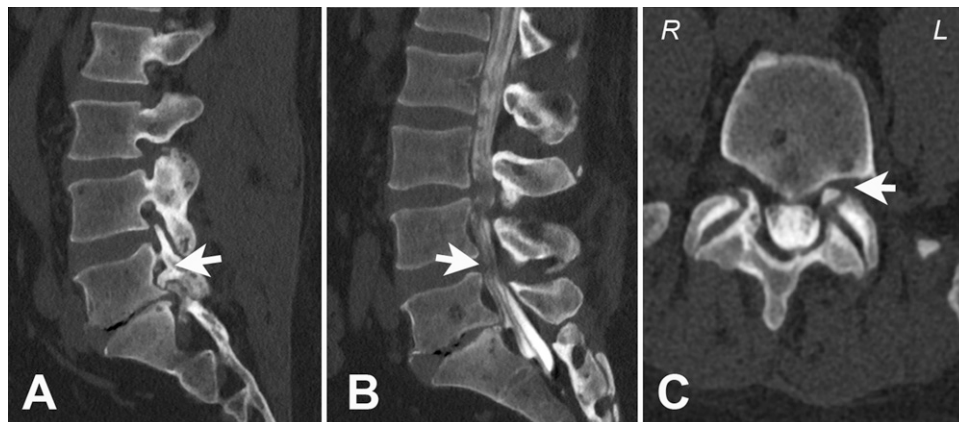


FIG. 1. A: Sagittal lumbar CT myelogram revealing severe left foraminal stenosis (arrow) compressing the L5 nerve root. **B:** Midsagittal view showing severe central spinal stenosis (arrow) at L4–5. **C:** Axial view revealed severe left foraminal stenosis (arrow).

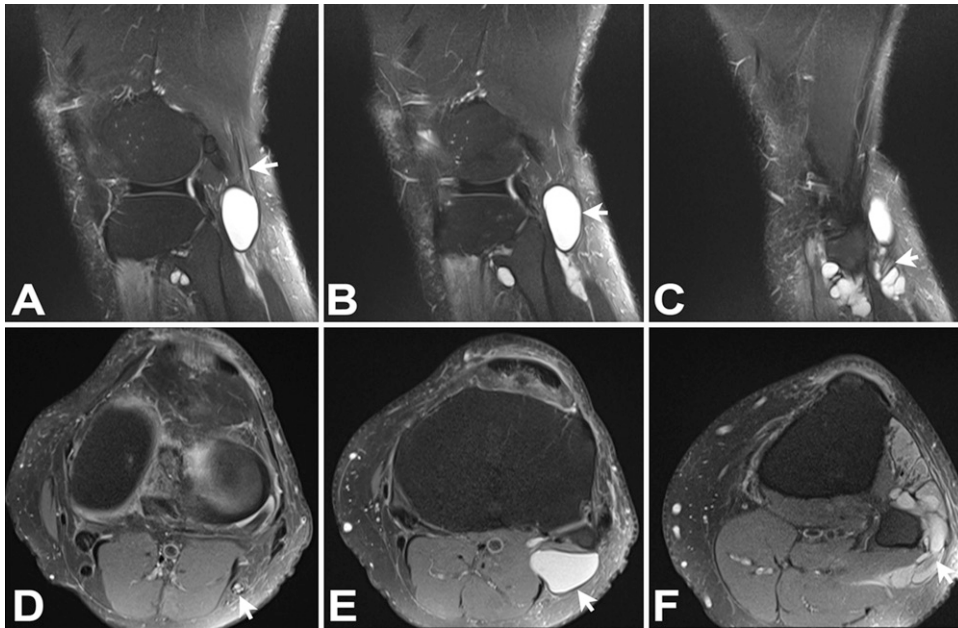


FIG. 2. A–C: Sagittal views of the left knee showing the common fibular nerve proximal to the cyst (A, arrow), the cyst (B, arrow), and the common fibular nerve distal to the cyst (C, arrow). **D–F:** Distal views of the left knee showing the common fibular nerve proximal to the cyst (D, arrow), the cyst (E, arrow), and the common fibular nerve distal to the cyst (F, arrow). (C) and (F) show that there are multiple additional cysts arising from the tibiofibular joint compressing the common fibular nerve.

A lumbar MRI scan with and without gadolinium contrast revealed moderate to severe lumbar spondylosis and severe foraminal stenosis at L4–5 and L5–S1 (right greater than left at both levels) (Fig. 4A and B).

Physical Examination

Marked weakness (0/5) and atrophy of the right TA and PL were noted.

EMG/NCV of the Legs and Right Knee MRI

Stimulation of the right common fibular nerve evoked a small-amplitude response over the EDB. The conduction velocity was slow across the right knee (Supplementary Fig. 2). Sensory potentials could be recorded over the right superficial fibula, with a lower amplitude on the right than on the left. Denervation changes in the right TA and PL were observed, with a single polyphasic

motor unit in the TA and one to two motor units in the PL. The TP and TFL showed increased polyphasic units. US showed large nerve fascicles in the right common fibular nerve. The EMG/NCV and US studies confirmed severe right common fibular nerve neuropathy at the fibular neck. The right TP and TFL abnormalities suggested L5 radiculopathy.

MRI of the right knee with and without gadolinium contrast showed a hyperintense nonspecific signal within the right common fibular nerve at the fibular neck. Fatty atrophy and denervation edema involving the anterior compartment muscles of the right lower leg were observed, suggestive of right fibular neuropathy. There was no intrinsic mass or enhancement and no extrinsic compressing mass lesion, ganglion cyst, or fluid collection of the right common fibular nerve.

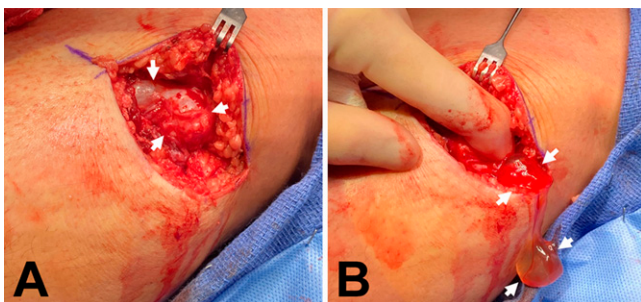


FIG. 3. A: Intraoperative cyst (arrows) corresponding to the large cyst seen on the MRI of the left knee. **B:** Opening and draining of the cyst demonstrate the two colloid-like cysts (arrows) that have been expressed from the lesion seen in (A).

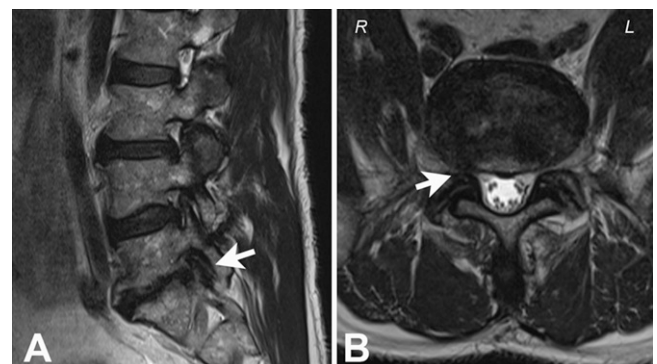


FIG. 4. A: Lumbar MRI (sagittal view) that revealed severe right foraminal stenosis (arrow) compressing the L5 nerve root. **B:** Axial view showing severe right foraminal stenosis (arrow).

Follow-Up

The patient experienced significant and spontaneous improvement of the right ankle dorsiflexion function within 6 months of onset; thus, a fibular nerve decompression or transfer surgery was not performed. Because the patient had undergone extensive treatment for his metastatic pulmonary adenocarcinoma and his right foot drop had resolved, lumbar intervention was not performed.

Discussion

Different scenarios of double crush syndrome have been reported in the lumbar area and lower extremities (Table 1).^{8–10,12–20} Two case reports and one study reported double crush lumbar radiculopathy and fibular nerve entrapment after failed back surgery.^{8–10} Reife and Coulis¹⁰ reported a case of a woman with a complaint of left leg pain who underwent left L5–S1 hemilaminectomy and discectomy at L5–S1. The left lateral calf pain increased postoperatively. Three weeks later, she underwent a second operation to repair a cerebrospinal fluid leak and remove a recurrent extruded disc at L5–S1. The symptoms continued postoperatively. Palpation of the left lateral leg inferior to the fibular head was tender. There was also grade 4 dorsiflexor weakness of the left foot and toes. Her symptoms resolved 2 months later after she was advised to stop crossing her legs. Ang and Foo⁸ reported a case of a man with low back pain and left leg pain/paresthesia who underwent a L4–5 and L5–S1 lateral recess decompression and discectomy without resolution of his symptoms postoperatively. Because a left calf MRI scan showed a peroneal muscle herniation compressing the superficial fibular nerve, the patient underwent surgical decompression of this nerve. Two pseudoneuromas of the superficial fibular nerve were identified, reflecting three locations of nerve entrapment, all of which were present at the patient's initial presentation. The symptoms significantly improved within 3 months after the second operation. Similar to the patient in the Reife and Coulis¹⁰ case report, this patient did not undergo electrodiagnostic tests either before or after the lumbar surgery.

In a study of 300 patients who underwent lumbar discectomies, 3 patients described pain of the lateral aspect of the leg postoperatively having a lower-grade intensity and different temporal and spatial patterns than preoperatively.⁹ Exquisite tender points were noted behind the femoral biceps tendon in these three cases. These authors attributed the new postoperative leg pain to entrapment of the fibular nerve crural branches, which had been masked by the root compression symptoms that became evident after lumbar surgery. Neurolysis was performed on the fibular nerve crural branches in all three cases with resolution of symptoms.

Clinical examination can distinguish between L5 radiculopathy and fibular nerve palsy by documenting weak ankle invertors in L5 radiculopathy but not in fibular nerve palsy. However, when double crush syndrome occurs, this clue is lost because the invertors are weak in both L5 radiculopathy and double crush syndrome. In double crush lumbar and common fibular nerve injuries, lumbar imaging studies (MRI, CT myelogram) may reveal lumbar pathology such as a disc herniation or foraminal stenosis, whereas electrodiagnostic and US testing is usually able to localize the common fibular nerve lesion. EMG/NCV tests can distinguish between an L5 root and fibular nerve lesion, with sensory potentials that are intact in an L5 radiculopathy across the knee and absent in a fibular nerve lesion. EMG/NCV studies are also beneficial when a patient's condition does not improve within a reasonable interval after treatment, when overlapping symptoms such as pain of the lateral aspect of the leg and foot drop are present, and in situations of equivocal MRI findings.²¹ However, loss of both motor and sensory responses on stimulation of the fibular nerve causes a diagnostic challenge in assessing concomitant L5 radiculopathy.

High-resolution US is valuable in identifying structural lesions such as an intraneural ganglion or inflammatory changes and to confirm the lesion site when electrodiagnostic testing is inconclusive.^{22,23} Although US has not been reported in the diagnostic evaluation of double crush lumbar and common fibular injury, it has been described as being valuable in diagnosing double crush ulnar nerve injury by showing enlargement of the ulnar nerve at the cubital tunnel and

TABLE 1. Lumbar and/or lower extremity locations of double crush syndrome

Authors & Year	Features
Giannoudis et al., 2005 ¹⁴	Acetabular fractures with injuries of sciatic nerve proximally and peroneal nerve distally at the fibular neck
Chodoroff & Ball, 1985 ¹³ Golovchinsky, 1998 ¹⁵ Zheng et al., 2016 ²⁰	Lumbosacral radiculopathy and tarsal tunnel syndrome
Augustijn & Vanneste, 1992 ¹²	Posterior tibial nerve compressed under flexor retinaculum (tarsal tunnel syndrome)
Kanamoto et al., 2016 ¹⁶	Lumbar nerve is compressed both medially and laterally in the spinal canal
Ang & Foo, 2014 ⁸	Lumbar radiculopathy and superficial peroneal nerve entrapments at two separate locations
Reife & Coulis, 2013 ¹⁰	Lumbar radiculopathy and peroneal nerve
Crotti et al., 2005 ⁹	Lumbar radiculopathy and peroneal nerve crural branches
Wu et al., 2020 ¹⁸	Simultaneous lumbar foraminal and/or extraforaminal stenosis
Yamada et al., 2011 ¹⁹	Fifth lumbar spinal nerve (compression at two or more sites from intraspinal to extraforaminal zone; failed back surgery)
Nishimura et al., 2020 ¹⁷	Lumbosacral epidermoid tumor and sacral Tarlov cyst at S2

within the flexor carpi ulnaris muscle.²⁴ Knee MRI is useful in visualizing the fibular nerve by detecting lesions caused by trauma, ganglia, peripheral nerve sheath tumors, and osteochondroma.^{25,26}

Treatment for double crush syndrome is based on the severity of compression and appropriate symptoms at each site. If significant symptoms persist after decompression at one site, then subsequent decompression should be performed at the second site. Nerve transfer surgery is an innovative option to reinnervate the TA muscle in patients who have sustained a profound, irreversible fibular nerve injury.^{27,28} This surgery involves a nerve transfer of either the superficial fibular nerve or tibial nerve fascicles to the motor branch of the TA and to the deep fibular nerve to obtain improvement in ankle dorsiflexion and eversion.

Observations

The two cases presented are the first to describe double crush involving a lumbar and common fibular injury diagnosed through both electrodiagnostic and US studies. A high level of suspicion of double crush is justified in patients who present with low back pain and symptoms involving L5 radiculopathy who do not experience resolution of muscle weakness after lumbar nerve root decompression. Performing EMG/NCV and US before lumbar surgical intervention may prove invaluable in cases of double crush to rule out peripheral nerve abnormalities and to potentially avoid unnecessary back surgery. Knee MRI may be beneficial in revealing a multilocular intraneural ganglion cyst surrounding the common fibular nerve (case 1) and revealing a hyperintense signal within the common fibular nerve and fatty atrophy and denervation edema in the anterior compartment muscles of the lower leg (case 2).

In case 1, the patient demonstrated improvement of his symptoms, including the foot drop, after lumbar surgery, suggesting that his symptoms were due to lumbar pathology. Despite severe lumbar spondylosis and foraminal stenosis in case 2, the patient's EMG/NCV and US studies detected a severe right common fibular nerve neuropathy, suggesting that the peripheral neuropathy was the primary cause of the foot drop.

Lessons

Spine surgeons should be aware of the lower extremity double crush syndrome caused by occurrence of lumbar radiculopathy and common fibular nerve entrapment neuropathy, which poses several diagnostic and therapeutic challenges. Electrodiagnostic studies play an essential role, especially in patients presenting with overlapping lumbar radiculopathy and entrapment neuropathy in the lower extremities. US is also helpful when EMG/NCV tests are nondiagnostic in situations in which no sensory or motor responses are elicited from the legs.

Acknowledgments

We acknowledge Norton Healthcare for their continued support.

References

1. Kane PM, Daniels AH, Akelman E. Double crush syndrome. *J Am Acad Orthop Surg.* 2015;23(9):558–562.
2. Upton AR, McComas AJ. The double crush in nerve entrapment syndromes. *Lancet.* 1973;2(7825):359–362.
3. Hurst LC, Weissberg D, Carroll RE. The relationship of the double crush to carpal tunnel syndrome (an analysis of 1,000 cases of carpal tunnel syndrome). *J Hand Surg Br.* 1985;10(2):202–204.

4. Lo SF, Chou LW, Meng NH, et al. Clinical characteristics and electrodiagnostic features in patients with carpal tunnel syndrome, double crush syndrome, and cervical radiculopathy. *Rheumatol Int.* 2012;32(5):1257–1263.
5. Molinari WJ 3rd, Elfar JC. The double crush syndrome. *J Hand Surg Am.* 2013;38(4):799–801.
6. Zahir KS, Zahir FS, Thomas JG, Dudrick SJ. The double-crush phenomenon – an unusual presentation and literature review. *Conn Med.* 1999;63(9):535–538.
7. Cohen BH, Gaspar MP, Daniels AH, Akelman E, Kane PM. Multifocal neuropathy: expanding the scope of double crush syndrome. *J Hand Surg Am.* 2016;41(12):1171–1175.
8. Ang CL, Foo LS. Multiple locations of nerve compression: an unusual cause of persistent lower limb paresthesia. *J Foot Ankle Surg.* 2014;53(6):763–767.
9. Crotti FM, Carai A, Carai M, Sgaramella E, Sias W. Entrapment of crural branches of the common peroneal nerve. *Acta Neurochir Suppl (Wien).* 2005;92:69–70.
10. Reife MD, Coulis CM. Peroneal neuropathy misdiagnosed as L5 radiculopathy: a case report. *Chiropr Man Therap.* 2013;21(1):12.
11. Schmid AB, Coppieters MW. The double crush syndrome revisited – a Delphi study to reveal current expert views on mechanisms underlying dual nerve disorders. *Man Ther.* 2011;16(6):557–562.
12. Augustijn P, Vanneste J. The tarsal tunnel syndrome after a proximal lesion. *J Neurol Neurosurg Psychiatry.* 1992;55(1):65–67.
13. Chodoroff B, Ball RD. Lumbosacral radiculopathy, reflex sympathetic dystrophy and tarsal tunnel syndrome: an unusual presentation. *Arch Phys Med Rehabil.* 1985;66(3):185–187.
14. Giannoudis PV, Da Costa AA, Raman R, Mohamed AK, Smith RM. Double-crush syndrome after acetabular fractures. A sign of poor prognosis. *J Bone Joint Surg Br.* 2005;87(3):401–407.
15. Golovchinsky V. Double crush syndrome in lower extremities. *Electromyogr Clin Neurophysiol.* 1998;38(2):115–120.
16. Kanamoto H, Eguchi Y, Suzuki M, et al. The diagnosis of double-crush lesion in the L5 lumbar nerve using diffusion tensor imaging. *Spine J.* 2016;16(3):315–321.
17. Nishimura Y, Hara M, Awaya T, et al. Possible double crush syndrome caused by iatrogenic acquired lumbosacral epidermoid tumor and concomitant sacral Tarlov cyst. *NMC Case Rep J.* 2020;7(4):195–199.
18. Wu PH, Kim HS, Jang IT. How I do it? Uniportal full endoscopic contralateral approach for lumbar foraminal stenosis with double crush syndrome. *Acta Neurochir (Wien).* 2020;162(2):305–310.
19. Yamada H, Yoshida M, Hashizume H, et al. The double-crush syndrome of the 5th lumbar spinal nerve as a cause of failed back surgery [abstract]. *Spine.* 2011(ISSLS Meeting Abstracts):GP132.
20. Zheng C, Zhu Y, Jiang J, et al. The prevalence of tarsal tunnel syndrome in patients with lumbosacral radiculopathy. *Eur Spine J.* 2016;25(3):895–905.
21. Fridman V, David WS. Electrodiagnostic evaluation of lower extremity mononeuropathies. *Neurol Clin.* 2012;30(2):505–528.
22. Iyer VG. Role of ultrasound in the EMG lab. *Open Access J Neurol Neurosurg.* 2021;15:OAJNN.MS.ID.555910.
23. Nodera H, Sato K, Terasawa Y, Takamatsu N, Kaji R. High-resolution sonography detects inflammatory changes in vasculitic neuropathy. *Muscle Nerve.* 2006;34(3):380–381.
24. Akyuz M, Yalcin E, Selcuk B, Onder B, Ozçakar L. Electromyography and ultrasonography in the diagnosis of a rare double-crush ulnar nerve injury. *Arch Phys Med Rehabil.* 2011;92(11):1914–1916.
25. Shields LBE, Iyer VG, Shields CB, Zhang YP, Rao AJ. Varied presentation and importance of MR neurography of the common fibular nerve in slimmer's paralysis. *Case Rep Neurol.* 2021;13(2):555–564.
26. Van den Bergh FR, Vanhoenacker FM, De Smet E, Huisse W, Verstraete KL. Peroneal nerve: normal anatomy and pathologic

findings on routine MRI of the knee. *Insights Imaging*. 2013;4(3): 287–299.

27. Giuffre JL, Bishop AT, Spinner RJ, Levy BA, Shin AY. Partial tibial nerve transfer to the tibialis anterior motor branch to treat peroneal nerve injury after knee trauma. *Clin Orthop Relat Res*. 2012;470(3): 779–790.
28. Nath RK, Somasundaram C. Gait improvements after peroneal or tibial nerve transfer in patients with foot drop: a retrospective study. *Eplasty*. 2017;17:e31.

Disclosures

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author Contributions

Conception and design: CB Shields, LBE Shields, Iyer, Harpring.
Acquisition of data: CB Shields, LBE Shields, Iyer, Harpring. Analysis and

interpretation of data: all authors. Drafting the article: LBE Shields. Critically revising the article: CB Shields, LBE Shields, Iyer, Harpring, Rao.
Reviewed submitted version of manuscript: all authors. Approved the final version of the manuscript on behalf of all authors: CB Shields.
Administrative/technical/material support: CB Shields, Study supervision: CB Shields, LBE Shields.

Supplemental Information

Online-Only Content

Supplemental material is available with the online version of the article.

Supplementary Figs. 1 and 2. <https://thejns.org/doi/suppl/10.3171/CASE21566>.

Correspondence

Christopher B. Shields: Norton Neuroscience Institute, Norton Healthcare, Louisville, KY. cbshields1@gmail.com.