

An arteriovenous malformation as a rare cause of sciatic neuropathy: a case report

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ABSTRACT

Sciatica is characterized by pain and discomfort occurring frequently with impingement at the level of spinal nerve along the regions innervated by the sciatic nerve. Compression and irritation of the nerve often occurs with a spinal cause such as lumbar disc hernia or spinal stenosis. Compression of the nerve by an arteriovenous malformation is very rare among the causes leading to non-discogenic sciatic neuropathy. Herein, we reported our case with clinically and electrophysiologically typical sciatic neuropathy and treated by us surgically. To our knowledge, cases with arteriovenous malformation -caused sciatica were limited in the literature review. Electromyography should be performed to exclude the nerve compression due to rare causes such as vascular causes which may lead to sciatic neuropathy in patients with sciatic distribution symptoms and signs, after initial negative spine imaging.

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Introduction

Sciatica is defined as pain or discomfort along the regions innervated by the sciatic nerve [1, 2]. Although sciatica is most often radicular in origin, new radiating pain despite negative lumbar imaging studies warrants consideration of such nondiscogenic origins as plexopathy or neuropathy. Infrequent causes include benign or malignant tumors, infections, mechanical entrapments, and vascular causes [3]. Arteriovenous malformations (AVMs) among vascular causes are frequently seen in head and neck region and it is too

rare to cause sciatic nerve compression by them. AVMs are the high-flow lesions which are aggressively growing and causing tissue destruction [4]. They are usually asymptomatic and seen in the brain, lungs and lower extremities. They rarely cause peripheral neuropathy.

Herein, we reported our case with sciatic neuropathy due to compression of AVM. Lumbar spinal magnetic resonance imaging (MRI) of the patient was negative, but clinical and

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electromyography (EMG) findings were supporting sciatic neuropathy. Surgical exploration was performed and then total excision was performed after ligation of the feeders and drainage veins of AVM compressing the sciatic nerve. External and limited internal neurolysis was performed. Pain of the patient was improved postoperatively and he had no pain at first year follow-up.

Case Presentation

A 53-year-old man presented with persistent left sided, radiating leg pain. The patient defined the pain present for 6 months in the left buttock, posterior and lateral thigh and lateral calf. He had no history of a known disease but he defined an intramuscular injection performed in left gluteal region 8 months ago. Laseque and straight leg raise test was negative. Weakness was determined in foot dorsiflexion and plantar flexion (3/5). Numbness in a sciatic distribution from the lower buttock toward the foot. An ankle jerk was decrease. Mild foraminal protrusion was determined at the L4-5 level on MRI (Figure 1a and 1b). Since the clinic could not be explained by MRI findings, EMG was performed. Nerve conduction studies showed reduced peroneal (recording over the extensor digitorum brevis and tibialis anterior) and tibial compound muscle action potential (CMAP). Sensory studies demonstrated

abnormality sural and superficial peroneal sensory nerve action potential (SNAP). Electromyography showed neurogenic abnormalities in the tibialis anterior, medial gastrocnemius and the short head of the biceps femoris. MRI was not performed because of strong suggestion of sciatic neuropathy by clinical presentation and electrophysiological studies. Given these findings, we considered sciatic neuropathy which could be as a late complication of gluteal intramuscular injection. We decided to perform a left sciatic exploration. Under endotracheal general anesthesia, the patient was placed in the genupectoral position. First, the posterior inferior iliac spine and the site 1 inch behind the greater trochanter tip were identified. Second, a longitudinal curved skin incision was made between these two points distally and extended about 4 cm along the femoral shaft. After muscle dissection, AVM was seen and it was located on the sciatic nerve and compressing it. The AVM was originated from the superior gluteal artery and drained to inferior gluteal vein. Engorged, purple veins and one prominent draining vein as well as small arterial feeders were noted. After ligation of the feeders and draining veins one by one, AVM was excised totally (Figure 2a and 2b). Surgical exploration with external neurolysis as well as limited internal neurolysis of the sciatic nerve. After surgery, the patient's symptoms resolved, and she could sit for about 1 hour and walk without support 3 months later, the visual analog pain scale score had dropped from 10 to 2. At the final

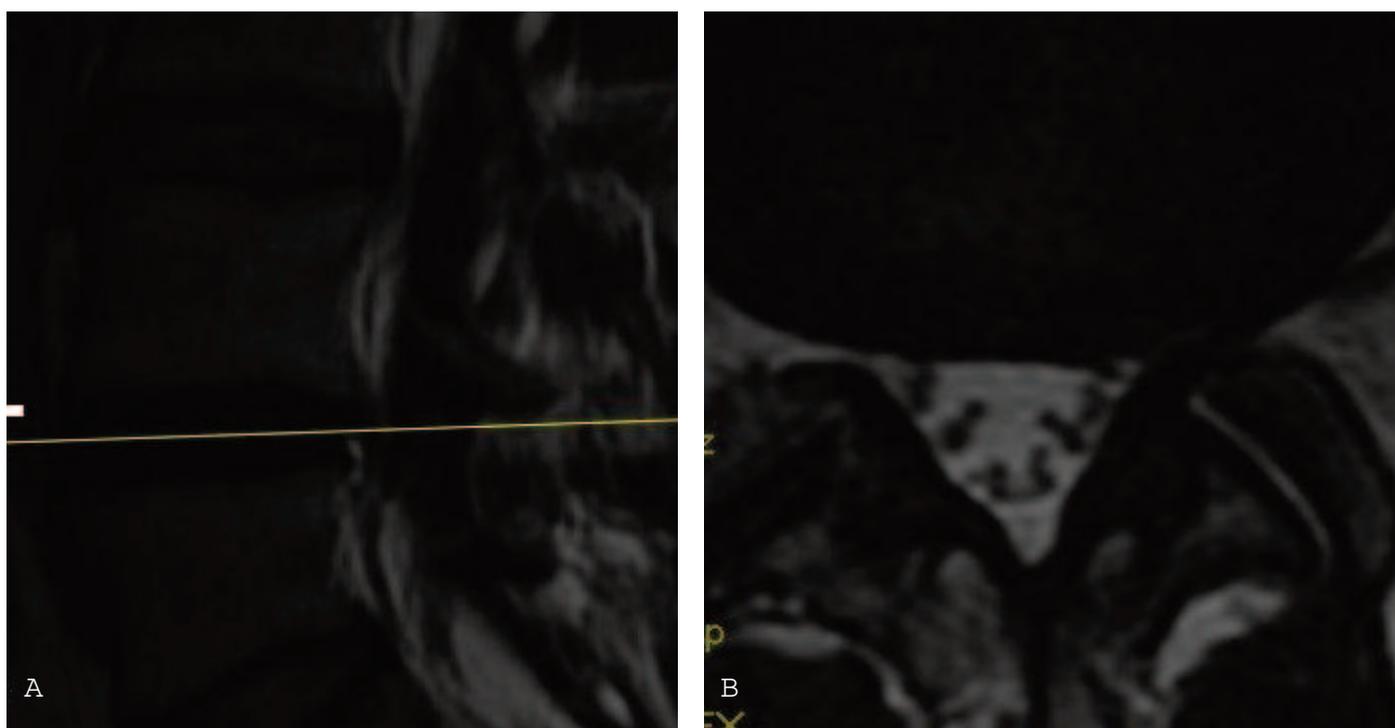


Figure 1. Mild foraminal protrusion was determined at the L4-5 level on MRI (a and b).

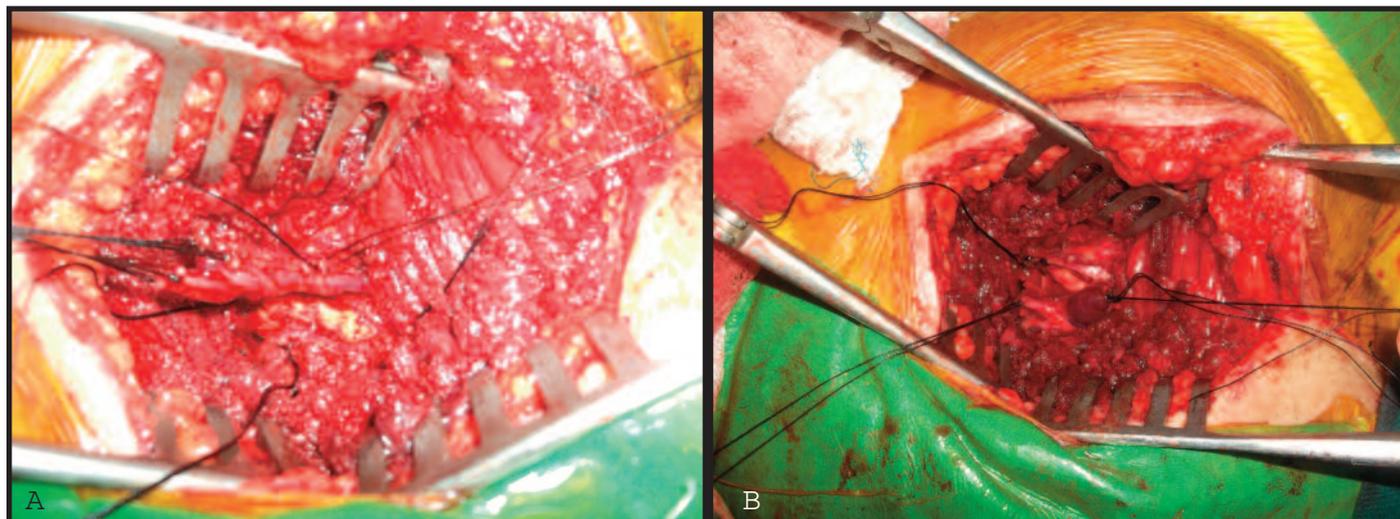


Figure 2. Engorged, purple veins and one prominent draining vein as well as small arterial feeders were noted (a). After ligation of the feeders and draining veins one by one, AVM was excised totally (b).

follow-up evaluation 12 months later, the patient reported no recurrence of his pain.

Discussion

The sciatic nerve has 2 divisions: the superficial and lateral peroneal nerves, and the medial tibial nerve. The sciatic nerve trunk innervates hamstrings and the distal adductor magnus. The peroneal and tibial nerves supply the anterior and posterior leg and intrinsic foot musculature. Through sensory branches of the tibial (sural, medial, and lateral plantar; and calcaneus) and the superficial peroneal nerves, it supplies sensation to the foot and posterior lower leg. As a consequence, sciatic neuropathies present with weakness below the knee and numbness over the calf and foot, and the ankle reflex is usually absent [1, 5]. Although the vast majority of sciatica is due to degenerative causes and is radicular in nature, various other causes are to be considered when spine imaging is negative [3]. Sciatic neuropathy is distinctly uncommon and is associated with a limited differential diagnosis [6]. Hip or femoral fractures and the complications of the surgeries of these fractures [6], tumors as another cause of common sciatic neuropathy (neurofibroma, schwannoma, neurofibrosarcoma, lipoma and lymphoma), infections and large Baker's cyst in the popliteal may compress the sciatic nerve [1, 7]. Sciatica has been reported, although rarely, to be related to vascular lesions along the course of the sciatic nerve from the pelvis to its bifurcation [6, 8-10]. Other vascular lesions discussed as causes of sciatica are anatomic anomalies such as a sciatic artery

or vein, gluteal varicoses, venous thrombosis, hemangiomatosis in Klippel-Trenaunay syndrome, and venous or capillary hemangioma [2, 3, 6, 7, 11]. Inferior gluteal artery aneurysm, usually caused by trauma, is more common in neurovascular compression mimicking lumbosciatic pain [12]. Vascular causes of peripheral neuropathy are extremely uncommon and require a focused workup. AVMs are uncommon vascular lesions formed by multiple abnormal communications between the arterial and venous systems without an intervening normal capillary network [4]. The etiology of these lesions has been a subject of controversy, although it is generally agreed that the greater part of these lesions are congenital, with a few that are acquired after trauma or surgery [4]. AVMs are high-flow lesions, characterized by aggressive growth and tissue destruction. The majority of AVMs are asymptomatic and affect the brain, lung, and the lower extremities. Lower extremities AVMs most commonly manifest with dermatological signs including discoloration and swelling. Development of peripheral neuropathy due to AVM compression is very rare [3, 5, 11].

Symptoms of the case presented by us were supporting the spinal etiology, but no significant pathology was determined in spinal imaging of the patient. Findings supporting the sciatic neuropathy were seen in the EMG performed (decreasing the CMAP and SNAP potentials in the nerve conduction study and neurogenic abnormalities in the tibialis anterior, medial gastrocnemius and the short head of the biceps femoris). We considered that the neuropathy of the patient with history of gluteal injection performed 8 months ago could be late complication of

injection injury firstly [13]. We explored left sciatic nerve. After the ligation of AVM's feeders and drainage veins compressing the nerve, we performed total excision, external neurolysis and limited internal neurolysis. Pain of the patient improved completely after the surgery.

Endovascular embolization of the AVMs causing sciatic neuropathy is a method of treatment that can be performed, but surgical excision is still successful since these cases are very rare and loss to long-term follow-up [4, 9]. Development of sciatic neuropathy due to a vascular etiology is a very rare condition. When the literature is scrutinized, the number of the cases with sciatic neuropathy developed due to compression of an AVM does not exceed [2].

In the cases presented with sciatic clinic but having negative spinal imaging, peripheral nerve MRI and/or EMG should be necessarily performed to exclude the other reasons caused sciatic neuropathy. MRI was not performed because of strong suggestion of sciatic nerve compression at the proximal region by clinic and electrophysiological studies.

Conclusion

With this rare case, we aimed to emphasize the necessity of presence of a pathology compressing the nerve and EMG and radiological investigation of the nerve in the cases with sciatic neuropathy clinic but having negative spinal imaging. We discussed how AVMs could be encountered in the clinic as a rare reason caused sciatic nerve compression and their treatments.

Informed consent

Written informed consent was obtained from the patient for the publication of this case report.

Conflict of interest

The authors declared that there are no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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