



Brain Arteriovenous Malformations (BAVMs) and Endovascular Catheter Embolization Treatment's Safety and Complications

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Abstract

Although endovascular catheter embolization (ECE) has been accepted as a therapeutic option for arteriovenous malformations (AVMs) in children and adolescents, considerable and substantial data are still lacking regarding the outcomes of CE for AVMs. This study aimed to clarify the outcomes and the complications of ECE for the treatment of AVM in patients aged less than 18 years.

This study reports a case of an <18 years old patient who presented a year ago with headaches, weakness, back pain and sift nick. A year later the patient was subject to bleeding and a ECE was performed. Endovascular embolization was performed according to the procedures, guide and techniques for AVM embolization.

Endovascular catheter embolization (ECE) of brain AVMS is relatively safe with low rate of complications if the patient had good or excellent outcomes at discharge after AVM embolization using right liquid embolic agents. Long term following up is needed to decrease the probability of the future complication and assure the patient safety.

Keywords: Endovascular Catheter Embolization; Brain; Brain AVM; Vascular Lesion; Arteriovenous Malformation (AVM)

Introduction

Brain AVMs result in 35% – 55% of haemorrhagic strokes in children [1] with an incidence of significant mortalities and morbidities [2]. In Spinal arteriovenous malformations (AVMs) are a rare condition of abnormal collection of blood vessels in the spinal canal located on, in, or near the spinal cord which have a direct connection between the arterial system and the venous system without intervening capillaries. Brain AVMs are also rare and affect less than 1 percent of the population. AVM use focal and more complex, intraparenchymal conglomerations of dilated arteries and veins that suffering from having normal vascular organisation at the subarterilar level. Its vascular lesions and the lack of normal

capillary bed can result in significant morbidity and mortality [3]. As shown in figure 1, the AVM is a tangle of abnormal and poorly formed blood vessels.

Thus these vessels have a higher rate of bleeding than normal vessels or arteries and veins. Figure 1 presented by MAYO Foundation shows the different between the normal and AVM blood flow.

AVM cause that blood passes quickly and directly from the arteries to the veins, bypassing the capillaries. This abnormal process is disrupting the normal blood flow and depriving the surrounding tissues of oxygen. When the AVM is in the brain, the bleeding can lead to stroke and/or brain damage because the blood flow inter-

ruption prevents surrounding cells from obtaining basic oxygen needs, hence, leading to the deterioration or death of cells in the related tissues [4].

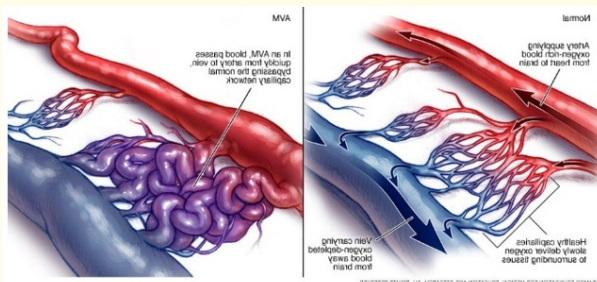


Figure 1: AVM Blood flow (MAYO foundation for medical education and research).

The exact reasons and causes of AVMs are still unknown or not clear. Majority cases of AVMs are present at birth but they can occur later in life. They are rarely passed down among families genetically. There are no known risk factors either for arteriovenous malformation. The AVMs condition and symptoms occurs almost equally in men and women with a little higher tendency for men.

A brain arteriovenous malformation symptoms vary widely from person to person depending on the age, severity and location of arteriovenous malformation [5]. Some people may not develop noticeable symptoms for many years, if not at all until the AVM ruptures, resulting in bleeding in the brain (hemorrhage). Others may experience symptoms that weaken or threaten their lives. Symptoms usually occur when people are in their twenties, although approximately 20 percent of people diagnosed with venous AVM are younger than 16 years old. Of about half of all brain AVMs, hemorrhage is the first sign. Other people with brain AVM may experience signs or symptoms other than bleeding related to the AVM such as:

- Seizures
- Headache or pain in one area of the head
- Severe headache
- Stiff neck
- Sudden numbness, tingling, Weakness, numbness or paralysis

- Vision loss
- Difficulty speaking
- Confusion or inability to understand others
- Severe unsteadiness
- Sensitivity to light.

Case Report

A 14 years patient presented with a recent brain bleeding. It was decided that the patient needs acute endovascular catheter embolization. The patient medical records, operative reports, endovascular reports and radiographic images were re-reviewed. The demographic data about the patient including age, sex, presentation date, presenting symptoms and history of previous hemorrhages and any prior treatment were studied to confirm the Spetzler –Martin grade for the AVM [6].

The patient had one year history of different signs and symptoms of AVMS such as severe headache, back pain and stiff neck. A year ago, a lumbar puncture (LP) procedure was performed for the patient by the insertion of a needle into the spinal canal to collect and examine the fluid that surrounds the brain and spinal cord (cerebral spinal fluid). The result of the analysis showed a clear case of AVMs and was treating by suitable and appropriate medications. By the end of the first year, the symptoms and signs developed and was more complicated in form of mild movement problems, frequently Pain, tingling and numbness in addition to the Severe headache, Weakness, Difficulty speaking.

Result

Another recent clinical examination and analysis of the C.T Angio Brain in three phases has been performed (Figure 2) to produce pictures of blood vessels in the brain.

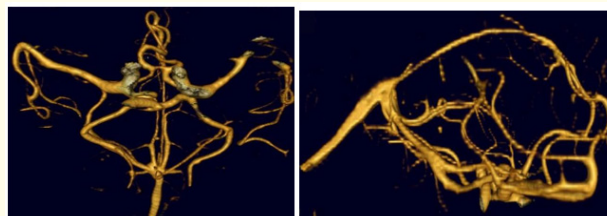


Figure 2: a. C,T Brain Angio; b. CT Brain Angio - Lateral View.

Digital Subtraction Angiography (DSA) was also conducted to provide an image of the blood vessels in the brain in order to be able to detect a problem with blood flow.

As shown in figure 3 and table 1, the Spetzler:

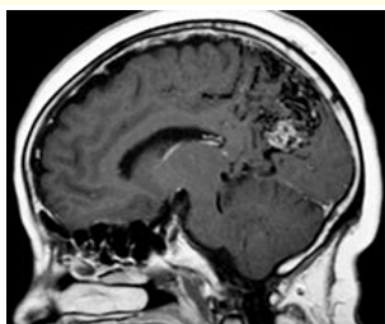


Figure 3: Grade 4 or 5 AVM are large, deep, and adjacent to eloquent brain.

A Grade 1 AVM considered as small, superficial, and located in non-eloquent brain, and low risk for surgery.

Middle Cerebral Artery (MCA) was used as a Detection of Arterial Lesions. As shown in Figure 5, the MCA cerebral artery is the largest branch of the internal carotid artery [7-9].

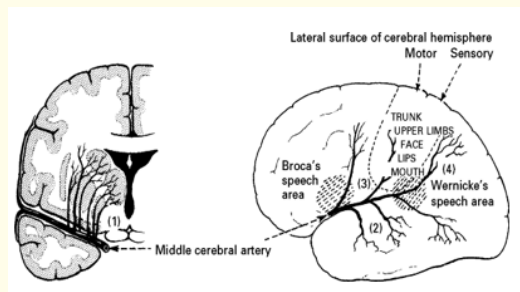


Figure 4: MCA.

Factor	Measurement parameter	Score
Size of AVM nidus		
Small	0-3 cm	1
Medium	> 3-6 cm	2
Large	> 6 cm	3
Location		
Loquence of adjacent brain	Non-eloquent	0
	Eloquent	1
Venous Drainage		
Superficial veins only	Non- present	0
	Present	1
Deep veins		

Table 1

Grade 6 AVM is considered inoperable lesions. The score correlates with operative outcome.

Martin Grading Scale allocates points for different characteristics or parameters of intracranial AVM to estimates the risk of open neurosurgery for a patient with AVM, by evaluating AVM size, pattern of venous drainage, and brain location of eloquence. It gives a grade between 1 and 6.

The knowledge of the origin and anatomic features of the AVMs feeders is important in the explanation of neurologic signs, and in a decision regarding the endovascular embolisation, neurosurgical and radiosurgical treatments. However, accessory middle cerebral artery AMCA can also be combined with different diseases such as intracranial aneurysms, cerebral infarction, and moyamoya disease [10-12] but an MCA combined with an AVM is very rare. The relation between AMCR, AVM and the blood vessels are illustrated in figure 5.

Li, *et al.* [13] illustrate in Figure that intraoperative relation and correlation between (A) which is the AMCA and the AVM (B). A is originating from the anterior communicating artery which is seen at the left of the chiasmatic cistern. The AVM (B) is located on the deep Sylvian fissure. (C and D) show the different blood vessels with an irregular shape and size. The brain tissue is also visible among the vessels.

The C.T Brain Angio examination was performed in 3 phases: pre contrast, arterial and venous. The examination reveals that an abnormal intraventricular vascular structure is seen within the left lateral ventricle extends to its occipital horn formed of abnormal communications between dilated arteries and veins forming an

AVM with a nidus measures is medium according to the Spetzler Martin Grading Scale, about 3x1.3 cm.

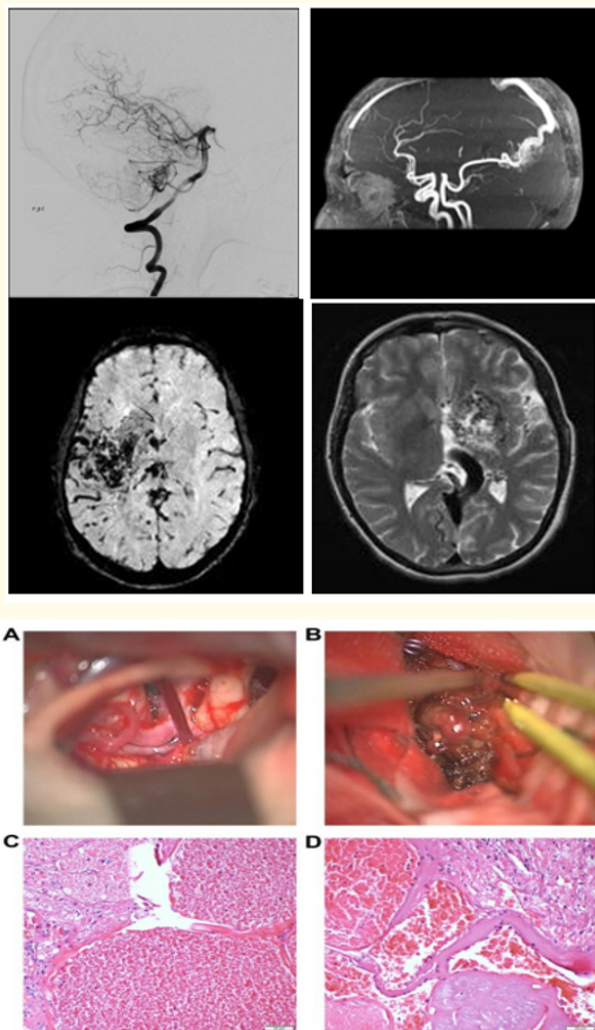


Figure 5: Intraoperative imaging and pathological images [13].

The AVM takes arterial supply from the left anterior choroidal artery as well as posterior choroidal branches of the left posterior cerebral artery and to a lesser extent the pericallosal branches of the anterior cerebral arteries with venous drainage into the left internal cerebral vein to the vein of Galen and straight sinus left interventricular AVM

Another suspected smaller AVM is seen at the right temporal lobe measures 7 mm in diameter supplied by the right MCA and drains into cortical veins for further confirmation by DSA.

The patient was treated by medication. But 2 weeks later the patient faced a bleeding symptoms. The patient was located in ICU for less than 24 hours and additional examination was performed. This clinical examination revealed that an intraventricular AVM caused intraventricular haemorrhage.

A consultation session was in place to decide the most appropriate bleeding treatment approach. However, according to J.O. Heidenreich, *et al.* [14], in addition to the medication if sufficient, there are three main alternatives or approaches for treating Brain arteriovenous malformations (BAVMs):

1. Neurosurgical interventions.
2. Stereotactic radiotherapy and irradiation
3. Endovascular and transarterial embolization.

Although a recent study shows that these conventional alternatives for the treatment of AVMs still have deficiencies [3], the endovascular therapy has prevailed as the first-choice treatment approach or strategy. Surgery and irradiation are more commonly used for treating any remnants that may have escaped embolization. Regarding decisions about the treatment approach and strategy, especially for intraventricular haemorrhage cases, a full explanation to the patients and the parents that Endovascular catheter embolization was the most rational treatment for the higher Spetzler-Martin grade (Grade II) lesions.

Endovascular catheter embolization was done same day and the patient awake calm and fine after the embolization. The patient was under observation in ICU for couple of days.

Discussion

Untreated AVM can lead to progressive disability by damaging the spinal cord and surrounding tissue. One of the basic finding of the C.T Brain Angio examination showed that the patient had an abnormal intraventricular vascular structure which caused an abnormal blood flow which formed AVM. The patient was diagnosed with having AVM in the cerebellum. The symptoms were considered to be caused by medium hemorrhages around AVM. The efficiency

and procedural risk of all 3 treatment approaches have been thoroughly examined before the final decision about the endovascular catheter embolization.

Endovascular embolization was performed according to the procedures, guide and techniques for AVM embolization [15]. The procedures were conducted intra-arterially via a transfemoral route with the patients under general endotracheal anesthesia. In this procedure, the Neurosurgeon inserts a thin, long tube (catheter) into the leg artery, and connects it through the blood vessels to the brain using an x-ray. As shown in Figure 6, the catheter is placed in one of the arteries feeding the malformed venous arteries, and the embolization factor, such as small particles, injects the glue-like substance to block the artery, reducing blood flow to the malformed venous arteries.

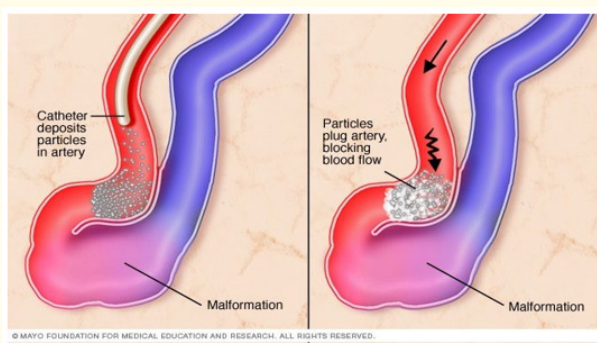


Figure 6: Endovascular catheter embolization (ECE).

The incidence of internal vein embolization is less extensive than conventional surgery. It may be performed alone, but is often used before surgical treatments to make the procedure safer by reducing the size of the malformed venous arteries, or the possibility of bleeding. Internal venous embolization may be used in some cases of large venous cerebral arteriovenous malformation to reduce stroke-like symptoms by redirecting blood to normal brain tissue.

A common complication is a post interventional bleeding in the first few days after embolization. However, its still unclear what factor puts patients at an above-normal risk for the postinterventional complication. Studies found that additional hemorrhages

can occur at a median of 59 months after treatment (range 30–151 months) and the annual hemorrhage risk in the follow-up period is calculated as 0.6%. The annual bleeding rate in the period between treatment and obliteration is also calculated as 1.3% per year and 0.2% after obliteration. Other studies disclose a wide unexplained variation of 1% to 10% in the incidence of such a complications [16].

Our study is focusing on the inexplicable finding that conservative intensive care units (ICUs) should have high incidence of such complications than in case of the other treatment approaches.

This risk of bleeding venous deformity of the brain is about 2% each year. Some cases of bleeding can be related to and associated with venous artery malformation are not detected because they do not cause significant brain damage or symptoms, but potentially life-threatening bleeding episodes may occur. About 2% of hemorrhagic cases lead to stroke each year. This bleeding in children and young people with brain haemorrhage are more common. Another complication and risk is that swelling may occur in the walls of blood vessels (aneurysms) and become more prone to rupture.

Conclusion

This study assessed the efficacy and safety of endovascular catheter embolization (ECE) for AVMs in children < 18 years old. Immediate and Longer-term follow-up suggested that ECE is an effective and relatively safe treatment for small and medium lesions, with relatively low risk of morbidity. Long term follow-up is necessary to eliminate adverse events which can occur many years after treatment.

Conflict of Interest

The authors have none to declare.

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