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A Case of Spinal Cord Injury following embolization for the treatment of AVM

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Background

Spinal vascular malformations, including spinal arteriovenous malformations (AVM), are rare diseases, but they can cause spinal cord injury (SCI). We report a case of SCI following embolization for the treatment of AVM.

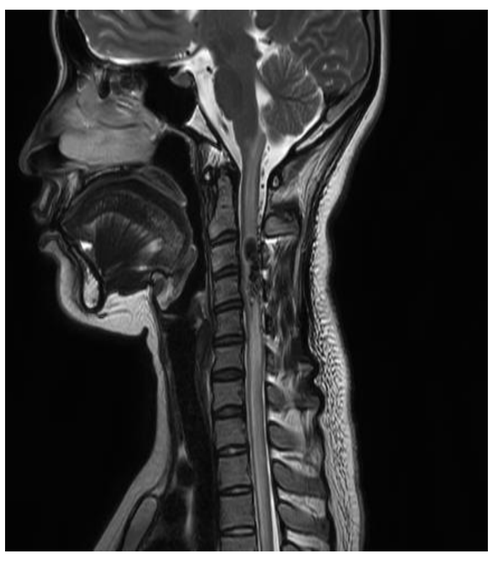

Figure 1. Cervical spine MRI showed an 8mm-sized, 2.1 cm-length lobulated vascular structure at the C3-4 level, compressing the spinal cord.

Case report

A 26-year-old woman presented with right arm numbness and right side weakness (proximal upper limb; grade IV, distal upper limb; grade II~III, leg; grade IV) for two weeks. Cervical spine MRI revealed an 8mm-sized, 2.1 cm-length lobulated vascular structure in the intradural region at the C3-4 level. This structure compressed the spinal cord and the perimedullary vessels in the upper cervical region were dilated. MRA, 4-vessel angiography and bilateral subclavian artery angiography were performed, leading to a diagnosis of spinal AVM. Feeders were identified from both deep cervical arteries, the right C2 radicular artery, and the anterior spinal artery. The lesion was located from the anterior aspect of the spinal cord at the C3-4 level, extending to the right side and posterior aspect, accompanied by spinal cord edema. Given the lesion's location, a surgical approach seemed challenging, so spinal AVM embolization was planned. The goal of the embolization was to block the flow entering the AVM, thereby reducing reflux into the medullary vein.

Post-procedural angiography showed that most of the AVM flow had disappeared. However, a very slow residual shunt flow remained through the anterior spinal artery descending from the vertebral artery to the C3 level. The patient's consciousness and respiration recovered after the procedure; however, motor strength and sensation in both upper and lower limbs were diminished (motor grade I). Tetraplegia was most likely caused by spinal cord infarction as a complication of embolization. Steroid pulse therapy was administered, and a follow-up cervical spine MRI was performed to differentiate other possible causes besides spinal cord infarction. MRI showed spinal cord infarction at the C3-C5 level, along with probable hemorrhagic portions at the C3 level and diffuse spinal cord edema.

One week after embolization, the patient had a complete SCI. The neurological level of injury was at C4 for sensory and motor function on both sides. Respiratory function was maintained without oxygen support. However, restrictive pattern of pulmonary dysfunction and diaphragmatic dysfunction were observed. Comprehensive rehabilitation therapies were administered.

Discussion

Spinal AVMs may present with acute or progressive sensorimotor deficits and nonspecific symptoms such as paresthesia, diffuse muscle pain. Spinal cord damage occurs due to bleeding, space-occupying effect and venous congestion, rarely by steal effect. Early treatment is associated with better long-term neurological outcomes. Therapeutic options include endovascular embolization or surgical resection. Success rates still depend directly on the lesion subtype and mode of presentation.