ENDOVASCULAR EMBOLIZATION OF RUPTURED BRAIN ARTERIOVENOUS MALFORMATION (AVM)

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ABSTRACT
AVM of the brain are anomalies of the blood vessels that are derived from maldevelopment of the capillary network, allowing direct connections between cerebral arteries and veins. The most common presenting symptoms are cerebral hemorrhage and seizures. Current therapeutic modalities include microsurgical resection, radiosurgery, endovascular embolization, and conservative treatment. Appropriate treatment regimen according to patient characteristics and AVM morphology. Endovascular embolization can used as primary therapy, particularly for smaller, surgically difficult AVMs that contain few arterial feeders. Male, 61 years old, presented with severe headache and weakness of right limbs suddenly. Headache especially felt on the left side, with Visual Analog Scale (VAS) = 8-9, Glasgow Coma Scale (GCS) was E4M6V5, and patient underwent brain Magnetic Resonance Imaging (MRI) which revealed a hemorrhage in the left frontal lobe with flow void sign around the lesion. Patient underwent palliative targeted embolization procedure with Onyx-18 in left precentralis artery and left frontal medial artery. The results obtained changes in angioarchitecture of malformed lesion in nidus size were significant with slowing flow to drainage veins. This case was follow up after 7 months with Brain MRI which revealed no nidus and flow void sign, and from the checked cerebral angiography revealed a very small nidus with significant hemodynamic changes.

Keywords: *Endovascular, embolization, ruptured, AVM*

INTRODUCTION

AVM of the brain are anomalies of the blood vessels that are derived from maldevelopment of the capillary network, allowing direct connections between cerebral arteries and veins. Brain AVMs occur most commonly sporadically but can be associated with genetic disorder.[1] The reported incidence of AVMs is 1.34/100.000 person-years, with prevalence of 10 to 18 / 100.000, and they account for approximately 1.4% - 2% of all hemorrhagic strokes.[2] The most common presenting symptoms are cerebral hemorrhage and seizures. Focal neurologic deficits and headache may develop independent of cerebral bleeding. Current therapeutic modalities include microsurgical resection, radiosurgery, endovascular embolization, and conservative treatment. Appropriate treatment regimen according to patient characteristics and AVM morphology (size, location, artery feeder, pattern of venous drainage, and eloquence of adjacent brain).[1] Although surgical resection remains the standard for the definitive eradication of most brain AVMs, endovascular embolization has the potential to enchance the

safety and efficacy of AVM treatment when applied as adjuvant therapy before microsurgery. Endovascular embolization also can used as primary therapy, particularly for smaller, surgically difficult AVMs that contain few arterial feeders.[3]

CASE PRESENTATION

Male, 61 years old, presented with severe headache and weakness of right limbs suddenly. Headache especially felt on the left side, with Visual Analog Scale (VAS) = 8-9. Patient has no history of hypertension, diabetes mellitus, traumatic head injury, and anticoagulant therapy. Glasgow Coma Scale (GCS) was E4M6V5, vital sign was normal. Neurological examination was found slight right facial nerve palsy and right hemiparesis (4/5). Patient underwent brain Magnetic Resonance Imaging (MRI) which revealed a hemorrhage in the left frontal lobe with flow void sign around the lesion (figure 1).

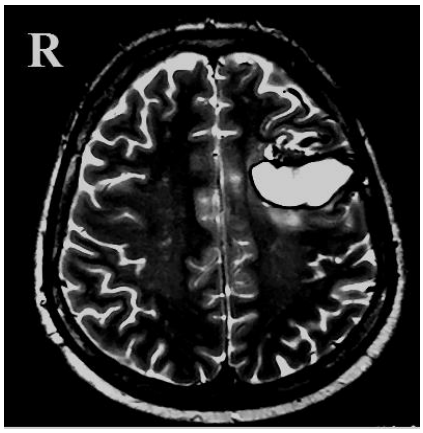

Figure 1. Brain Magnetic Resonance Imaging (pre embolization)

The cerebral angiography was performed which revealed angioarchitecture high flow nidus in the left precentralis sulcus which are supplied from the left precentralis artery, left postcentralis artery and left frontal medial artery with cortical vein drainage to the superior sagitalis sinus. There wasn't angiopathic changes (figure 2A,2B).

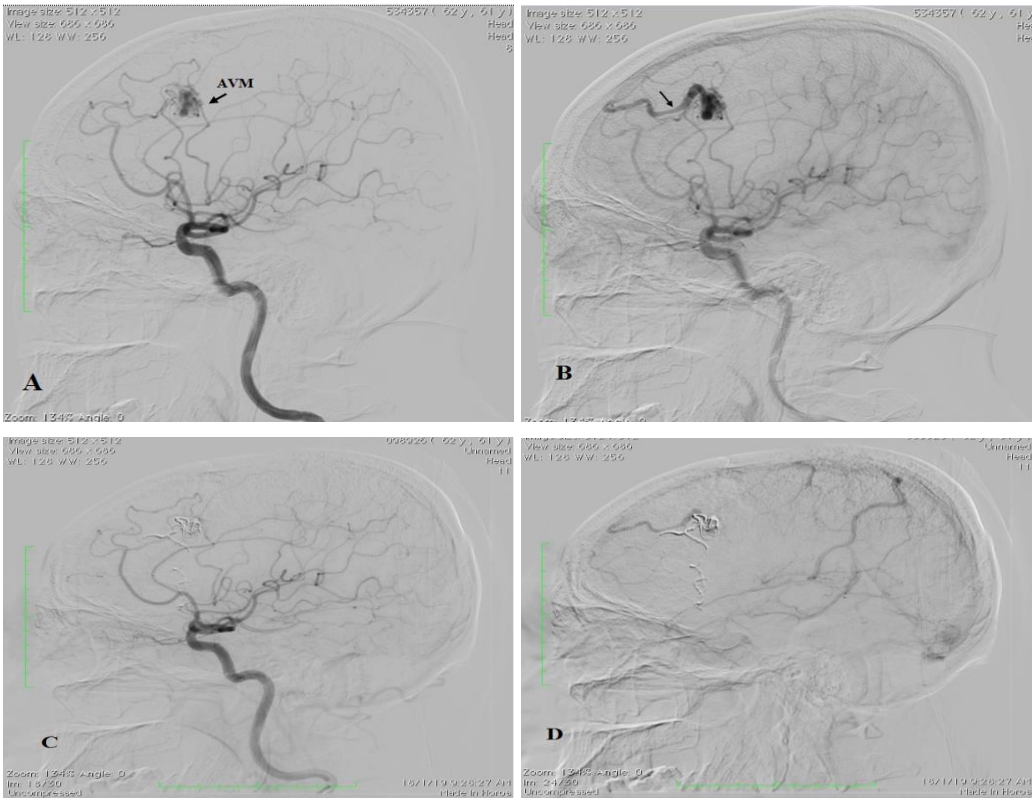

Figure 2. Cerebral DSA before embolization procedure (A, B). In figure B (arrow) revealed a stenosis in draining vein which can cause rupture of this AVM. Check angiography after embolization procedure which revealed the changes of draining flow pattern (C,D).

Patient underwent palliative targeted embolization procedure with Onyx-18 in left precentralis artery and left frontal medial artery. The results obtained changes in angioarchitecture of malformed lesion in nidus size were significant with slowing flow to drainage veins. This case was follow up after 7 months with Brain MRI which revealed no nidus and flow void sign (figure 3), and from the checked cerebral angiography revealed a very small nidus with significant hemodynamic changes.(figure 2C,2D).

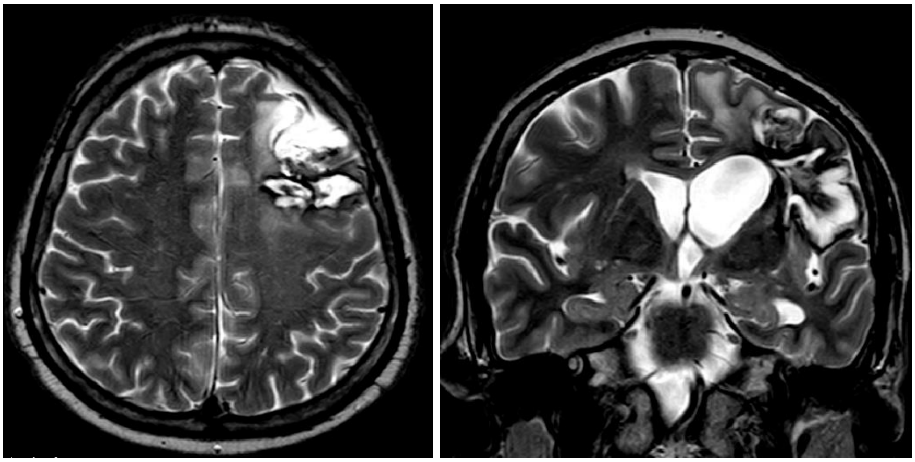

Figure 3. Brain Magnetic Resonance Imaging (post embolization)

DISCUSSION

Analysis of angioarchitecture is the first step and most important process to manage AVMs. Angiography with high resolution 3D images provides a lot of information to understand the angioarchitecture of AVMs. The angioarchitectural analysis of brain AVM should include the vascular composition of the nidus, types of feeding arteries, modes of supply, types and patterns of venous drainage. In this case, AVM was located in cortical, sulcal type, medium size, high flow, with feeder artery from left precentralis artery and left frontal medial artery, vascular composition intranidus were fistulo-plexiform component with fistulous dominant and multi compartment, and superficial vein drainage.[4]

In this case, Brain AVM presented with intracranial hemorrhage. There are several factors have been elucidated which predipose an AVM to rupture such as fistulous type of nidus, the presence of intranidal and feeder artery aneurysms, and venous anomalies (stenosis, varicosis). In this case, from the cerebral DSA was found stenosis of the drainage vein, it can be a predisposing factor for AVM ruptured. Mortality rates after the first hemorrhage are 10-30%, and morbidity rates of 25-60% have been reported.The risk of rebleeding during the first year following initial rupture are 6-17%. The high incidence of rebleeding event requires treatment plan.[3,5]

The ability to estimate the treatment risk for brain AVM is extremely difficult as the variability in complexity of AVM. The most widely utilized system for relative surgical risk analysis was spetzler and martin. The grading system is based on three criteria : AVM size, pattern of venous drainage, and neurological eloquent of adjacent brain regions. In this case, AVM had grade III lesion based on spetzler and martin, grade III are generally considered safe for surgery, with very low incidence of surgically induced neurological deficits. Endovascular embolization can be a safe therapeutic choice in this case, because AVM lesion was eligible with scoring of Lariboisiere grading was 1.[3,4]

CONCLUSION

In this case, embolization of the ruptured Brain AVM was done with follow-up after 7 months showed changed in AVM architecture so that the risk of rebleeding decreases.

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