SPINAL ARTERIOVENOUS FISTULAE

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ABSTRACT

Spinal vascular malformation consists of normal or enlarged arteries feeding into venous channels without passing through capillaries. We present the case of a 50-year-old man who was reffered to our clinic with a suspicion of spinal arteriovenous fistulae (AVF) and a clinical course of progressive myelopathy.
Key words: arteriovenous fistulae, medulary infarction

BACKGROUND

Spinal vascular malformation consists of normal or enlarged arteries feeding into venous channels without passing through capillaries. While arterial enlargement is variable, the draining veins are typically enlarged and tortuous. Vascular malformations (AVM's and AVF's) may cause increased local venous pressure, decreased perfusion pressure, decreased tissue perfusion and tissue ischemia resulting in a slowly progressive myelopathy[1]. Eventualy increased intravascular pressure can progress to be sufficient enough to cause venous infarction of the spinal cord. There are several classifications of spinal vascular malformations but commonly used is the classification of Anson & Spetzler[2] and the revised classification of the former made by Kim and Spetzler (2006)[3]. The clinical course is variable with an insidious, acute or relapsing/remitting myelopathy. The most common type of spinal vascular malformation is spinal dural AVF, typically found in erderly men with 9:1 ratio compared to women[4]. MRI of the affected segment is the diagnostic procedure of choice in the initial evaluation of suspected AVM's and also can track the success of therapeutic interventions[5]. Contrast enhanced three dimensional spinal MR + angiography allows direct visualization of the abnormal intradural veins. Selective spinal angiography is the definitive approach to evaluate spinal vascular malformations.

CASE STUDY

We present a case of a 50-year-old man who was reffered to our clinic with a suspicion of an AVF and medulary infarction extended from T9 to T11 on the right lateral columns of the spinal cord (the suspicion was made on a previous MRI of the thoracic spinal cord). From the history we found out that in 2003 he presented left leg monoparesis followed next year by contralateral motor involvement and sensory deficits in the lower extremities, urinary and bowel incontinence. He was reffered to a neurosurgey clinic with the diagnostic of compressive myelopathy were he underwent an exploratory laminectomy at the level of T1-L1. After the surgery he was transferred to a neurology clinic were he underwent a myelography; immediate after this procedure the motor deficit became worse with plegic intensity. He was released home without a correct diagnosis and he had multiple admissions to recovery clinics but the motor deficit never improved. He came to our clinic after he made a MRI of the thoraco-lombar spine who raised the suspicion of an AVF. The neurological examination revealed plegic motor deficit of both legs, spread inequaly (the right leg was more affected), the right tight was smaller with visible fasciculations; increased tendon reflexes, plantar and rotulian clonus, bilateral Babinski sign; sensory deficit with T10 level, bowel and urinary incontinence. He performed a selective spinal angiography which revealed an intradural,

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extramedulary arteriovenous filstulae who had two feeding arteries: one from the right T12 radicular artery and another from a left L1 lombar artery, and a large tortuous draining vein suggesting an AVF Type IVB or Type C-according to the new classification. The decision was to try endovascular treatment and the next day the patient underwent angiographycally directed embolization of the malformation. The postembolization evolution of the patient was good with no periprocedural problems, but after five days the patient developed acute pain with a radicular topography (L1-L3). We started the patient on Dihydrocodeine 50 mg/day and Pregabalin 75 mg/day with a good pain response and we checked the angiographically guided embolization by performing a MRI of the thoraco-lumbar region which showed no signs of bleeding, only the medular infarction and also we found that the patient had multiple discopathies (probably due to the long period of bed rest); we also discovered the patient with diabetes mellitus type 2 associated with diabetic neuropathy. The motor and sensory deficit improved in the following days (he became paraparetic) and we obtained a good control of the pain with Pregabalin 150 mg/day and Dihydrocodeine 50 mg in case of acute pain. Later he was transferred to a recovery clinic to continue physical exercise and kinetotherapy.

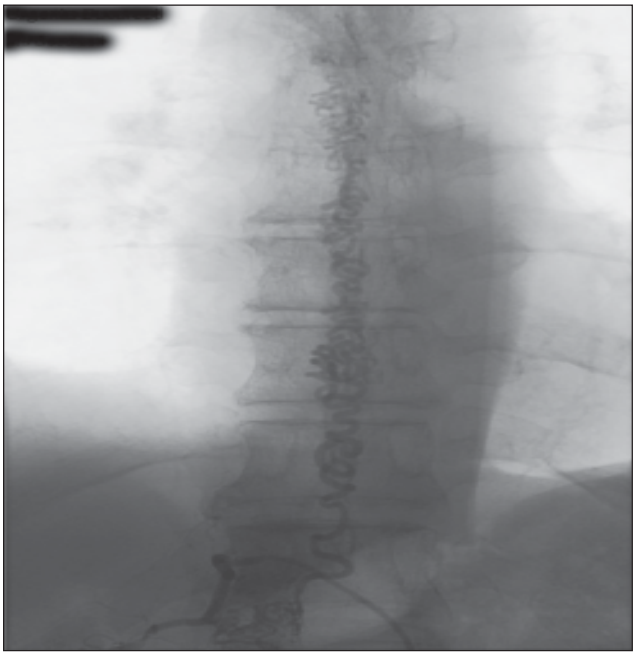

Figure 1. *Selective angiography showes the large extramedullary, intradural arteriovenous fistula – the large tortuous vein and the T12 right feeding vessel.*

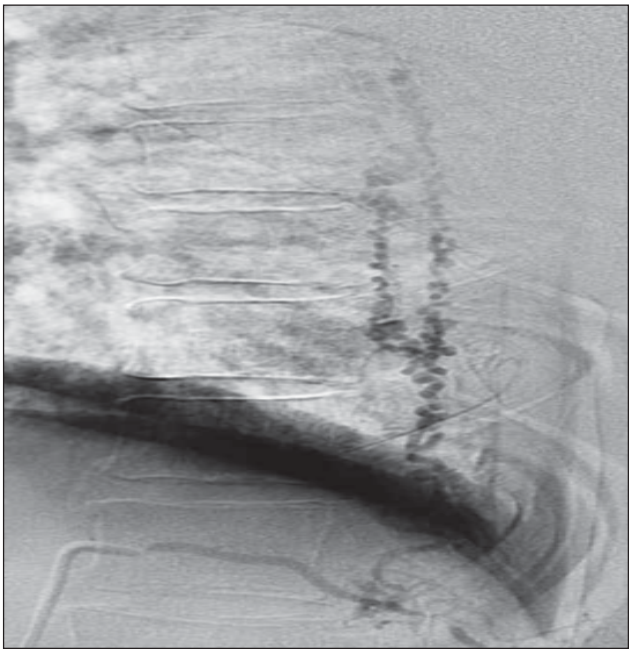

Figure 2. *Same aspect, lateral view*

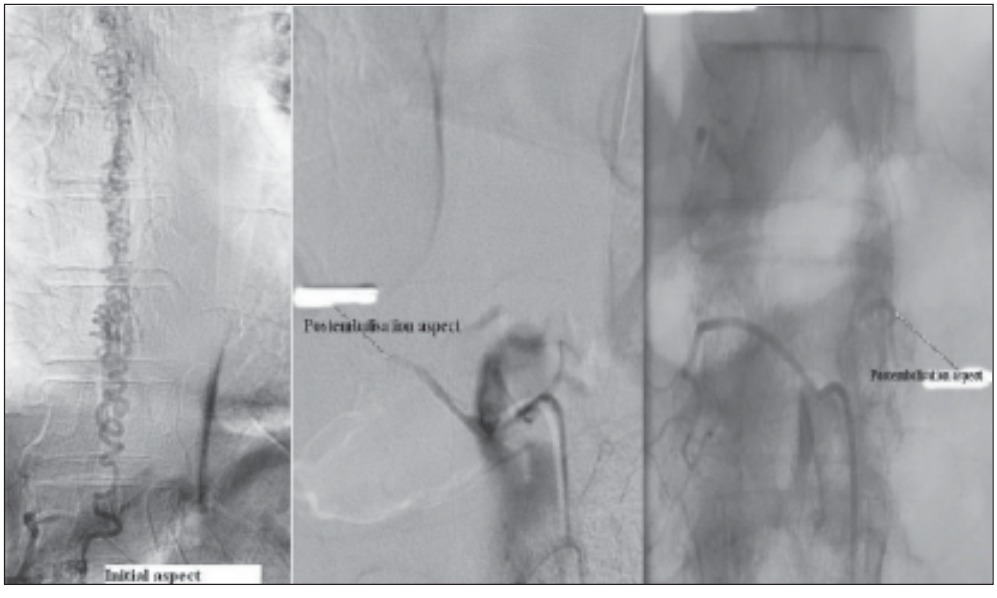

Figure 3. *Initial aspect of the AVF and the postembolisation aspect – the results are very good with no residual flow from the feeding arteries to the large drainage vein.*

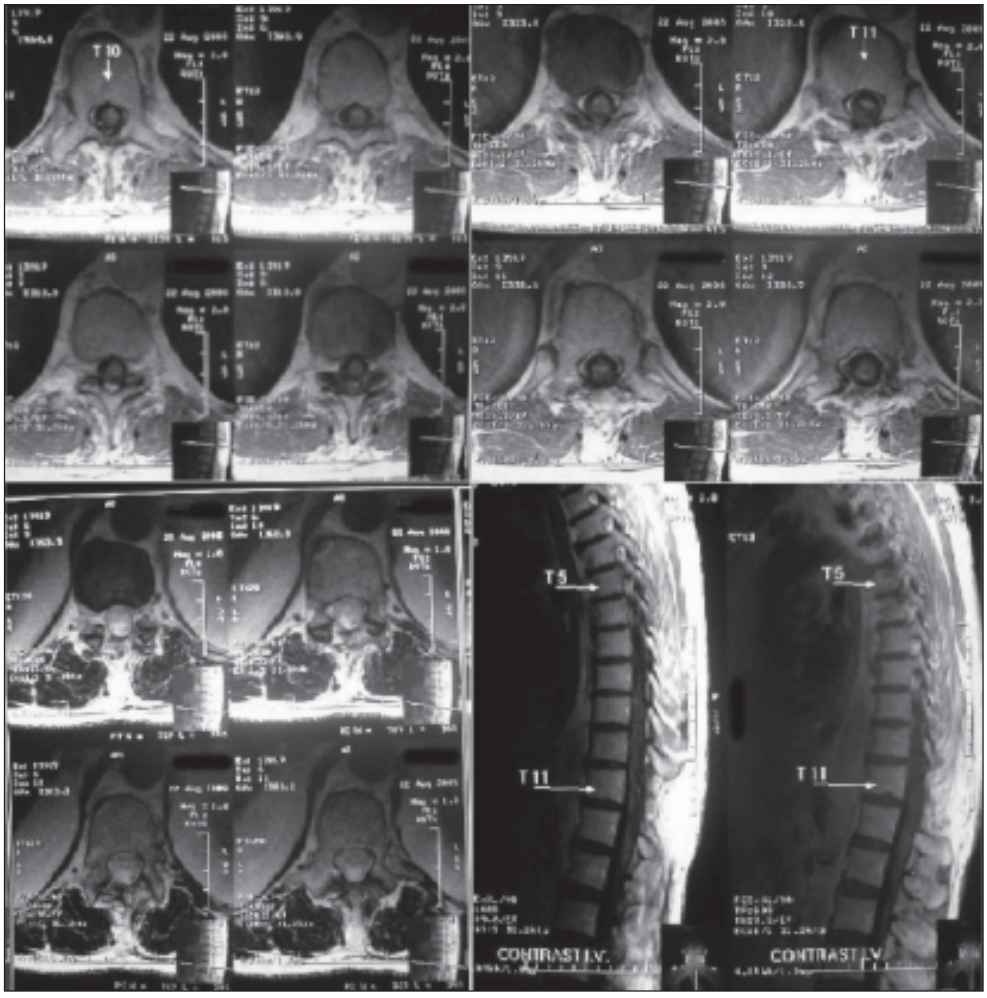

Figure 4. *T1 spinal cord MRI of the thoracic region made 5 days after the embolisation. The result is good, there is no sign of bleeding, only the old medulary infarction and the shrinking of the spinal cord in this segment after the long compression made by the AVF*

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