Peripheral nerve entrapment due to steroid-induced lipomatosis of the popliteal fossa

Case report

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A case of peroneal nerve entrapment is reported in a patient with scleroderma. Compression was due to a lipoma in the popliteal fossa and resulted in increasingly severe foot-drop. Complete recovery occurred after the lipoma was resected. A brief review of peroneal nerve palsies and lipomatosis is presented.

KEY WORDS • popliteal fossa • peroneal nerve palsy • lipomatosis

Peripheral nerve entrapment syndromes resulting in weakness or sensory deficits, or both, are well documented. This entity most frequently involves the carpal tunnel, tarsal tunnel, and ulnar nerve at the elbow. In approximately 25% of all cases, a history of local trauma is well documented.

Common peroneal nerve palsy is also a frequent clinical entity. Most patients with this disorder present with a foot-drop after trauma to the knee joint. Other causes of this palsy include direct damage, traction, and compression of the nerve, as well as ischemia, generalized peripheral neuropathy, and tumors. This report describes a patient who developed an atraumatic partial foot-drop, sensory changes, and electrophysiological evidence of nerve compression in the popliteal fossa in association with a popliteal fossa mass. The patient was receiving steroid therapy for myositis associated with scleroderma at the time of his admission.

Case Report

This 36-year-old man had scleroderma associated with myositis, which was controlled with chronic steroid therapy. Approximately 1 year prior to admission, he noted a painless mass in his left popliteal fossa. Concomitantly, he began to experience mild paresthesias distal to his left knee over the superficial peroneal nerve distribution. This slowly progressed until, approximately 1 month before admission, he noted acute left hip pain and pain distal to his left knee on standing. Subsequently, the popliteal fossa mass became more prominent, with numbness, and the left foot weakness rapidly progressed. He denied symptoms of bladder or bowel sphincter dysfunction.

Examination. General physical examination revealed only acral and facial skin changes consistent with scleroderma. Neurological examination was significant for decreased power in the left anterior tibialis and extensor hallucis longus muscles, a burning dysesthetic numbness to light touch and pinprick in the left superficial peroneal nerve distribution, and a decreased ankle jerk on the left. Fullness in the popliteal fossa suggested a small mass on the left. There was a positive Tinel's sign over the left popliteal fossa and fibular head.

Laboratory studies, including hematocrit, white blood cell count, platelet count, coagulation studies, electrolyte and creatine phosphokinase levels, and urinalysis, were normal. The popliteal fossae appeared normal on ultrasound studies, and computerized tomography (CT) demonstrated calcification of the fascia overlying the sartorius muscles (more on the right side than the left), with no evidence of a mass lesion. An electromyogram and nerve-conduction velocity study showed a prolonged F-wave on the left side and chronic reinnervation changes in the left anterior tibialis and gastrocnemius muscles. The sural nerve sensory latencies were increased bilaterally.

Operation. The patient was taken to the operating suite for a left popliteal fossa exploration. The fossa was
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exposed by means of an S-shaped incision across the joint line. The posterior cutaneous nerve was identified as it emerged from the popliteal fascia and was followed upward to the posterior tibial nerve. In the midportion of the fossa, the nerve was totally surrounded by a large lipoma which encased the common peroneal, and a portion of the posterior tibial nerves. The lipoma was dissected and removed from the nerves, including its extension deep toward the floor of the fossa. Its upward extent was also dissected and removed at the point where it disappeared beneath the hamstring muscles (Fig. 1). The nerves were then tested with a nerve stimulator and found to have a reduced sensory and motor threshold.

Postoperative Course. The postoperative course was uneventful and the patient was discharged on the 9th postoperative day with improving symptomatology. At a clinic visit 2 weeks later, the motor strength had improved in his left extensor hallucis longus muscle to 4.8/5 power. The sensory examination was normal except for mild hypesthesias in the peroneal distribution, but his left ankle jerk was still diminished. At his last follow-up visit, 2 months postoperatively, power was only slightly diminished in the extensor hallucis longus muscle, the sensory examination was normal, the Tinel's sign was no longer present, and the ankle jerk was improved.

Discussion

We present a patient with a 1-year history of a popliteal fossa mass coupled with a 4-month history of leg paresthesias and numbness. This man was receiving chronic steroid therapy for myositis associated with scleroderma. Clinically, he had involvement of both the distal peroneal and the tibial nerves, which proved to be due to a large lipoma of the popliteal fossa causing compression of the nerves in and superior to the fossa. Postoperatively, he did well with a return of both motor and sensory function.

Popliteal fossa masses are probably most commonly caused by a distention of a popliteal bursa or Baker's cyst. This is generally found in young to middle-aged males, and is characterized by the gradual development of a tense, cystic, fluctuant, and painless mass in the fossa. The other causes of popliteal fossa masses occur much less frequently. The differential diagnosis includes aneurysm (generally of the popliteal artery), hemangioma, or neoplastic growth. Popliteal aneurysm is characterized by pulsation and bruit synchronous with the pulse, and is easily demonstrated on arteriography. A hemangioma is painful, rubbery, soft, and warm, and is associated with dilated surface vessels and multiple flecks of calcium on roentgenograms. Neoplasms include benign growths such as lipomas, which are soft, localized, and mobile, or malignant tumors such as fibrosarcomas, which are firm, tender, invasive, and fixed to surrounding tissues. In our patient, the leading differential diagnosis included a Baker's cyst or benign neoplasm. A normal CT scan and ultrasound study, however, argued against both of these.

Compression of peripheral nerves in the popliteal fossa is a fairly well characterized entity. Probably the most common type of compression involves the common peroneal nerve, causing a palsy and resulting in foot-drop. In 1968, Sunderland reviewed a series of military wounds with palsy due to disruption of the peroneal nerve; in many of these cases the palsy was permanent. Kline in 1972 summarized this type of palsy in civilian injuries, most of which involved gunshot wounds. Other injuries included ankle inversion or trauma of the knee or hip. Other causes of peroneal palsy include such unusual entities as ischemic palsy, traction injuries, compression of the nerve within its sheath due to hematoma, and ganglion of the nerve. In 1976, Berry and Richardson published a detailed clinical and electrophysiological analysis of peroneal nerve palsies. They listed trauma in the knee or hip area, compression, or neuropathies as the most common causes. Some palsies were caused by prolonged compression during coma or crossed-leg compression. It was noted that the prognosis was excellent in these patients once the compressive factor was removed. This correlates with the findings of Garland and Moorhouse and with the results in our patient.

Banerjee and Koons reported two patients with superficial peroneal nerve palsies. Both patients presented with pain and numbness in the appropriate distribution, caused in one by compression of the nerve against its fascial opening, and in the other by a lipoma compressing the nerve at its fascial opening. Opening the fascia in the first patient and removing the lipoma in the second gave good relief. Our patient is comparable, in that a lipoma was found at surgery and removal of the lesion gave complete relief with full recovery.

Fat deposition may occur in the setting of excessive
endogenous or exogenous glucocorticoids and can result in various conditions; 8-12 it is more commonly centripetal than centrifugal. Two cases of special interest are those of Lipson, et al.,9 and Lee, et al.8 Both patients had spinal cord compression caused by epidural fat expansion secondary to prolonged steroid therapy. Lipson, et al., described a patient with classic symptoms of spinal stenosis who obtained complete relief after laminectomy and removal of a large deposit of epidural fat. Lee, et al., reported spinal cord compression due to epidural fat compression caused by steroid therapy after renal transplants. As noted previously, our patient was receiving chronic steroid therapy for myositis associated with scleroderma. Centripetal obesity was manifested by slightly rounded facies and supraclavicular fat pads. His symptoms correlated with an increase in steroid dosage over a period of months. We propose therefore that the popliteal lipoma may have been caused or enhanced by exogenous steroids, and thus represents centrifugal fat deposition.

Conclusions

We describe a patient with symptomatic peroneal and tibial nerve palsies due to compression by a lipoma of the popliteal fossa; he was receiving chronic steroid therapy. After decompression by removal of the lipoma, his symptoms of sensory deficit, Tinel's sign, and footdrop resolved. This is in accord with the literature which describes the excellent prognosis of patients with a compressive peroneal nerve palsy. This case appears to be another example of compression by lipomatosis or endogenous fat deposits under the influence of chronic steroid therapy; in this patient the peripheral nerves were affected.

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References


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