Spontaneous Resolution of Cerebral Arteriovenous Malformation

Sqn Ldr AK Dash*, Col PK Sahoo*, Col N Kumar†

MJAFI 2003; 59 : 157-158

Key Words : Cerebral arterio venous malformation; Spontaneous resolution

Introduction

Cerebral arterio venous malformations (AVMs) are known to have variable natural history. Amongst them, the incidence of spontaneous obliteration occurs extremely rarely in clinical practice [1]. Factors associated with spontaneous resolution of AVM are poorly understood. A rare case of spontaneous resolution of cerebral AVM is presented.

Case Report

A 37 year old serving Air Force personnel, non smoker, non obese, presented with sudden onset of left sided frontal headache, abnormal behaviour and global aphasia. He had no history of head trauma, fever, seizure or loss of consciousness. There was no past history of hypertension, diabetes mellitus or any other significant past illness.

Clinical evaluation revealed an averagely built, well-nourished individual with normal peripheral pulses and blood pressure. Neurological evaluation revealed, global aphasia with no other neurological deficit. Other systemic examinations were essentially normal. There were no neurocutaneous markers.

Laboratory investigations including haemogram and biochemical parameters were within normal limits. He was subjected to magnetic resonance imaging (MRI) which was suggestive of large AVM involving left fronto temporal region (Fig 1).

A digital subtraction angiography (DSA) was done, which revealed a large left fronto temporal AVM with arterial feeder from left middle cerebral artery (MCA) and a compact nidus of 1.5 cm. The AVM was directly draining into hypertrophied vein of Galen, right lateral sinus and straight sinus (Fig 2).

Individual was put on anti-cerebral oedema measures and anti-convulsant therapy. The AVM was large and located in the left fronto temporal region. The AVM was not amenable to surgery in view of the large size and eloquent location. Hence, a therapeutic embolization was planned via super selective DSA because of definite risk of intracranial haemorrhage in such a large AVM. Therapeutic embolization was planned in Sep 1999. Surprisingly, pre-embolic DSA revealed complete resolution of AVM (Fig 3).

Discussion

Surgical excision remains the gold standard in the management of intracranial AVM. The aims of surgery are to interrupt the natural history of AVM and future haemorrhage and simultaneously to improve the existing neurological deficit and seizure control. Other modalities available for treatment of AVM include, radio surgery, cryosurgery if the AVM is small and selective embolization alone or in combination with surgery if the AVM is large.

Small AVMs located in the polar region or non-eloquent cortical surfaces with or without bleed are managed efficiently with surgical excision without problem. Other modalities include selective embolization of feeder vessel. However, large AVMs with high flow or deep arterial feeders pose problem during one-stage surgical excision. Hence, they are more effectively managed by selective embolization alone or preoperative embolization of arterial feeders in particular followed by surgical excision with much reduced risk of intraoperative bleeding.

As reported from various series of AVMs [2], the AVMs present most commonly with intracranial haemorrhage (30% to 82%), seizure (16% to 53%, majority of which are partial or complex partial

Fig. 1 : MRI suggestive of left fronto temporal AVM.

*Graded Specialist (Medicine), *Senior Advisor (Medicine), Neuro Sciences Centre, Command Hospital (Air Force), Bangalore - 560007.
†Senior Advisor (Neurosurgery), Army Hospital (R & R), New Delhi 110 010.

Fig. 2 : DSA showing large left fronto temporal AVM with feeder from left MCA and draining to Vein of Galen, right lateral sinus and straight sinus.

seizures), headache in (7% to 48%) and focal neurological deficit (in 1% to 40% cases out of which 4% to 8% cases show progressive neurological deficit). Small and deep-seated AVMs are associated with increased incidence of intracranial haemorrhage [3]. In the haemorrhage group of unoperated AVMs, the incidence of re-bleeding is 6.9% in the first year after initial rupture of AVM, 1.91% risk per year after 5 years and 0.92% per year after 15 years [4].

Spontaneous resolution of AVMs does occur in the natural history but this phenomenon is extremely rare [1]. The incidence is as low as 1%, as reported from the largest ever reported series of AVM of 700 cases followed up over 20 years, out of which only 4 cases were detected to have spontaneous obliteration without any operative or non operative intervention. Various factors have been proposed to play a role in the pathogenesis of spontaneous resolution of AVMs. These include haemorrhagic presentation with haematoma compression of the nidus of AVM or lesional thrombosis [5].

Fig. 3 : DSA showing complete resolution of left fronto temporal AVM.

In our case the individual had no history or evidence of intracranial haemorrhage nor any history of seizure. The large left fronto-temporal AVM revealed spontaneous resolution of AVM without any kind of intervention as evident in subsequent DSA prior to embolization. Investigations to rule out thrombotic state were within normal limits. It seems there was spontaneous lesion thrombosis.

References

1. Abdulrauf SI, Malik GM, Awad IA. Spontaneous angiographic obliteration of cerebral arteriovenous malformations. *Neurosurgery* 1999;44:280-7.
2. AVM study group. Arteriovenous malformations of the brain in adults. *N Engl J Med*, 1999;340:1812-7.
3. Brown RD Jr, Wiebers DO, Forbes G. The natural history of unruptured intracranial arteriovenous malformations. *J Neurosurgery* 1988;68:352-7.
4. Mast H, Young WL, Koennecke HC. Risk of spontaneous haemorrhage after diagnosis of cerebral arteriovenous malformation. *Lancet* 1997;350:1065-8.
5. Itoyama Y, Uemura S, Ushio Y. Natural course of unoperated intracranial arteriovenous malformations : study of 50 cases. *J Neurosurgery* 1989;71:805-9.