CASE REPORT

Management of Endovascular Intervention Complication in Spinal Vascular Malformation—Report of Two Cases

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Abstract

Aim: Management of cases of spinal vascular malformation, complicated by vessel injury during catheterization.

Background: Vascular abnormalities of the spinal cord are rare group of spinal cord disease and complex angioarchitecture of these lesions makes its treatment both by surgical or radiological intervention technically demanding.

Case description: We report two cases of spinal vascular malformation, complicated by vessel injury during catheterization. In case 1, extravasation of embolic material and blood could be documented by the digital subtraction angiogram (DSA), the patient developed progressive sensorimotor deficits after the next 48 hours, whereas in case 2, the patient developed immediate postprocedural weakness in both lower limbs and postprocedural computed tomography (CT) showed glue material in the spinal canal. Both the patients underwent decompressive laminectomy and showed improvement in motor power.

Conclusion: Following intervention, if the patient develops weakness, it is generally thought to be due to ischemia, and not due to extravasated material (glue + hematoma). When imaging shows the mass effect due to this complication, the patient should undergo decompression instead of taking a pessimistic attitude of not doing anything due to supposedly arterial ischemia and infarct.

Keywords: Complication, Intervention, Laminectomy, NBCA, Onyx.

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Introduction

Vascular abnormalities of the spinal cord entitle a special and rare group of spinal cord disease. The disabling natural history of the lesion caused due to hemorrhage, mass effect, venous congestion, and vascular steal prompts early diagnosis and treatment. Since the first surgical intervention by Elsberg in 1914, the understanding of the pathophysiology and treatment modalities of the spinal arteriovenous malformations (AVM) had made considerable advances.[1]

Since the inception of spinal angiography over half century earlier by pioneers Djindjian, Doppman, Di Chiro, and others, significant advances have been made.[2] Current scientific advances had brought better imaging techniques, better instruments, and embolization materials at our disposal. Still, the complex angioarchitecture of these lesions makes its treatment both by surgical or radiological intervention treacherous and technically demanding. Though some cases are better managed with endovascular techniques, surgery may be required due to any complication arising out of endovascular procedures.

Case Report 1

A 35-year-old lady presented with complaints of gradually progressive weakness of both lower limbs for 2 months. It was asymmetrical in onset with tingling and numbness involving the right followed by the left lower limb and progressive weakness of the distal lower limbs without any bladder or bowel involvement. On presentation, she had spastic paraparesis with power grade 4/5 and reduced sensation in all modalities from the L2 dermatomal level below.

Imaging

The first case shows spinal digital subtraction angiogram (DSA) (Fig. 1) showing a perimedullary arteriovenous (AV) fistula type 3 at D10–D11 levels with multiple feeders from posterior and anterior spinal artery. The patient underwent endovascular intervention for the fistula.

Treatment

She underwent spinal *N*-butyl cyanoacrylate (NBCA) embolization for the same. During the procedure, there was inadvertent rupture of the radiculopial artery and obliteration of the perforated feeder was done using 0.5 mL of 15% NBCA glue. However, the check angiogram showed persistent contrast extravasation. Due to this, the procedure was then abandoned. Magnetic resonance imaging (MRI) dorsolumbar spine (Fig. 2) was obtained which showed spinal subdural hematoma and intramedullary hematoma, and compression of the cord with the cord signal changes. Post procedure, she was conscious and obeying without any fresh sensorimotor deficits for the next 48 hours except for severe back pain, which was treated with analgesia, and hydration was maintained both orally and intravenously. Then she was noticed to have paraplegia, and in view of neurological deficits which developed over time and based on MRI findings, she underwent

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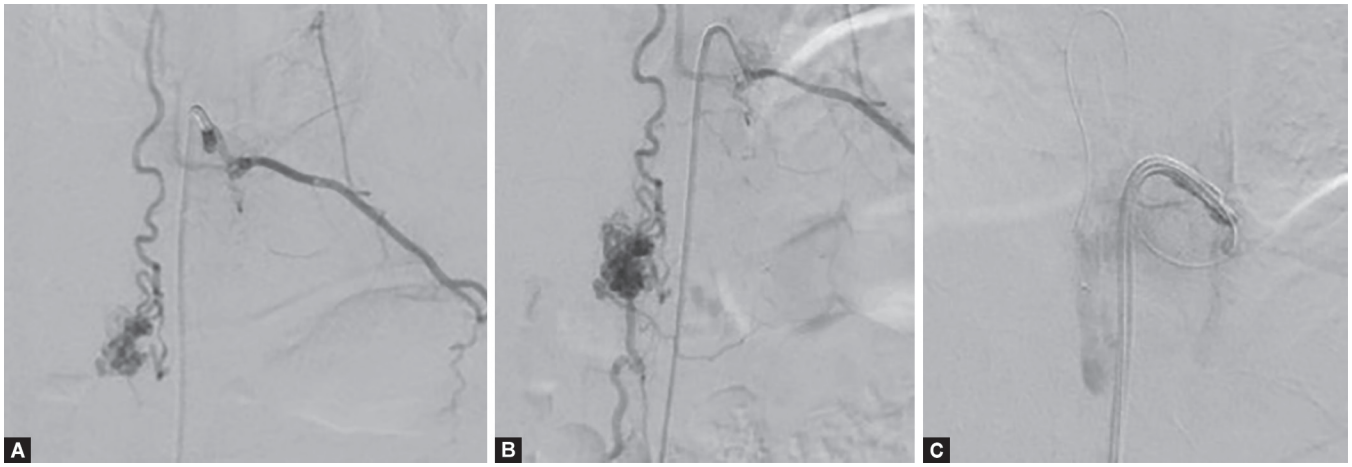
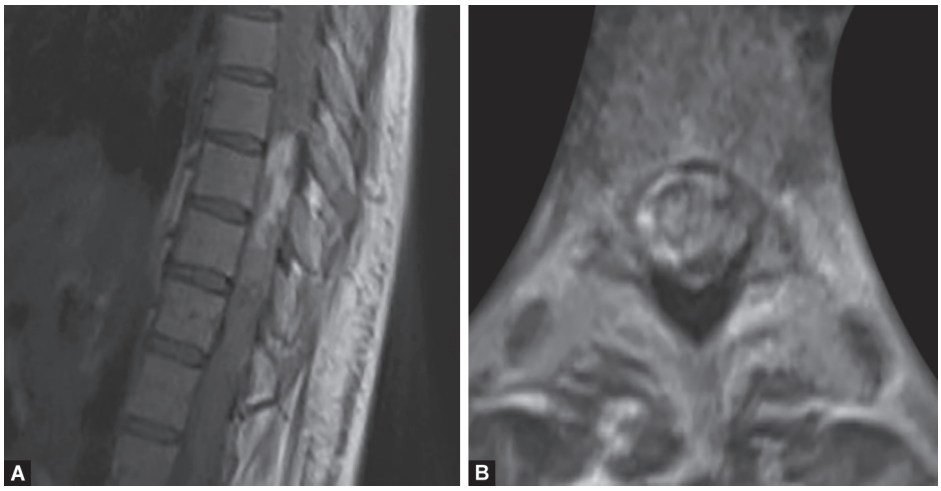


Figs 1A to C: Spinal DSA images: (A) and (B) AVF at D10 and D11 levels, and (C) rupture of the radiculopial vessel and extravasation of the embolic material



Figs 2A and B: MRI images (A) T1 weighted image sagittal section at the D10–D11 levels showing extravasated embolic material and (B) T2 weighted image showing dorsal cord compression due to hematoma and extravasated material

D6–D12 decompressive laminectomy, evacuation of subdural hematoma, and extravasated glue material causing compression of the cord.

Postoperative Course

She improved slowly over a period of 4 days. Her paresthesia in both lower limbs persisted, and her lower limb power was 4/5 distally and 2/5 proximally. Follow-up after 1 year showed that she improved minimally. She preferred wheel chair to walk due to proximal weakness. The follow-up angiogram showed residual perimedullary arterio-venous fistula (AVF) (Fig. 3) and the MRI scan showed postoperative changes with no canal stenosis (Fig. 4). The patient was offered further surgical or radiological intervention. However, in view of previous complication, she was not keen on further intervention. She was given medication for symptomatic relief of paresthesia and advised to continue physiotherapy.

Case Report 2

A 26-year-old male patient presented with the history of lumbar region back pain for 1 year, gradually progressive and increased in the last 6 months. He also developed difficulty in walking from the last 1 month and urinary urgency. On presentation, he had spastic paraparesis with power 3/5 proximally and 2/5 distally and reduced sensations below the D10 level.

Imaging (spinal DSA) shows spinal AVM at D8–D9 levels with multiple feeders from D7–D11 (Fig. 5) with enlarged venous saccules extending intradural on the left side of the spinal cord into the canal.

The patient underwent embolization of epidural AVM at the D10 level (Fig. 6) with NBCA and there was no complication during the procedure but occurred immediately post procedure. The patient developed weakness in both the lower limbs and MRI showed glue cast in the spinal canal (Fig. 7). The patient underwent D9–D10 decompressive laminectomy (Fig. 8). The postoperative patient improved to preoperative power in both lower limbs.

Discussion

Complications associated with onyx embolization with spinal AVF have been discussed by Kim et al.[3] Similarly in the case of treatment of intracranial dural AV fistula, the patient developed cervical cord infarction due to occlusion of vertebral artery.[4] Retrieval of the catheter with both onyx and NBCA may be associated with traction

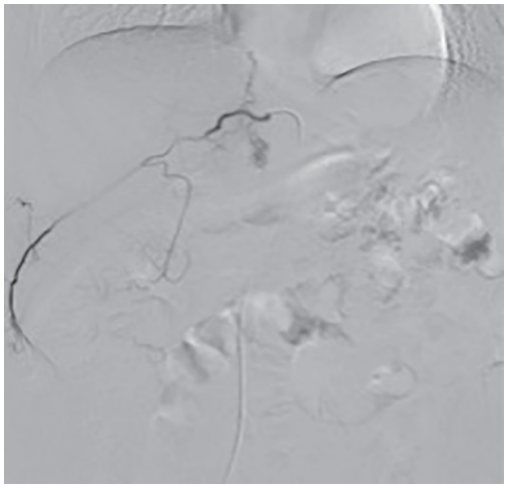


Fig. 3: Spinal DSA obtained after 1 year showing residual AVF

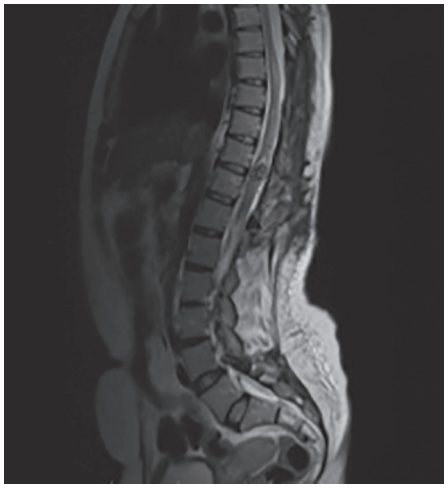


Fig. 4: MRI T2 weighted image showing residual AVF and postoperative changes at D10 and D11 levels after 1 year

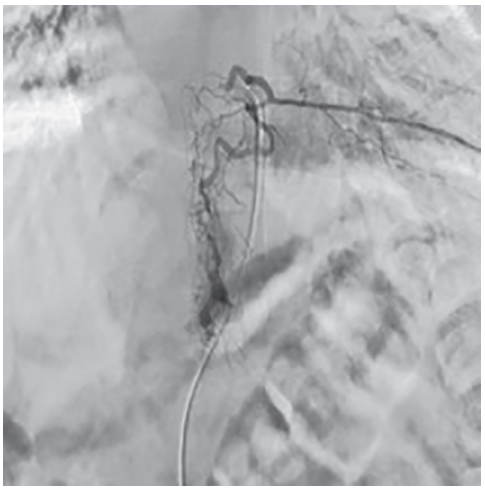


Fig. 5: Preoperative angiogram showing spinal AVM

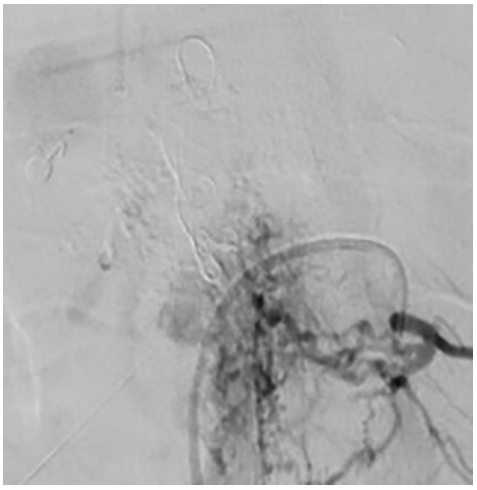
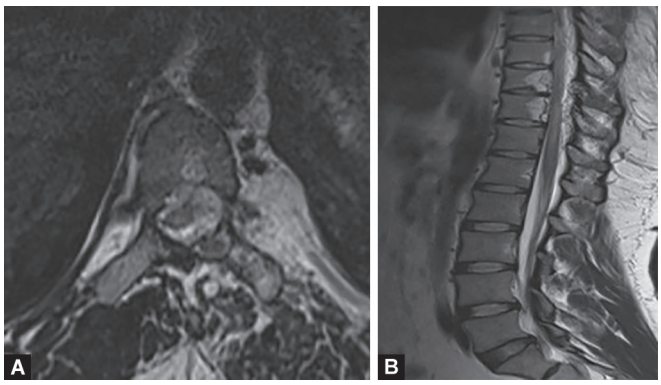


Fig. 6: Right D10 coiling and glue insertion



Figs 7A and B: (A) MRI T2 weighted image axial section and (B) MRI T2 weighted sagittal image showing glue in the canal at the level of the D10 vertebral body

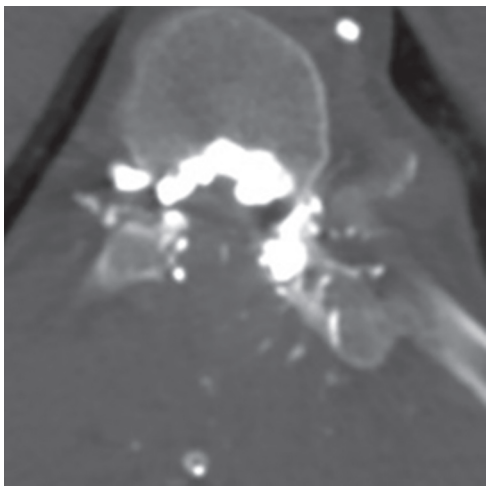


Fig. 8: CT scan dorsal spine axial section image showing laminectomy and residual glue in the canal

force on radicular artery in our patient. The rupture of radicular artery probably resulted in extravasation of glue material and subdural hematoma. Some part of hematoma in the first patient was intramedullary and part of it was subdural, this is because AVF is perimedullary in location. The usefulness of the surgery was debated since the differential diagnosis of MRI finding was venous infarct. However, there would not have been any debate in a similar case of supratentorial AVF. Since the patient had developed weakness, and MRI showed the extravasated material and the hematoma, decision was made to evacuate the same. The

decompression was useful as evidenced by improvement in power in the lower limb. Kim et al. reported a similar complication, but in their patient, there was no neurological deficit.[3]

The second patient had presented 2 years after the first patient with a similar complication, also he had presented with rapid weakness intraprocedural, and CT scan had shown extravasated embolic material in the spinal canal, hence he was taken for immediate decompressive laminectomy and removal of the offending material, resulting in immediate post decompression improvement in motor power of both lower limbs.

Conclusion

We suggest that the occlusion of spinal vascular malformations with NBCA/onyx may be associated with the risk of radicular artery rupture resulting in hematoma or extravasated material causing compression and neurological deficit. Following intervention if the patient develops weakness, it is generally thought to be due to ischemia, and not due to extravasated material (glue + hematoma). When imaging shows the mass effect due to this complication, the patient should undergo decompression instead of taking a pessimistic attitude of not doing anything due to supposedly arterial ischemia and infarct.

Informed Patient Consent

The patient has consented to submission of this case report to the journal.

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