Early Disappearance of Cord Signal Changes Following Endovascular Treatment of a Spinal Dural AV Fistula

G. SRIKANTH S., D. SIVAKUMAR, N. JAYAKUMAR P., M. VEERENDRAKUMAR*, M. THOMAS*

Department of Neuroimaging and Interventional Radiology

*Department of Neurology, National Institute of Mental Health and Neurosciences, Bangalore, India

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Summary

Spinal dural arterio venous fistulas (SDAVFs) are the commonest type of spinal vascular malformations and are encountered mostly among adult patients. Magnetic Resonance Imaging (MRI) in patients with SDAVFs demonstrates features like spinal cord swelling and signal changes. Cord signal changes are believed to be due to venous stasis. Treatment of a spinal vascular malformation may result in complete or partial resolution of MR findings with associated clinical improvement. The earliest documentation of reduction or disappearance of cord signal changes on post treatment MR images, was one month later in one series in the literature. We present and discuss disappearance of cord signal changes on MRI within 72 hours following endovascular treatment by liquid glue injection in a patient with SDAVF.

Introduction

Spinal vascular malformations (SVMs) constitute about 3-16% of all spinal space occupying lesions¹. Spinal Dural Arterio venous fistulas (SDAVF) are the commonest malformations in adults and constitute about 80% of all SVMs¹. SDAVFs are usually present adjacent to an intervertebral foramen getting their blood supply from a dural branch of a radicular artery and

draining into pial veins of the cord¹. MRI plays an important role in the diagnosis and treatment of such lesions and findings include swelling of the affected segment of the cord, signal changes, dilated medullary veins and contrast enhancement of the cord parenchyma². These changes in the cord are believed to be due to venous stasis, which results in edema and ischemia. Clinical symptoms in such patients include progressive motor and sensory deficits followed by bladder dysfunction¹. These symptoms are the result of spinal venous hypertension, which can be reversible if treated early¹. Treatment of SDAVFs includes surgical ligation of the fistula or superselective injection of liquid glue into the fistula by endovascular technique^{3,4}. Treatment of a spinal vascular malformation may result in complete or partial resolution of MR findings with clinical improvement⁵. The earliest record of a disappearance or reduction of cord signal changes was one month after endovascular treatment⁵. We report a case of SDAVF treated by endovascular glue injection and disappearance of cord hyperintensity on MR imaging within 72 hours of treatment.

Case Report

A 45-year-old gentleman presented with an acute episode of moderate to severe low backache which had occurred four months previous-

Figure 1 TSET2W sagittal (A,B) and axial (C) MR images of dorsal and dorsolumbar cord showing increased signal of the cord. Note also multiple abnormal flow voids in the posterior subarachnoid space (arrow). Post Gadolinium SET1W sagittal image (D) shows enhancing vessels on the dorsal surface of the cord (arrowheads).

ly and was initially managed conservatively. Two months later, he developed numbness in the lateral aspect of the right foot and back of the thigh. MRI examination of dorsal and lumbar spines, at that time was reported to be normal. He was prescribed analgesics and physiotherapy. However, he did not make significant improvement and four weeks later he developed urinary hesitancy and erectile dysfunction. A repeat MRI with gadolinium now showed extensive diffuse hyperintensity of the lumbosacral and thoracic cord up to D5 on T2W images and enhancing flow voids situated on the dorsal surface of the thoracic cord (figure 1). Another four weeks later he developed sudden onset of weakness of both lower limbs simultaneously.

On neurological examination he had complete flaccid paraplegia (MRC grade 0/5 bilaterally, with absent knee and ankle jerks) and a sensory level for pinprick at D4 level. Cranial nerves were normal and upper limb examination was unremarkable. Catheter spinal angiography showed a SDAVF fed by the left D6 intersegmental artery with draining veins seen on the dorsal aspect of cord in the thoracic region (figure 2). A 5F-guiding catheter was placed in the feeding intersegmental artery, and the fistula was catheterized using a 1.2F micro catheter over a 0.010" microguidewire. Super selective injection of contrast material revealed good opacification of the fistula and the draining veins. A dose of 0.2-0.3 ml of 50% Histoacryl and Lipiodol mixture was injected under continuous angiogram acquisition to fill the proximal portion

of draining vein, fistulous site and the distal segment of feeding artery. A post embolization check angiogram revealed absence of the fistula and draining vein (figure 2). After the treatment he started improving within the next day, developing the ability to move his toes and foot on the left side. Sensory symptoms also improved. He showed further improvement during 72 hours and a repeat MRI performed at this time showed complete resolution of cord T2 hyperintensity (figure 3). At the time of discharge he was able to stand for a few minutes and walk with the help of a support.

Discussion

According to their angioarchitecture SVMs are classified morphologically as: 1) Type 1- Dural AVF, 2) Type 2-intramedullary arterio venous malformations (AVM), 3) Type 3 - juvenile malformations with intramedullary AVM with extramedullary and extraspinal components and 4) Type 4 - intradural extramedullary (perimedullary) arterio venous fistula⁶. Type 1 is usually seen in males during 5th to 8th decades with clinical features of chronic progressive myelopathy due to venous hypertension of spinal cord. Both type 2 and 3 have an intramedullary compact AVM nidus and present at younger age with clinical features caused by an acute intramedullary haemorrhage and have a poor prognosis. Type 4 malformations are encountered during 3rd to 6th decades with progressive neurological deficits.

Figure 2 Pre and post embolization selective spinal angiogram (A,B) showing abnormal dilated veins (arrow), which were not visualized in the post embolization check angiogram (B).

SDAVF (Type 1 SVM) is believed to be acquired lesion and commonly encountered below the midthoracic spine, where increased venous pressure exists in a standing position^{7,8}. Shunt in SDAVF results in blood flow into the intradural perimedullary coronal venous plexus, which becomes dilated, tortuous and elongated. The absence of valves between the coronal venous plexus and the intramedullary radial veins facilitate the venous pressure being transmitted to the cord tissue causing venous stasis and ischemia⁹. Clinical symptomatology in these patients is usually due to the presence of spinal cord venous hypertension and results in progressive features of myelopathy with both motor and sensory deficits^{1,10}. Our patient also presented with progressive sensory and then motor disturbances of both lower limbs; numbness started initially on the right side and progressed over the next two days to involve the left side. In addition, our patient also presented with bladder and sexual dysfunction as reported in previously published cases¹. Exacerbation of symptoms by physical exercise reported in the literature was also noticed in our patient¹. MRI plays an important role in the diagnosis of SDAVFs demonstrating dilated pial veins as flow voids in cerebrospinal fluid space¹. MRI findings in SDAVFs include abnormal signal intensity on T1W and T2W images¹. Slow flowing veins and small flow voids can be missed on routine MRI however, use of gadolinium and gradient echo MRI may improve the sensitivity^{1,11}. Other findings, like T2 hyperintensity of the cord is due to

edema and in more advanced disease, ischemia and infarction. Cord infarction is also shown to enhance on gadolinium MRI scans². Such patients with enhancing infarction might not show improvement following intervention irrespective of duration of clinical symptomatology².

Catheter spinal angiography is the diagnostic gold standard however and, requires extensive opacification of bilateral iliac, lumbar and, intercostals arteries. In addition lesions above the midthoracic level may require aortography, bilateral vertebral and ascending cervical artery angiographies⁶. Endovascular and surgical interventions are the mainstay of treatment for SDAVFs. The aim of intervention, including the endovascular technique, is to isolate the vein draining the fistula from the perimedullary veins. Endovascular technique involves the in-

Figure 3 Post embolization TSET2W sagittal (A) and axial (B) MR images showing disappearance of cord signal changes.

jection of liquid adhesive like N-butylcyanoacrylate into the fistula^{12,13}. Other embolizing agents like polyvinyl alcohol particles (PVA) is not recommended as the material of choice because of significant recurrence¹⁴. However, PVA and coils are used to occlude normal branches or collateral circulation to assist good penetration of liquid embolizing agent into the fistula^{12,15}. Contra-indications for embolization include: difficulty in catheterization of feeding pedicle and the common origin of radiculomedullary & radiculopial branches where surgical intervention could be offered¹⁵. Glue embolization offers the benefits of less invasiveness and early rehabilitation and in general the results of glue embolization are better than surgical ones with regard to sphincter disturbance and paraesthesias^{13,15}. Inadequate embolization is regarded as failure to embolize the proximal portion of the draining vein and may result in recurrence of fistula¹². Hence such patients need close follow up and repeat MR imaging is advisable³. Nearly 80% of cases show clinical improvement following embolization⁵. Follow up MR images in treated cases shows reduction or absence of ab-

normal flow voids and cord T2 hyperintensity⁵. The earliest documentation of disappearance of cord T2 hyperintensity was one month in Robert's series. Disappearance or reduction of cord hyperintensity is due to reduction in spinal venous hypertension following closure of the shunt. If the cord is edematous because of venous hypertension, T2 hyperintensity may disappear following treatment. However, if the cord is already ischemic or infarcted, cord hyperintensity may not completely disappear following treatment. In our case cord signal disappeared completely within 72 hours after the treatment and suggested edema was the cause for T2 hyperintensity. This was also supported by absence of enhancement of the cord lesion with gadolinium in the pre-intervention MR images.

In conclusion, spinal dural AVFs usually present with progressive motor and sensory dysfunction of lower limbs. Endovascular treatment using liquid embolising material like N-butylcyanoacrylate is advisable provided super selective catheterization of the feeder is possible. Cord T2 hyperintensity is reversible if it is due to edema with clinical improvement.

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Srikanth S. Govindappa, M.D.
Associate Professor
Department of Neuroimaging
and Interventional Radiology
National Institute
of Mental Health and Neurosciences
Hosur Road, Bangalore, 560 029
India
E-mail:sgsrikanth@yahoo.com