

Microsurgical Resection of Cerebral Arteriovenous Malformation: Our Precious Experience

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Abstract

Introduction : Intracranial arteriovenous malformation (AVM) is a non-neoplastic congenital vascular abnormality which appears as a mass lesion composed of tangled blood vessels connecting arteries and veins in the brain.

Case Report : Twenty seven years old male was present in our emergency department (ED) with decreased of consciousness and recurrent generalized seizure. The physical examination reveal the patient Glasgow coma scale (GCS) of 12 points (E4M5V3). Head CT scan with contrast shows contrast enhancing lesion on the frontoparietal region with extension to right lateral ventricle which suggest vascular lesion. We performed surgical resection of the AVM using the temporary clip on M2 branch which supply the nidus to reduce the risk of bleeding on operation.

Discussion : Patients with grade I or II AVM tolerate resection with acceptable morbidity, whereas those with a grade IV or V AVM may be considered for conservative management because of the high anticipated risk of surgical morbidity

Conclusion : AVMs are abnormalities of the intracranial vessels that constitute a connection between the arterial and venous systems and lack an intervening capillary bed. Microsurgical excision of the AVM involves a craniotomy, careful dural opening with circumferential nidus dissection until complete AVM resection is achieved.

Keyword : Cerebral Arteriovenous Malformation, Vascular, Microsurgical, Neurosurgery

Introduction

Intracranial arteriovenous malformation (AVM) is a non-neoplastic congenital vascular abnormality which appears as a mass lesion composed of tangled blood vessels connecting arteries and veins in the brain. AVMs formed on the 4th-8th weeks of gestation. It can be enlarged, static or in some cases shrink.[1]

Normal cerebral vascular structure consist of arteries which flows to capillary system where it decrease the intravascular pressure before draining into the vein.[1] In AVMs, shunt presents between the high pressure arterial vessel and the low pressure vein with no normal capillaries as connector. Gross anatomy of the AVMs consist of feeding arteries, nidus and draining veins.[1][2]

Arteriovenous malformation are relatively uncommon which appears on about 4,3% of population from the autopsy data. In another autopsy series, 46 AVMs were noted among 3200 brain tumor cases. Netherlands population based data between 1980 and 1990 shows the annual incidence of symptomatic AVMs was 1,1 per 100.000 population. Most literature agree that AVM is the most common type of vascular malformation detected in the brain.[1][3]

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Multiple natural history studies consistently show the overall average patient age at AVM presentation to be in the third decade, without significant sex predilection. Patient with AVMs present most commonly after hemorrhage which accounts for 52% of the clinical presentation in this case followed by seizure in 27% of the patients. Other potential presentation variables include headaches, ischemia, and steal symptoms, which can be an incidental findings.[3][4]

Noncontrast computed tomography (CT) of the head should be the first imaging modality in any case of AVM. Enhancement is seen after contrast administration. Magnetic Resonance Imaging (MRI) is very sensitive, showing an inhomogenous signal void on T1 and T2 weighted sequence. CT angiography (CTA) is helpful in recognizing brain AVMs. CTA allows calculation of the volume of an AVM nidus, identifies associated vessels and aids in quantifying any embolic material present within the AVM. In addition, CTA is useful for characterization and stereotactic localization of the AVM before surgical resection or radiosurgical treatment.[5] Gold standard imaging technique for the diagnosis of cerebral AVMs is called digital subtraction angiography (DSA). This technique could identify arterial supply; nidus location, size, and architecture; feeding artery and intranidal aneurysm; and drainage pattern (deep or superficial).[5][6]

Case Report

Twenty seven years old right handed male was present in our emergency department (ED) with decreased of consciousness which happen for 1 year and worsen in last month. From the history taking gathered from his family, it was founded that patient has a reccurent generalized seizure for the last 10 years with the frequency of 2-3 times per month and the duration of approximately 30 seconds. Patient had been consuming valproic acid as the anticonvulsant treatment. Reccurent headache was found in the last 10 years. There are no history of projectile vomitting.

Vital sign was taken in the ED with the blood pressure of 125/75 mmHg and the Heart rate of 92x/min. Respiratory rate was found normal. No fever was recorded on the ED. The physical examination reveal the patient Glasgow coma scale (GCS) of 12 points (E4M5V3). Pupillary diameter happens to be 3 mm and reactive. Lateralization was not found. Head CT Scan shows contrast enhancing lesion on the frontoparietal region with extension to right lateral ventricle which suggest vascular lesion.

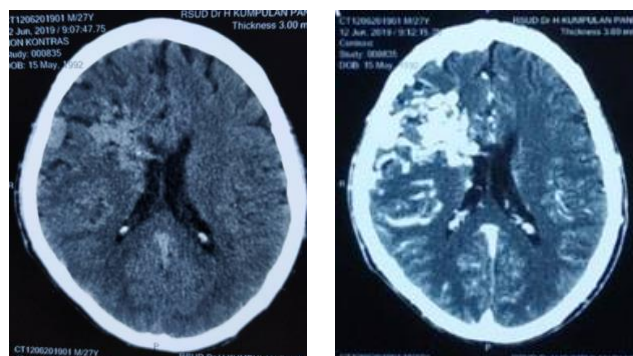


Figure 1. Serpiginous vascular enhancement on the contrast CT that distinctively typical of AVM

Patient was then admitted to ward and scheduled for digital subtraction angiography (DSA). DSA was commenced and it suggest lesion which correlate with AVM. Procedure went well and post procedure, the patient condition was stable and uneventful.

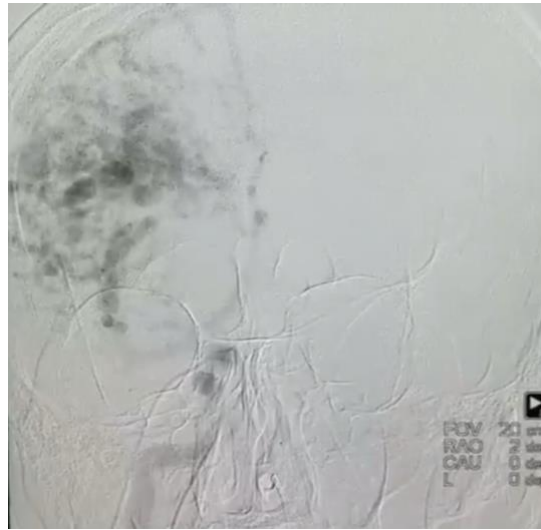


Figure 2. DSA patient

We decided to undergo surgical resection of the AVM using the temporary clip on M2 branch which supply the nidus to reduce the risk of bleeding on operation. we achieve total resection of the nidus with a good control of bleeding. Resection of AVM was commenced and we successfully achieve total resection of the AVM nidus without any significant bleeding or hemodynamic fluctuation.

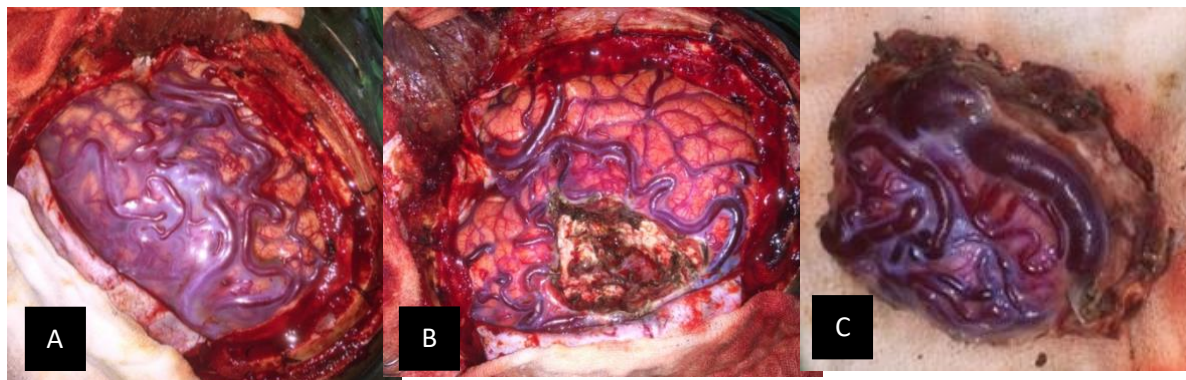


Figure 3 . (A) Durante operation exposed AVM with nidus (B) Post microsurgical excision AVM (C) AVM post removal

Post operatively patient was admitted to the intensive care unit (ICU) with the respiratory support of ventilator in controlled mode and the systolic blood pressure was within the range of 120-130 mmHg. Patient regain consciousness with the GCS score of 10 (with tracheostomy) but showing the sign of generalized seizure which we suspected from the irritation of cortex. We administer phenytoin and valproic acid as a initial treatment for the seizure and it shows good control of the seizure. We gradually taper the dose of phenytoin and valproic acid and change the seizure treatment into levetiracetam because of the increased of the liver function and we successfully achieve seizure free state. Post operatively the patient shows disability in the left side of the

extremity with the motoric score of 2 out of 5. Patient was discharged on the fourth week post operation.

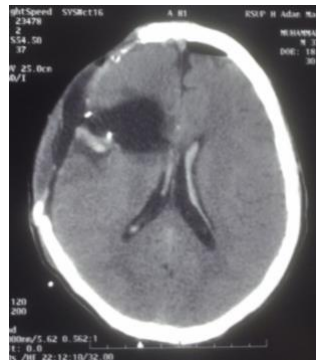


Figure 4. Post Operative CT-Scan shows complete removal of AVM without any sign of bleeding

Discussion

AVMs are abnormalities of the intracranial vessels that constitute a connection between the arterial and venous systems and lack an intervening capillary bed. Hemorrhagic presentation of AVM is associated with significant morbidity and mortality; it is an independent predictor of future hemorrhage. [5]

AVMs have three components: feeding arteries, nidus, and draining veins. The gross features of an AVM include the presence of single or multiple direct arteriovenous connections that permit high-flow arteriovenous shunting through small feeding arteries that lack a muscularis layer and the absence of a capillary bed. Cerebral AVMs may present with intracranial hemorrhage, seizures, headaches, and long-term disability; the most common presenting symptoms are hemorrhage and seizures. [6]

Conventional cerebral angiography is the gold standard in the evaluation of AVM angioarchitecture, and it shows the following essential features: the feeding arteries, location of nidus, draining veins, morphology, presence, and location of associated aneurysms, venous varices, and vasculopathic stenotic segments on arteries and veins. These features are important because they are commonly used for treatment planning [7].

Imaging modalities which are often the initial studies used to evaluate symptoms that are not specific to AVMs include CT, CT angiography, MRI, and magnetic resonance angiography. However, these imaging techniques are limited in their sensitivity and ability to provide detailed imaging of AVM architecture [7]. Each imaging technique provides its own unique strength: CT angiography provides better vascular detail of AVMs, whereas MRI and magnetic resonance angiography provide greater visualization of surrounding structures adjacent to the nidus. Furthermore, MRI can detect thrombosed vessels as hyperintense signals and show any associated hemorrhage at various stages of evolution. T2-weighted and GRE sequences are the most sensitive to breakdown products. MRI can be important for preoperative planning because it allows for an appropriate surgical approach while demonstrating the relationship of the AVM and important parenchymal structures. [6][7]

There are numerous classification for AVMs with the spetzler-martin grading system as the most commonly cited classification for predicting the morbidity of surgical resection of an AVM. It consist of three variables: size of an AVM (1-3 points), the

eloquence of the location (1 point if in an eloquent location), and the presence of deep venous drainage (1 point, if present). [8]

Characteristic	Number of points assigned
Size of AVM	
Small (<3 cm)	1 point
Medium (3–6 cm)	2 points
Large (>6 cm)	3 points
Location	
Noneloquent site	0 points
Eloquent site*	1 point
Pattern of venous drainage	
Superficial only	0 points
Deep component	1 point

*Sensorimotor, language, visual cortex, hypothalamus, thalamus, internal capsule, brain stem, cerebellar peduncles, or cerebellar nuclei.

Table 1. Spetzler Martin Grade

The sum up of all three variables provides the final grade, which ranges from I to V. Patients with grade I or II AVM tolerate resection with acceptable morbidity, whereas those with a grade IV or V AVM may be considered for conservative management because of the high anticipated risk of surgical morbidity.[8]

The Spetzler-Martin Scale is used to estimate the risk of surgical resection of an AVM with higher grades being associated with greater surgical morbidity and mortality [9]. Microsurgery is the gold standard for definitive treatment of AVMs. Microsurgical excision of the AVM involves a craniotomy, careful dural opening with circumferential nidus dissection until complete AVM resection is achieved. Post-operative angiography is performed to demonstrate complete AVM excision. The advantage of microsurgical resection is the high rate of complete obliteration, while the limitations of this approach include anatomic accessibility, edema from retraction, intraoperative rupture, resection of normal brain tissue, and feeding vessel thrombosis. [10]

The factors that dictate treatment options (which may include single or multimodal therapy) are operator skill, AVM size and location, surgical or endovascular accessibility, venous drainage, and presence of high-risk features, such as a feeding artery aneurysm [11].

Conclusion

The cure of AVM in adults is definitive with complete microsurgical resection and angiographic confirmation of obliteration. However, AVMs in children are more dynamic and may have the ability to regenerate after negative angiographic studies. Management of cerebral AVMs includes observation with medical management, microsurgical resection, stereotactic radiotherapy, and endovascular embolization. Invasive treatment modalities are the reasonable choice for ruptured cerebral AVMs due to the high rate of morbidity and mortality, and the goal of treatment is eradication of the AVM

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