

# Postoperative Presentation of Lumbar Spinal Stenosis: A Case and Review of the Literature

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## Introduction

Only around 5% of all spinal cord vascular lesions are abnormalities of the spinal cord's blood vessels. The term "cavernous hemangiomas" was also used to describe spinal cord vascular malformations prior to the 2018 International Society for the Study of Vascular Anomalies (ISSVA) classification. [1] Despite being relatively rare, they frequently result in severe clinical symptoms (such as low back pain or radicular pain, progressive neurological deficits, abrupt severe neurological manifestations caused by spinal cord hemorrhage or embolism, subarachnoid hemorrhage), a poor prognosis, and should be taken seriously given that the incidence of subarachnoid hemorrhage caused by spinal vascular malformations has exceeded 20%. [2]

The question of whether spinal vascular abnormalities are inherited or acquired illnesses remains unresolved. Numerous instances of family or hereditary illnesses coupled with spinal arteriovenous malformations or arteriovenous fistulas in the literature imply that certain types of spinal vascular malformations may have a genetic basis. [3-6] This case report, however, raises the

possibility that spinal vascular abnormalities are an acquired disorder, especially in connection with surgical trauma.

## Case report

The patient, a 46-year-old man, underwent L4/5 spinal canal decompression with pedicle nail fixation after being diagnosed with lumbar spinal stenosis 8 years prior. He first had lumbar discomfort three months ago, which has now spread to both of his lower limbs, with a heavy left-side predominance and numbness and electrical sensations. The pain got worse after extended bed rest and walking, making it difficult for the patient to sit and sleep. The patient walks like a duck and has numbness mostly in the buttocks, the back of the legs, the heel, the ball of the foot, the bottom of the foot, and the two toes. Since the beginning of the illness, there has been no appreciable change in weight and regular bowel movements. Although the preoperative lumbar spine DR and MRI of the patient did not reveal any nerve root compression, patchy epidural low signal shadows were visible in the surgery location on the T2-weighted serial sagittal plane.



Figure 1: Lumbar DR front and side view of the patient before surgery



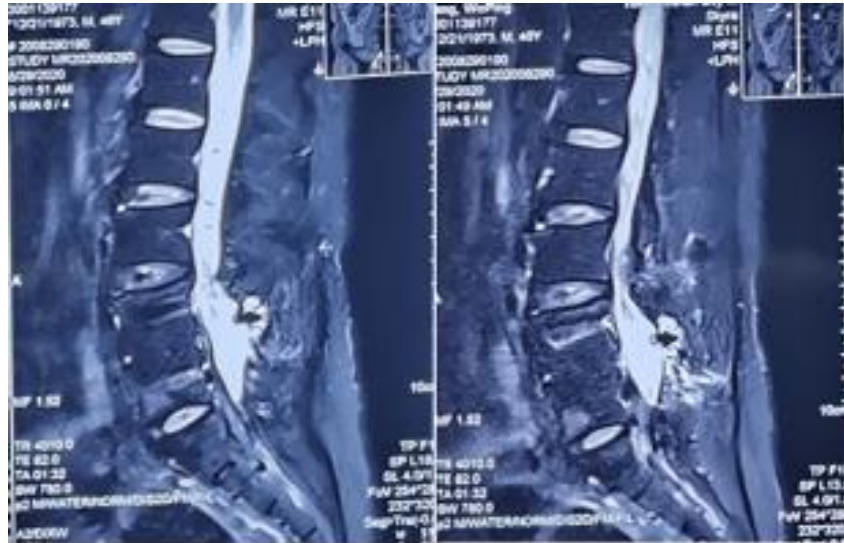


Figure 2 : The patient's preoperative MRI showed no significant compression of the spinal cord, but the T2W2 sagittal plane showed patchy low signal shadows in the epidural area in the surgical region

We concluded that the patient most likely had a spinal vascular malformation at the location of his previous surgery based on the patient's pre-operative imaging and symptoms. The patient was scheduled for surgery since his symptoms were serious, had been for three months, and significantly impacted his quality of life. In order to remove the sick, deformed vessels, a portion of the L4 plate had to be removed during the treatment.

A thick vessel in the epidural of the L4 plane that is directly connected to the dorsal coronary vessels can be seen when the L4 lamina is removed to expose the dura, as shown in Figure 3. In order to clot the aberrant vessel, we performed bipolar cautery and dissection. The process did not change the nerve detection. Our preoperative suspicions were validated when the excised material's postoperative pathology showed abnormal vascular tissue.



Figure 3 : Intraoperative visualization of a malformed blood vessel outside the dura

Radiating numbness and pain in the lower back and both

lower limbs were significantly lessened on the first day following surgery, and by the time the patient was discharged two weeks later, his walking gait was normal and his sleep issues had been resolved. Figure 4's post-operative DSA demonstrates that there was no discernible vessel malformation in the initially operated segment.

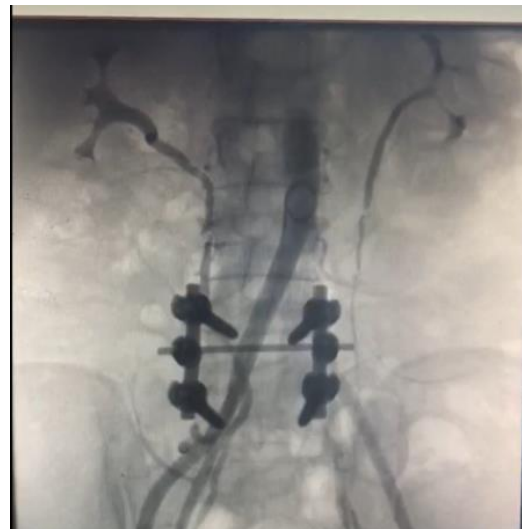


Figure 4 : Post-operative DSA shows no more malformed vessels in the operated area

### Discussion

The first reports of spinal vascular malformations date back to 1888, when Ellsberg successfully operated on the AVM. [7]The anterior median spinal vein, posterior median spinal vein, anterior lateral spinal vein, and posterior intermediate spinal vein are some of the longitudinal anas- composing veins of the spinal cord. Intrinsic spinal cord veins and pial veins are the first to enter the spinal cord. [8]The spinal cord's network of superior anastomosing veins is capable of significant

change. The anterior and posterior spinal arteries are frequently followed by these veins. The epidural venous plexus also provides venous drainage to the radiculomedullary veins, where they empty into. The segmental (or intervertebral) veins, which the radiculomedullary veins enter, continue to empty into the subcostal veins, as well as the azygous and hemizygous veins.[9]

Spinal vascular shunts, including fistulas and malformations, are rare and complex vascular lesions for which multiple classification schemes have been proposed. The most widely adopted scheme consists of 4 types: type I, Dural AVFs; type II, intramedullary glomus AVMs; type III, juvenile/metameric AVMs; and type IV, intradural perimedullary AVFs. MR imaging and angiography techniques permit detailed assessment of spinal arteriovenous shunts, though DSA is the criterion standard for delineating vascular anatomy and treatment planning. Diagnosis is almost exclusively based on imaging, and features often mimic more common pathologies. The radiologist's recognition of spinal vascular shunts may improve outcomes because patients may benefit from early intervention.[10-12]

There is no agreement on the ideal time for surgical intervention for symptomatic vascular abnormalities, which is relevant to the scheduling of surgery for AVF. [13]Because of the early spinal cord edema and the increased risk of bleeding associated with early surgery, Reitz et al. advise that surgery be carried out between 2 and 6 weeks after the injury. [14]The scar between the haemangioma and the spinal cord, on the other hand, can make tumor dissection more challenging and raise the risk of harm to healthy spinal cord tissue if the deformed artery is removed after 6 weeks.

For a better neurological outcome, Imagama et al. advised delaying surgery so that patients might rebuild muscle strength before surgery. It took an average of 1.7 months for full recovery, while the exact duration was not known. This presumption is based on the fact that patients who have fully recovered have higher muscle strength readings before surgery.

Although the exact cause of spinal vascular malformations is unknown, AVFs are believed to be acquired rather than developmental in nature (perhaps post-traumatic). We can see from this case that the patient did not exhibit any overt signs of AVFs prior to the lumbar spine surgery, but several years later the

patient began to exhibit symptoms. Our follow-up surgery confirmed the development of abnormal blood vessels at the site of his surgery, indicating that trauma from the surgery may also have contributed to the development of spinal vascular malformations.

## Conclusion

Spinal vascular malformations are a rare condition and we have to consider the possibility of AVF when there is a patchy low signal shadow outside the dura on T2W2 on enhanced MRI. The etiology of AVF is unknown, but in this case, the trauma associated with surgery is also a cause of its development.[15]

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