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Multiple Locations of Nerve Compression: An Unusual Cause of Persistent Lower Limb Paresthesia

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ABSTRACT

A paucity of appreciation exists that the "double crush" phenomenon can account for persistent leg symptoms even after spinal neural decompression surgery. We present an unusual case of multiple locations of nerve compression causing persistent lower limb paresthesia in a 40-year old male patient. The patient's lower limb paresthesia was persistent after an initial spinal surgery to treat spinal lateral recess stenosis thought to be responsible for the symptoms. It was later discovered that he had peroneal muscle herniations that had caused superficial peroneal nerve entrapments at 2 separate locations. The patient obtained much symptomatic relief after decompression of the peripheral nerve. The "double crush" phenomenon and multiple levels of nerve compression should be considered when evaluating lower limb neurogenic symptoms, especially after spinal nerve root surgery.

The "double crush" phenomenon was first described by Upton and McComas (1) in 1973 based on their observation that some patients with ulnar or median peripheral neuropathy had associated cervicothoracic root lesions. To this day, the exact pathogenesis behind why a primary nerve lesion predisposes to another along the same nerve remains elusive (2). Although the term has since been applied to the description of 2 or more separate neurologic lesions along a peripheral nerve (3–6), much of the available data have concentrated on the associations between upper limb compressive neuropathy (in particular, carpal tunnel syndrome) and cervical radiculopathy (7–12). It has also been reported that the most common cause of double crush is an underlying neuropathy such as diabetes, with the metabolic neuropathy serving as the "first crush" rather than spinal nerve root compression (13).

When it comes to the lower limbs, few similar studies have been published. One report described peroneal nerve entrapments in patients with failed back surgery syndrome who also had myofascial pain syndrome (14). Also reports have been published of isolated superficial peroneal nerve entrapments (15,16). We believe a paucity of appreciation exists that the "double crush" phenomenon (or even multiple crush) can also occur in the lower limb. We present the case report of a patient with leg paresthesia and pain who had initially undergone back surgery for a radicular pathologic entity, only to have his symptoms persist. He was later also found to have peripheral superficial peroneal nerve entrapment at not 1, but 2 separate locations.

Case Report

The male patient presented to our institution in the summer of 2008 at 40 years of age with symptoms of low back pain and left lower limb radicular pain and paresthesia down to his posterolateral calf and sole. The symptoms were aggravated by standing and ambulation. The clinical examination revealed signs of L5 and S1 nerve root pathologic features. Magnetic resonance imaging (MRI) of his lumbar spine revealed L4-L5 and L5-S1 degenerative disc disease with left-sided disc protrusions causing lateral recess stenosis (Fig. 1). Neurodiagnostic tests were not performed at that point. The patient underwent L4-L5 and L5-S1 lateral recess decompression and discectomy in August 2008.

Postoperatively, the patient's symptoms over his sole had resolved; however, the paresthesia over his lateral calf persisted, and his back pain was similar to that preoperatively. The patient continued to have persistent left lateral calf paresthesia 18 months after his spinal surgery. A repeat MRI scan was performed, which showed recurrent L4-L5 and L5-S1 disc protrusions causing stenosis of the lateral recess again (Fig. 1). The option of spinal fusion surgery was discussed, but the patient declined this. Again, neurodiagnostic tests were not ordered for the patient.

At 24 months after his initial spinal surgery, the patient brought our attention to a mass over his left lateral calf that he had recently discovered. The location of the mass was at the junction of the

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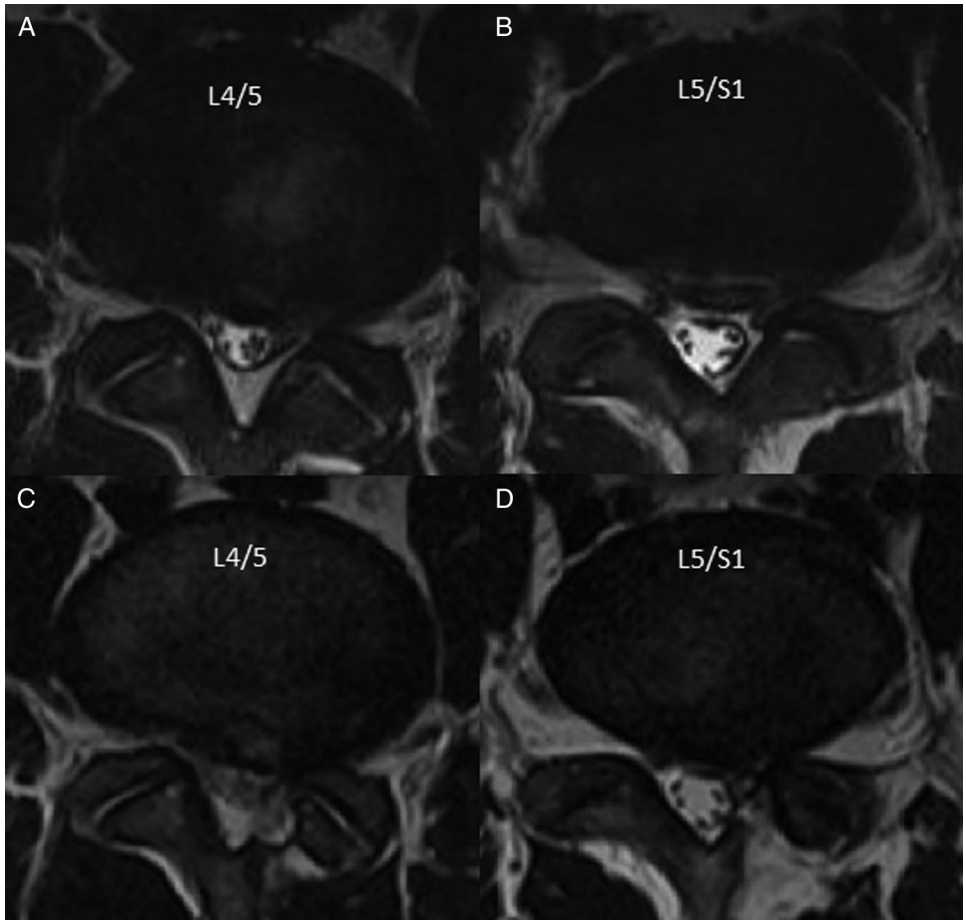

Fig. 1. Lumbar spine magnetic resonance imaging scan in 2008 (*A* and *B*) and 2010 (*C* and *D*). Both L4-L5 and L5-S1 segments show left-sided lateral recess stenosis with impingement of the L5 and S1 nerve roots. The 2010 images showed recurrence of the prolapsed discs, causing stenosis of the lateral recess again.

proximal two thirds and distal one third of the lateral side of the calf. It was firm and tender and had a positive Tinel's sign. The area of paresthesia, however, started more proximally, from the mid-lateral calf to just superior to the lateral malleolus. An MRI scan of his left calf was performed and showed peroneal muscle herniation through the opening in the deep fascia, where the superficial peroneal nerve traversed from deep to superficial, compressing the nerve (Fig. 2). Reviewing the patient's history showed that the patient jogged for leisure, but he had no history of any injury to his calf.

The patient underwent open surgical decompression of his superficial peroneal nerve in September 2011. A longitudinal incision was made over the lateral calf (Fig. 3). The superficial peroneal nerve was identified distally and traced back to the point at which it passed from deep to superficial through the fascia. A fasciotomy was performed to adequately decompress the nerve. A pseudo-neuroma was identified at this point of nerve compression. Tracing the nerve further proximally revealed a second pseudo-neuroma where a branch of the nerve also traversed the deep fascia. The location of this second neuroma corresponded to the proximal limit of the area of paresthesia that the patient was experiencing. At both locations, peroneal muscle herniation was present, with fatty overgrowth through the fascia openings causing the pseudo-neuromas. An adequate fasciotomy was performed, as shown in Fig. 3, and layered closure of the wound was performed. The patient was allowed to weight bear as tolerated immediately after the surgery, and his wound had healed well by 2 weeks postoperatively.

The patient was initially examined in the clinic 3 months after surgery. His left lateral calf paresthesia had improved markedly, although not yet completely. At the 12-month follow-up visit in the clinic, he no longer had paresthesia or pain over the left calf, and the patient had returned to his full function in both work and sports. He was remarkably satisfied with the treatment outcome.

Discussion

Our report highlights the phenomenon of multiple locations of nerve compression causing persistent paresthesia in the lower limb. After the initial spinal surgery, it was 2 years before a separate superficial peroneal nerve entrapment was diagnosed. Furthermore, it was only intraoperatively that a third location of nerve entrapment was discovered. It is highly likely that the multiple pathologic locations were already present at the initial presentation, thus accounting for the persistent symptoms over the lateral calf even after the spinal surgery.

It is perhaps not uncommon for lower limb neurogenic symptoms to persist or to have obtained only partial relief after lumbar spine decompression surgery. The surgeon often attributes this to the chronicity of the nerve root impingement and the possibility of adjacent level pathologic features. Our patient is a typical example. When his left lateral calf paresthesia failed to improve, the index of suspicion for a separate pathologic entity was not aroused. The subsequent MRI findings of recurrent nerve root compression at the L4-L5 level reinforced the diagnosis of a recurrent spine problem.