

Spinal Arteriovenous Malformations: Surgical Outcome

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Spinal arteriovenous malformations are relatively rare, being one tenth as common as cerebral AVMs and one tenth as common as primary spinal neoplasms. The aim of this retrospective study was to see distribution of this disease in spinal cord and analyse outcome of surgical treatment in our set-up. 5 patients with neurologic deficit due to spinal AVMs were operated upon from March 2000 to Feb 2005. Age ranged from 25-45 years with mean of 35 years. There were 2 females and 3 males with sex ratio of 1:1.5 respectively. Spinal AVMs were categorized as one of 4 types based on pattern of arterial and venous supply. We found that 60% (3 cases) in our series were intradural AVMs while 40% (2 cases) were dural type. We conclude that glomus variety of intradural AVMs was the commonest. The commonest presentation was acute spinal dysfunction and paraparesis. Selective spinal angiography can be negative in spinal AVMs, (20%) in our series, in which CT angiography provided clue to the feeding vessels.

Key words: Spinal arteriovenous malformation, Paraparesis, surgical outcome

Spinal arteriovenous malformations are a rare cause of myelopathy. Frequently, AVM is not suspected and the diagnosis of tumour or disc disease is made. Dural AVMs are more common and affect predominantly middle age male. They have predilection for thoracolumbar spine¹⁷. These are thought to be acquired lesions. Intradural AVMs are considered to be congenital lesions and affect males or females of younger age group. They can affect entire spine although the common site is thoraco lumbar area.

In dural AVMs the onset of symptoms⁷, in 85% of patients is gradual onset of myelopathy due to venous hypertension and ischemia. They never present with subarachnoid haemorrhage. Intradural AVMs may have acute presentation in 37% of patients due to subarachnoid or intramedullary haemorrhage. In rest of patients it is due to ischemic myelopathy and is slowly progressive. The aim of surgical intervention in dural AVMs is to eliminate venous congestion of cord and to prevent further myelopathy. For intradural AVMs the surgical aim is to excise the nidus while preserving blood supply to spinal cord. Although endovascular treatment may help in partly or completely achieving above goals, it is not widely available in Pakistan and our patients are mostly left with surgical option. The aim of present study, carried out on series of 5 patients with angiographically proven spinal AVMs was to evaluate the safety of surgery and determine main causes and consequences of unfavourable outcome.

Material & methods:

It was a retrospective study. Names of patients who underwent surgery for spinal AVMs over last 5 years between March 2000 to Feb 2005 were noted from theatre register. The admission notes of these patients were studied in detail for age, sex, neurological status at the time of admission, findings of investigations, surgical approach, postoperative course, hospital stay and follow up. MRI and spinal angiogram findings were discussed

with our radiologist in detail. Myelography was not done in any of patients. MRI was the basic screening test and was positive in all patients with spinal AVMs. Spinal angiography was done in all patients with MR evidence of AVM. Angiography could not pick up AVM in one patient with intradural AVM. This patient underwent CT angiography which provided clue to AVM and its feeders. AVMs were classified as dural (with nidus at dural and dorsal nerve root) or intradural with nidus on pia or within spinal cord on basis of angiogram (Table 1)



Fig 1 MRI showing haematoma in cord as high signal



Fig 2 CT angiogram showing AVM

Age of our patients ranged from 25-40 years with mean of 33 years. There were 3 males and 2 females. 3 patients had intradural AVMs (2 females & 1 male) while 2 patients had dural AVMs (both males). Patients with intradural AVMs were younger (mean 28 year) age than dural AVMs (mean 40 year) age. 2 patients presented with acute onset of backache followed by paraparesis 2 months prior to admission (2 females). While one male with intradural AVM had gradually progressive myelopathy over 3 months. All these patients had

intradural AVMs in mid thoracic spine. 2 patients (both males) presented with gradual onset of progressive legs weakness and sphincter disturbance over 2 years and had dural AVM. The AVM in both of these patients were at thoracolumbar junction. None of the patients had cervical AVM.

Table 1 Classification of Spinal vascular malformations

A. Dural arteriovenous fistula
B. Intradural vascular malformations
1-Juvenile arteriovenous malformations
2-Glomus arteriovenous malformations
3-Arteriovenous fistula
C. Cavernous angioma

MRI findings demonstrated serpentine pattern of low signal in 3 patients, high signal on T1 image in the cord suggesting sub-acute haemorrhage in 2 patients. Two patients showed areas of high signal on T2 images in conus area suggestive of ischemic changes. None of the MRI scans were negative in our patients.

Spinal angiography detected nidus of AVM in lateral aspect of spinal canal in 2 patients while in other 2 it confirmed the nidus as dense mass of blood vessels inside the cord for short segment and was considered as glomus type intradural AVM. In one female patient angiography was negative. CT angiography was performed only in this patient and provided clue to feeding vessels.

Surgery was performed in all patients. 2 patients with dural AVM underwent only interruption of arteriovenous fistula in root canals. 3 patients with intradural AVMs underwent complete excision of nidus which was partly intramedullary as well. Hospital stay ranged from 10-20 days with mean of 14 days.

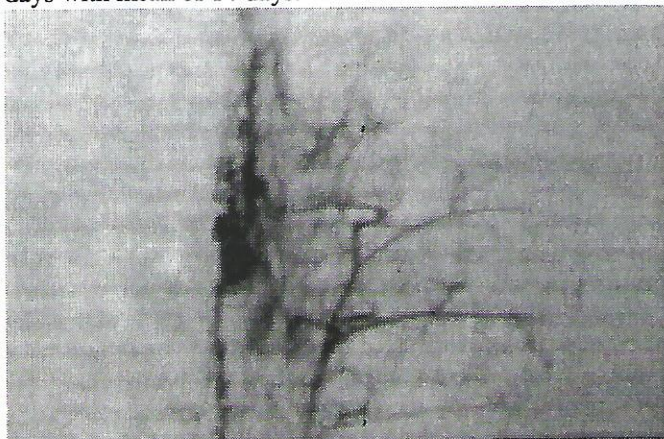


Fig 3 Spinal angiogram showing AVM nidus, arterial feeder and draining veins

Operative detail: After general anaesthesia, patient was positioned prone with bolsters under chest and pelvis to keep the abdomen free. Midline skin incision was marked according to the site of nidus on MRI/angiogram. After skin incision, thoracodorsal fascia was incised with unipolar diathermy and bilateral paraspinal muscle strip

was done. Laminectomy was performed using Leksell and Kerrison punches for at least one level above and below the malformation. The dura was incised in midline preserving arachnoid and and tackling 4/0 silk sutures were applied to keep it open. Now microscope was brought in and arachnoid was opened and dissected as second layer, while preserving underlying coronoid venous plexus.

For dural AVMs, the nidus of fistula was typically located in intervertebral foramina and lateral aspect of spinal canal. The fistula drained into the dilated tortuous intradural vein on cord surface. The site of intradural penetration of the vein draining the AVF was identified. This vessel was almost always adjacent to the site of dural penetration of nerve root. The intradural vein was then divided, after bipolar coagulation, between the site of dural entry and engorged coronoid venous plexus. Thus for dural AVM, surgical excision of dural fistula was unnecessary, as the fistula was to undergo retrograde thrombosis and fibrosis after its only venous drainage was interrupted.

For intradural AVMs, once arachnoid had been separated from AVM, the arterial feeders of malformation were correlated with spinal angiogram so they could be interrupted early and medullary feeders preserved. The malformation could be separated from surrounding cord by dissecting the gliotic plane between malformation and the adjacent cord tissue. The arterial feeders were coagulated with bipolar forceps and cut with microscissors while preserving veins till the end. At the end large vein was excised and malformation was removed en-block. Dura was closed with 4/0 vicryl and muscles and lumbodorsal fascia were closed with no 1 vicryl. 2/0 vicryl was applied to subcutaneous tissue and 2/0 silk interrupted stitches to skin. No drain was used.

Outcome: 4 patients remained same as pre op. (grade 3) at the time of discharge and improved to grade 4 power after 6 months. They were considered to have good outcome. One patient got worse to grade 1 power after operation while preoperatively she was grade 3.

Discussion:

Over the past few years, many advances in understanding and treatment of spinal vascular malformations have been made. These new fundamental observations regarding the true anatomical and pathophysiologic nature of spinal AVMs, now enable clinicians to provide safer and more effective treatment. Patients with spinal AVM, may have acute, subacute or insidious onset of symptoms, depending upon type of lesion and mechanism of cord injury.

Dural AVMs have strong male predilection and present late in life⁴. At least 80% of patients are male and 80% have symptom onset after 40 years. Since dural AVMs are located predominantly in lower thoracic and lumbar regions, patients usually have gradual onset of paraparesis and sphincter dysfunction and are unlikely to have arms involvement. Because symptoms are produced

by venous hypertension⁸, which may reach levels distant from that of dural nidus, the level of dural nidus and the spinal level of neurologic syndrome may correlate poorly. Back pain or radiculopathy often precede the onset of myelopathy. Most patients report exacerbation of symptoms during physical exertion or with certain posture. SAH is extremely rare with dural AVMs.

If they remain untreated, there is progressive neurologic decline. The study by Aminoff and Legue¹ clearly showed that 20% of untreated patients of their series required crutches or were non ambulatory by 6 month after onset of symptoms. Half of these patients were severely disabled (confined to wheel chair or bed) within 3 years of onset of gait impairment.

Intradural AVMs are result of inborn error of embryogenesis. They occur in males and females with equal frequency. Children and young adults between 15-40 years are commonly affected. Intradural AVM, particularly the glomus type, occurs more commonly in the upper thoracic and cervical region than do dural AVMs and consequently may induce neurologic symptoms in the arms. Intramedullary haemorrhage, which may present as single sudden deterioration or as a stuttering step wise decline, is certainly one mechanism responsible for myelopathy. The markedly higher incidence of associated arterial and venous aneurysms in patients with intradural AVM may in part explain the increased risk of haemorrhage. 50% of patients with intramedullary AVMs of spinal cord have gradual loss of neurologic function. This may represent different mechanism of cord injury, like mechanical compression by aneurysm or varix, ischemia secondary to vascular steal and medullary venous congestion.

In our series of 5 cases of spinal AVMs, 2 patients had dural AVMs. Both patients were 40 year old and had progressive paraparesis from thoracolumbar myelopathy. Preoperatively they were grade 3 power and both improved to grade 4 power at 6month follow up.

Our 3 cases had intradural AVMs. 2 young females with upper thoracic AVM had acute onset of severe backache followed by paraparesis. Both had intramedullary haemorrhage and sphincter dysfunction. One female got worse to grade 1 postoperatively. She had significant intramedullary component of AVM preoperatively. However the other patient improved to grade 4 power at 6 M followup. The 3rd male patient had thoracolumbar intradural AVM and had progressive myelopathy. He also improved.

Conclusion:

Our study clearly shows that for this rare spinal pathology, good surgical outcome can be achieved with proper spinal angiography and microsurgical techniques. Only one patient got worse compared with preop. status. while in the rest of 4 patients, surgical excision was curative. We envisage that with the availability of endovascular

treatment in Pakistan, it may be possible in future, to offer combined modality of treatment to our patients with spinal AVMs. We conclude that in patients with negative angiography, CT angiogram may provide clue to the feeding vessels.

References

1. Aminoff M, Logue V: Clinical features of spinal vascular malformations.. *Brain* 97:197,1974
2. Alvaro L, Areitio E, Freijo M, Gonzalez-Garay M, Oleaga L, Aranzabal I
3. Spinal cord dural arteriovenous fistulae: analysis of a series with surgical curative treatment *Neurologia*. 2002 Feb;17(2):69-76.
4. Drislane FW, Matheson JK. Spinal arteriovenous malformation. *Arch Neurol*. 2003 Jan;60(1):125.
5. Ferch RD, Morgan MK, Sears WR. Spinal arteriovenous malformations: a review with case illustrations. *J Clin Neurosci*. 2001 Jul;8(4):299-304. .
6. Hayashi K, Takahata H, Nakamura M. Two cases of spinal arteriovenous malformation presenting with subarachnoid hemorrhage. *No Shinkei Geka*. 2004 Jun;32(6):605-11.
7. Hasegawa M, Fujisawa H, Kawamura T, Yamashita J, Matsui O. The efficacy of CT arteriography for spinal arteriovenous fistula surgery: technical note. *Neuroradiology*. 1999; 41(12):915-9.
8. Inci S, Akdemir P, Ozgen T. Spinal dural arteriovenous fistula and venous aneurysm: an unusual association. *Surg Neurol*. 2003 Oct;60(4):334-7; discussion 337-8.
9. Kataoka H, Miyamoto S, Nagata I, Ueba T, Hashimoto N. Venous congestion is a major cause of neurological deterioration in spinal arteriovenous malformations. *Neurosurgery*. 2001 Jun;48(6):1224-9; discussion 1229-30.
10. Lasjaunias P. Spinal cord vascular lesions. *J Neurosurg*. 2003 Jan;98(1 Suppl):117-9.
11. Oldfield EH, Bennett A 3rd, Chen MY, Doppman JL. Successful management of spinal dural arteriovenous fistulas undetected by arteriography. Report of three cases. *J Neurosurg*. 2002 Mar;96(2 Suppl):220-9.
12. Rodesch G, Lasjaunias P. Spinal cord arteriovenous shunts: from imaging to management. *Eur J Radiol*. 2003 Jun;46(3):221-32..
13. Rodiek SO. Successful endovascular treatment of a spinal dural arteriovenous fistula with trisacryl gelatin microspheres. *Minim Invasive Neurosurg*. 2002 Sep;45(3):173-6.
14. Spetzler RF, Detwiler PW, Riina HA, Porter RW. Modified classification of spinal cord vascular lesions. *J Neurosurg*. 2002 Mar;96(2 Suppl):145-56.
15. Song JK, Vinuela F, Gobin YP, Duckwiler GR, Murayama Y, Kureshi I, Frazee JG, Surgical and endovascular treatment of spinal dural arteriovenous fistulas: long-term disability assessment and prognostic factors. *J Neurosurg*. 2001 Apr;94(2 Suppl):199-204.
16. Tai PA, Tu YK, Liu HM. Surgical treatment of spinal arteriovenous malformations: vascular anatomy and surgical outcome. *J Formos Med Assoc*. 2001 Jun;100(6):389-96.
17. Vodoff MV, Gilbert B., Paraplegia and medullary arteriovenous malformation. Role of surgery, corticosteroids and embolization *Arch Pediatr*. 2001 8(6):608-10.
18. van Santbrink H, de Witt Hamer PC. Spinal AV malformation. *Lancet*. 2003 May 24;361(9371):1766.