Superficial peroneal nerve syndrome: an unusual nerve entrapment

Case report

LYNDA J. S. YANG, M.D., PH.D., VISHAL C. GALA, M.D., M.P.H., AND JOHN E. MCGILLCUDDY, M.D.

Department of Neurosurgery, University of Michigan Health System, Ann Arbor, Michigan

Lower-extremity pain and paresthesia have multiple origins. Early recognition of the symptoms of peripheral nerve entrapment leads to timely treatment and avoids the cost of unnecessary studies. The authors report on a case of superficial peroneal nerve syndrome resulting from nerve herniation through a fascial defect, which was responsive to surgical treatment.

This 22-year-old man presented with pain and paresthesias over the lateral aspect of the right calf and the dorsum of the foot without motor weakness. Exercise led to the formation of a tender bulge approximately 12 cm above the lateral malleolus. Percussion of this site worsened his symptoms. Radiography and electromyography studies were nondiagnostic. The patient underwent surgical decompression that involved division of the fascia overlying the nerve and neurolysis of the superficial peroneal nerve. The operation resulted in symptom-free relief.

Superficial peroneal nerve syndrome is an entrapment neuropathy that results from mechanical compression of the nerve at or near the point where the nerve pierces the fascia to travel within the subcutaneous tissue. Surgical decompression of the mechanical entrapment usually provides relief from pain and paresthesia.

Key Words • nerve entrapment • peripheral nerve • peroneal nerve

ENTRAPMENT neuropathies are common clinical entities encountered in everyday neurosurgical practice. Among the most prevalent are median nerve entrapment at the wrist and ulnar nerve entrapment at the elbow. Other nerve entrapments and their attendant syndromes pose more difficult diagnostic challenges and may often be confused with more common clinical conditions. In this report, we describe a case of superficial peroneal nerve syndrome, a relatively rare and unusual nerve entrapment disease.

Case Report

History. This 22-year-old man presented with a 3-year history of progressive sensory disturbances in the right lower extremity. He experienced severe pain and paresthesias over the lateral aspect of the calf and over the dorsum of the foot, causing mild difficulties in walking. Exercise resulted in worsening symptoms and led to the formation of a bulge on the lateral aspect of the calf, approximately 11 to 12 cm above the lateral malleolus. The patient attempted to alleviate his symptoms by massaging the bulge; however, rubbing only increased the pain, numbness, and tingling in the aforementioned distribution. Sitting and resting partially relieved his symptoms, and gabapentin was only minimally effective in reducing symptoms. The patient denied having muscular weakness or atrophy of the foot or leg. There was no history of trauma.

Before presentation at our facility, the patient had undergone magnetic resonance imaging studies of the lumbar spine and EMG of the right lower extremity, both of which were unremarkable. He had also undergone an orthopedic examination, and multiple plain x-ray films were obtained; neither of these studies permitted a diagnosis. Results of magnetic resonance imaging of the right lower extremity performed while the patient was supine and resting were similarly nondiagnostic. The patient had no other neurological complaints. His general medical history was unremarkable.

Examination. Neurological examination revealed no gross motor weakness. Sensory testing by pinprick showed mild hypesthesia over the dorsum of the foot, with sparing of the web space between the great and second toes. We observed no unusual swelling while the patient was at rest; however, percussion of the anterolateral aspect of the right leg approximately 10 to 11 cm above the lateral malleolus reproduced symptoms of pain and paresthesia. His reflexes and gait were normal.

Operation. Because of the persistent symptoms, the site of maximal tenderness was explored while the patient was

Abbreviation used in this paper: EMG = electromyography.
in a state of general anesthesia, beginning with a 10-cm incision centered at the site. Careful dissection of the subcutaneous tissues proximal to the site revealed the deep fascia of the leg. Dissecting distally, a circular defect in the fascia was seen, with a knuckle of fatty tissue protruding from the defect (Fig. 1). Slightly distal to the circular defect was a transverse linear opening in the fascia, which transmitted the superficial peroneal nerve into the subcutaneous tissue 10 cm above the lateral malleolus. The nerve was dissected free from within the subcutaneous tissue and followed proximally. The transverse fascial band was divided (Fig. 2 left), as was the fascia 5 to 7 cm proximal to the circular defect (Fig. 2 right), leading to a completely free nerve. At the point of fatty protrusion pushing the nerve through the circular fascial defect, however, we were unable to complete the neurolysis because some of the fat appeared to infiltrate the nerve (Fig. 3). Following decompression, the nerve was free of any tension. The incision was irrigated and closed in the standard fashion.

Postoperative Course. The postoperative course was uneventful, and the patient was discharged home on the day of surgery. Two years after the operation, the patient remains free of pain and paresthesia, even during exercise.

Discussion

The common peroneal nerve branches into the superficial peroneal nerve as it passes behind the head of the fibula, laterally to the neck of the fibula. The superficial peroneal nerve (previously known as the musculocutaneous nerve) then passes deep to the peroneus longus muscle and sends branches to supply both the peroneus longus and brevis muscles. It continues along this route until it pierces the deep fascia of the leg, 10 to 14 cm above the lateral malleolus, to travel within the subcutaneous tissues. At this point, the nerve provides only sensory function to the skin of the anterolateral ankle and the dorsum of the foot. Note that the deep branch of the common peroneal nerve serves the web space between the great and second toes, and sensation in this distribution was accordingly preserved in this patient.

Superficial peroneal nerve syndrome represents an unusual type of nerve entrapment. The constellation of symptoms associated with it was first described by Henry in 1945 as “mononeuralgia in the superficial peroneal nerve.” Patients with this syndrome generally present with pain and paresthesias over the lateral aspect of the lower calf and over the dorsum of the foot. These symptoms usually increase with walking or other exercise. Signs of this syndrome include hypesthesia of the dorsum of the foot, a soft-tissue bulge over the anterolateral aspect of the leg approximately 10 cm above the lateral malleolus, an increase in the size of the bulge with exercise, and worsening of symptoms with percussion over the bulge.

Note that retrograde pain has been reported with this syndrome, which together with pain in the lateral aspect of the calf and dorsum of the foot may mimic an L-5 radiculopa-
thy. Indeed, in the literature there is one reported case of a patient who, despite having undergone laminectomy and discectomy at L4–5 for a minor extradural defect, had no postoperative change in symptoms for 6 months prior to the subsequent diagnosis and definitive surgical treatment of superficial peroneal nerve entrapment. Likely, other cases have gone unreported, and the incidence of superficial peroneal nerve entrapment resulting in anterolateral leg pain is perhaps higher than that suggested by the literature.

Conservative management is usually ineffective. Symptoms are generally suffered for an extended period of time, usually because they are attributed to a variety of other causes. The differential diagnosis for this syndrome includes lumbar radiculopathy, pseudoradicular pain syndrome, nerve sheath tumors, anatomical defects resulting from prior trauma, and chronic or exertional lateral compartment syndrome. Radiographic imaging usually fails to produce a definitive diagnosis. In some instances, EMG, nerve conduction velocities, and compartment pressure testing may assist in a diagnosis. Typically, decreased nerve conduction velocities are observed, with normal EMG findings on innervation of the peroneal muscles; however, nerve entrapment syndrome may exist even with entirely normal EMG and nerve conduction studies. Chronic lateral compartment syndrome may be diagnosed when muscle compartment pressure exceeds 30 mm Hg during exercise, when intramuscular pressure remains greater than 30 mm Hg at rest following exercise, or if normalization of pressures is prolonged (> 10 minutes). Relief produced on localized injection of lidocaine or hydrocortisone has also been reported to aid in a diagnosis. Only when a thorough workup is unrevealing does one usually arrive at a diagnosis of superficial peroneal nerve syndrome by exclusion.

Potential sites of entrapment of the superficial peroneal nerve include the peroneus tunnel (before the exit through the deep fascia) or the site of exit from the tunnel. Compression by herniating muscle or fat distal to the exit from the tunnel might also occur. Prior reports of this syndrome are few. After Henry’s first description, Kopell and Thompson described the syndrome as an “entrapment neuropathy” in 1963. In 1977 Mackey, et al., reported on surgical decompression as a successful treatment for the condition. Surgical observation in one of the patients in that study led to the suggestion of muscular herniation through a fascial defect as the origin of the nerve entrapment. In 1981 Banerjee and Koons reported nerve entrapment at the site of entry into the subcutaneous tissue by fatty herniation at the fascial opening. In the mid-1980s the syndrome was further characterized in the orthopedic and physical medicine literature, leading to a synthesis of syndrome features described earlier.

In 1997 Styf and Morberg reported that decompression of the superficial peroneal nerve at the fascial opening had resulted in a relief of symptoms in 80% of surgically treated patients.

Conclusions

In the present report, we provide the first photographic documentation of entrapment of the superficial branch of the peroneal nerve by fatty herniation through a fascial defect, proximal to the point where the nerve normally pierces the fascia to travel within the subcutaneous tissue. In the patient in our case, the fatty tissue not only compressed the nerve but also seemed to infiltrate it. Decompression and limited neurolysis of the nerve resulted in relief of symptoms.

We recognize that this case represents one of a series of variations in anatomical defects that compose the superficial peroneal nerve syndrome. Although muscle, fat, fascia, or other tissues may cause compression, nerve entrapment is mechanical and therefore best treated using surgical decompression.

References

Superficial peroneal nerve syndrome


Manuscript received June 29, 2005. Accepted in final form January 20, 2006.

Address reprint requests to: John E. McGillicuddy, M.D., Department of Neurosurgery, University of Michigan Health System, 1500 East Medical Center Drive, Room 3552 TC, Ann Arbor, Michigan 48109-0338. email: jemc@med.umich.edu.