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Two Unusual Cases of Sciatic Neuropathy

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Two different unusual cases of acute sciatic neuropathy are described. They appeared to have been caused by complications from a procedure performed on a patient in the lithotomy position, and an uncertain etiology associated with either a previous femur fracture or a recent blunt trauma to the buttock. Comprehensive histories of weakness, with radiographic or electrophysiological studies, or even exploratory surgery are important in order to understand the etiologies of the sciatic neuropathies.

Key Words: Sciatic neuropathy, Lithotomy position

The sciatic nerve is the longest and largest nerve in the body, but sciatic neuropathy is an uncommonly diagnosed type of focal mononeuropathy. Most sciatic nerve lesions are associated with trauma such as that caused by hip fractures and dislocations, or with complications of hip surgery. External compressive lesions occur during coma, anesthesia, protracted periods of confinement in bed, when hard objects pressed against the buttock, and muscle fibrosis following intramuscular injections.[1] Several internal masses can compress the nerve; these may include hematomas related to hip surgery, contusions, the use of anticoagulants, endometriosis, synovitis of the hip, hernias, soft tissue tumors, and vascular anomalies.[1-4] We describe two different cases of sciatic neuropathy with reviews of other reported cases in the literature.

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Case reports

Patient 1.

A 64-year-old man presented for abdominoperineal resection because of anal cancer. He was slender because of weight loss during the previous few months. Under general anesthesia, he was placed on the operating table in the lithotomy position. He remained in the same position for 8 hours. The surgery was uncomplicated, with no episodes of severe hemorrhage or hypotension. On post-operative day 1, he complained of left foot drop. Neurological evaluation revealed muscle weakness measured at 3/5 for the left knee flexors, with 2/5 measured for dorsiflexors, evertors, invertors and the plantar flexors of the foot. Additionally, he had hypesthesia to light touch and pin prick on the lateral surfaces of the leg and the dorsum of the foot, with an absence of ankle jerk on the left side. Posterior tibial, peroneal, and sural nerve conduction studies performed at 2 and 4 weeks after operation failed to reveal any asymmetrical changes. EMG performed at 4 weeks revealed a few scattered positive sharp waves and few fibrillation potentials in the left tibial and peroneal nerve innervated muscles

(Table 1). Motor unit potential analysis showed some medium voltage polyphasic waves, but giant motor unit potential was not found. The patient's weakness was resolved with conservative treatment by 2 months after operation.

Patient 2.

A 34-year-old man presented with a sudden onset of left foot drop. He had fallen onto his buttock following excessive alcohol consumption a week ago. He felt a sharp, jabbing pain in the distal sciatic distribution a few hours later. The next morning, he noticed left foot weakness. His previous medical history revealed that he had suffered a left thigh wound in a traffic accident 3 years ago. He underwent an internal fixation for a subtrochanteric fracture of the femur at that time. Subsequently, he had some limitations in internal rotation of the left hip. Physical examination showed no obvious gluteal swelling. Neurological examination revealed that the patient had strengths of 3/5 in the left knee flexors, with strengths of 1/5 in dorsiflexors, evertors, invertors, and the plantar flexors of the foot. He had hypesthesia to light touch and pin prick on the surface of the lateral leg, dorsum and sole of the foot. The ankle jerk was absent on the left side. Lumbar spinal magnetic resonance imaging and hip and pelvis X-rays showed no recognizable abnormalities. Routine nerve conduction studies 2 weeks later revealed the absence of the H-reflex and a prolonged tibial F-wave on the left side. EMG showed large amount of positive sharp waves and fibrillation potentials in tibial and peroneal nerve innervated muscles (Table 1). Lumbosacral paraspinal muscle EMG did not reveal any spontaneous activity. Motor unit potential analysis in the denervated muscles showed long duration medium voltage polyphasic waves. He underwent exploratory surgery for decompression. During the removal of the screws and nails, no discernible compressing mass or fibrous band was noted. Exploration of the sciatic nerve revealed no focal abnormalities. Hypertrophy of the piriformis muscle was absent. Following the adhesiolysis of the sciatic nerve, the first sign of improvement, the restoration of knee flexor strength, was noted within a week. The patient had minimal residual foot weakness and paresthesia one year following the treatment.

Discussion

The incidence of postoperative peripheral neuropathy has been reported to be between 0.03% and 25%, depending on the criteria.[3-6] Persistent neuropathies after procedures performed on patients in lithotomy positions were identified in 55 cases among 198,461 procedures. Out of 55 patients, sciatic neuropathy was found in 8 patients; none of these regained complete motor function within 1 year.[5] However, a prospective evaluation in 991 adult patients undergoing general anesthetics and surgical procedures while in

Table 1. EMG results of the two patients

	Case 1		Case 2	
Muscle	Normal	Abnormal	Normal	Abnormal
Tibialis anterior		+		++
Short head of BF*		+		+
Peroneus longus		+		++
EDB†		+		+
Gastrocnemius		+		++
Long head of BF	+		+	
Tibialis posterior		+	+	
Abductor hallucis		+		+
Paraspinalis	+		+	

* BF; biceps femoris

† EDB; extensor digitorum brevis

the lithotomy position showed that motor dysfunction did not develop.[6] Clearly, sciatic neuropathy accompanying motor weakness associated with the lithotomy position is extremely rare. Although the most likely causes of perioperative neuropathies are compression, stretching, and ischemia, the mechanism of neuropathy is often unclear. Prolonged duration of procedures in lithotomy, the patient's very thin body habitus, and smoking in the perioperative period have been suggested as risk factors.[6] The relatively rapid, full recovery of motor function in patient 1 is unusual compared with other reports. The primary cause of this patient's weakness appeared to be neurapraxia, since the patient had nearly normal conduction studies, recovering full muscle strength within 2 months.

Unlike patient 1, the cause of sciatic neuropathy in patient 2 was still unclear. Initially, it was explained as either the compartment syndrome or an acute external compression from traumatic hematoma. However, surgical exploration failed to reveal a hematoma. We were not sure that adhesiolysis and removal of nails and screws actually relieved the pain and weakness. Some reports indicated that the appearance of sciatic neuropathy might be delayed months or even years following hip replacement.[2] However, clinical observations of long-delayed neuropathies following hip trauma without hip arthroplasty, as seen in patient 2, have not been reported. Although there was no evidence of mass effect, compression, hypertrophy, inflammation or change in the volume of the sciatic nerve, it is still unclear that patient 2 really had a delayed sciatic neuropathy after the intertrochanteric fracture of the femur. However, it seems likely that the surgical removal of screws and nails at least contributed to the reduction of his weakness. If the surgical procedures relieved the pain and weakness to some extent, then the chronic compression could be one of the causes of the delayed neuropathy. Post-traumatic piriformis syndrome should be also considered because he had blunt trauma to the buttock, subsequently showing signs and symptoms suggestive of lumbar nerve-root compression, with adhesiolysis leading to improvement.[7] However, the piriformis syndrome is perhaps overdiagnosed and therefore somewhat controversial. Further, our intra-operative findings did not reveal sufficient adhesions between the piriformis muscle, the sciatic nerve, and the roof of the greater sciatic notch. Finally, it is unlikely that he had an idiopathic sciatic neuropathy. Idiopathic cases are known to have painless neuropathies compared to those with a known etiology, and most idiopathic cases are chronic and progressive in nature.[8] Exploratory surgery appeared to be an ineffective treatment for the cases of idiopathic sciatic neuropathy.[9] In fact, a history of acute contusion and internal fixation for femur fracture, with some improvement after the surgical procedures, excludes the possibility of idiopathic neuropathy.

In conclusion, we suggest that a number of different causes should be considered when making a differential diagnosis of unexplained sciatic neuropathy. Comprehensive histories of weakness, with radiographic or electrophysiological studies, or even exploratory surgery are important in order to understand the etiologies of the sciatic neuropathies.

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