EMBOLIZATION OF ANTERIOR CRANIAL FOSSA DURAL ARTERIOVENOUS FISTULA

Bazli Md Yusoff[1], Ahmad Aizuddin Mohamad Jamali[1], Mohd Syafiek Abdul Haq Saifuddin[1], Mohd Shafie Abdullah[1], Abdul Rahman Izaini Ghani[2]

[1]Department of Radiology, School of Medical Sciences, Universiti Sains Malaysia, 16150 Kubang Kerian, Kelantan, Malaysia
[2]Department of Neurosciences, School of Medical Sciences, Universiti Sains Malaysia, 16150 Kubang Kerian, Kelantan, Malaysia

Corresponding author :
Bazli Md Yusoff, Department of Radiology, School of Medical Sciences, Hospital Universiti Sains Malaysia, 16150 Kubang Kerian, Kelantan. Tel No: +609767300, Fax No +6097673468

DOI: https://doi.org/10.32896/cvns.v1n1.8-10
Published: 31.12.2019

ABSTRACT
Dural arteriovenous fistulas (DAVFs) are abnormal connections between branches of the intracranial arteries and dural veins or sinuses. Advancements in the technique of endovascular embolization has made it the treatment of choice for DAVFs. The goal of treatment is to completely occlude the fistula orifice while maintaining the normal cerebral venous drainage. Depending on the site of the DAVF, endovascular treatment has its own challenges to the performing physician. In this case report, we will discuss complex anterior cranial fossa DAVFs, treatment approaches, and complications of the treatment.

Keywords: Anterior cranial fossa, Dural arteriovenous fistula, endovascular embolization

1. INTRODUCTION

Dural arteriovenous fistulas (DAVFs) are rare vascular abnormalities, which involve abnormal connections between branches of the intracranial arteries and dural veins or sinuses[1]. The exact etiology of DAVFs is unknown. A common predisposing factor for DAVFs appears to be venous sinus thrombosis[1]. DAVFs account for 10-15% of all intracranial arteriovenous malformations.

Although DAVFs can occur anywhere in the dura mater covering the brain, they occur most frequently in the cavernous and transverse-sigmoid sinuses. Patients may be asymptomatic or present with symptoms ranging from mild to severe, which include fatal hemorrhage. The venous drainage pattern of dural AVFs is the best predictive factor of severity and possible complications. Several classifications have been developed to stratify the risks of DAVFs. Both the Cognard and Borden classifications are the most widely used for this purpose[2].

2. CASE REPORT

A 52-year-old lady presented with sudden onset of unconsciousness while exercising. She was previously well, with no underlying co-morbidities. Upon arrival at the hospital, the Glasgow Coma Scale (GCS) was 3/15, the pupils were unequal and sluggish. Urgent non-contrasted CT brain was immediately performed which revealed a right frontal lobe intraparenchymal hemorrhage with associated right parietal subdural hemorrhage and midline shift [Figure 1].

The patient was immediately referred to the neurosurgery team. She underwent right decompressive craniectomy and evacuation of the clot. CT angiography of the brain was subsequently performed, showing dilated and tortuous vessels at the right parasagittal (frontal) region, which is continuous with the anterior part of the superior sagittal sinus. There was early opacification of the superior sagittal sinus in the arterial phase.

On diagnostic cerebral angiography, there was a dural arteriovenous sinus, with the feeding artery noted coming from the left ethmoidal branch of the ophthalmic artery. The dilated vessel is continuous with the anterior part of the superior sagittal sinus. Early opacification of the superior sagittal sinus was seen [Figures 2a, 2b]. After discussion with the neurosurgery team, we decided to proceed with an endovascular approach. Due to the complexity of the DAVF, we attempted trans-venous embolization via the internal jugular vein into the superior sagittal sinus. The procedure was complicated with spontaneous thrombosis at the fistula site.

The thrombosis caused spontaneous closure of the fistula at the dural side. Acute subdural haemorrhage was noted along the venous system. NCCT brain performed after the procedure showed well defined hyperdense areas, suggestive of thrombosis [Figure 3]. We proceeded with another attempt at embolization, via a trans-arterial approach. Cerebral angiography prior to second embolization showed pseudoaneurysm formation at the superior sagittal sinus [Figures 4a, 4b]. Selective cannulation of the branch of the feeder vessel with a microcatheter was done, followed by embolization with 45% N-butyl cyanoacrylate (NBCA). Complete occlusion of the fistula was achieved with no immediate complications [Figure 5]. Due to a prolonged intubation period, patient was put on tracheostomy tube and was discharged well with nursing care.

3. DISCUSSION & CONCLUSION

Anterior cranial fossa DAVFs account for about 2-3% of the total prevalence of DAVFs[2]. The specific etiology of anterior cranial fossa DAVFs is still unknown, but some cases have

been reported being secondary to head trauma[3]. It is frequently associated with intracranial hemorrhage or other neurological symptoms; 12-15% presented with headache, 5-33% present with central nerve deficits, and 44-84% present with intracranial hemorrhage[2]. Angiography remains the gold standard for diagnosis and planning of therapy[1].

DAVFs located in the anterior cranial fossa usually drain through the retrograde leptomeningeal-cortical venous system only, which categorizes it into at least type III in the Cognard and Borden classifications. This results in a higher risk of intracranial hemorrhage. Complete cure is mandatory to prevent fatal complications.

Treatment options include radiation therapy, endovascular embolization, and surgical resection. There have been good results reported from the use of stereotactic radiotherapy for the treatment of DAVFs. However, it carries an unacceptable delay of about 1 - 3 years in curing DAVFs with cortical venous reflux and therefore is not recommended as a primary therapeutic measure[4]. Trans-arterial embolization (TAE) with N-butyl-2cyanoacrylate (NBCA), trans-venous embolization (TVE) with a cortical venous approach, as well as surgery have shown similar success rates for complete occlusion of anterior cranial fossa DAVFs[2]. In terms of potential risk and technical difficulty, TVE and TAE are at par. However, they appear to be potentially riskier and technically difficult compared to surgery, which in turn is more so than radiation therapy. Anterior cranial fossa DAVFs is almost always supplied by the bilateral ophthalmic artery, in which a trans-arterial approach is difficult and dangerous[2]. Among the branches of the ophthalmic artery is the retinal artery, which supplies the optic nerve. Slight mistakes in embolization of the ophthalmic artery can cause occlusion of the retinal artery with complete loss of vision in that eye.

Anterior cranial fossa DAVFs can safely be treated via a trans-venous approach. However, trans-venous approaches are often tortuous. The micro catheter should be advanced over the guidewire very gently to avoid spasm and rupture of the draining vein, or, eventually, a venous aneurysm[5]. In our case, we observed our patient developing spontaneous thrombosis and pseudoaneurysm formation after we attempted fistula closure via a trans-venous approach. Several studies have reported the complications associated with trans-venous embolization of DAVFs. A case series by Kim et al. found common complications post trans-venous embolization of DAVFs include cranial nerve palsy, venous perforation, and venous congestion[6]. Due to the difficulty of trans-venous access, and the risk of visual deficit by trans-arterial embolization, a surgical approach is relatively safe in treating anterior cranial fossa DAVFs by disconnecting the venous connection[7].

Embolic agents that are available include polyvinyl alcohol (PVA), N-butyl cyanoacrylate (NBCA) glue, platinum or stainless-steel coils, absolute alcohol, or Onyx. PVA is the easiest material to use, but it is a temporary agent. NBCA is more permanent compared to PVA, but is more difficult to administer with limited injection time due to polymerization. Onyx is a preferred embolic material because it is mechanically occlusive but non-adherent to the vessel wall. This allows prolonged feeder injection. However, the drawbacks include reflux proximally along the microcatheter, and slow injection with delayed penetration. It will also cause prolonged stay of the microcatheter in the ophthalmic artery, increasing the risk of thromboembolism and central retinal artery occlusion[5].

Figures

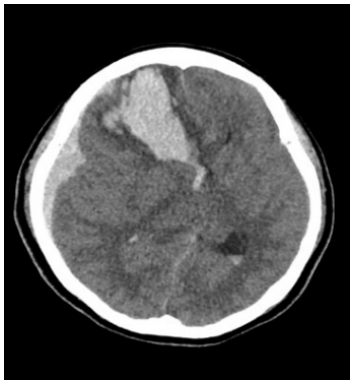

Figure 1. *Non-contrasted CT Brain showing right frontal lobe intraparenchymal hemorrhage, with right parietal subdural hemorrhage and midline shift.*

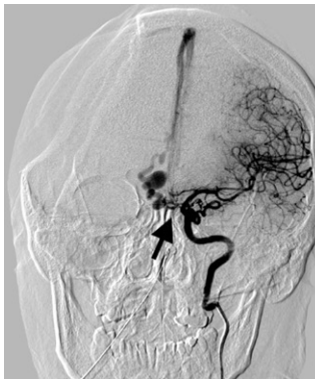

Figure 2a. *Antero-posterior projection of the left common carotid artery digital subtraction angiogram showing dural arteriovenous sinus with the feeding artery from the ethmoidal branch of the ophthalmic artery (arrow).*

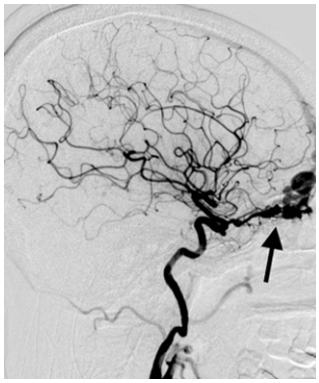

Figure 2b. *Lateral projection of left common carotid artery digital subtraction angiogram showing dural arteriovenous sinus with the feeding artery from the ethmoidal branch of the ophthalmic artery (arrow).*

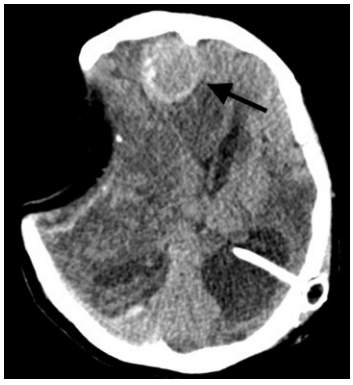

Figure 3*. Non-contrasted CT brain performed after the procedure. There is a bone defect at the right fronto-parieto-temporal region, in keeping with previous craniectomy changes. A ventriculo-peritoneal shunt tube is noted at the posterior horn of the left lateral ventricle. A well-defined hyperdense area at the right frontal lobe is seen, in keeping with thrombosis (arrow).*

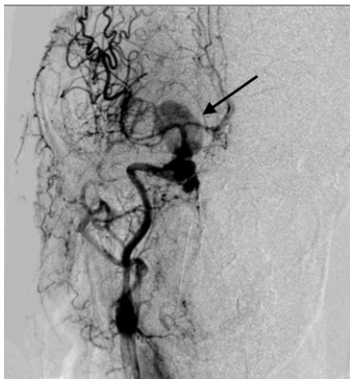

***Figure 4a**. Cerebral angiogram prior to second embolization showing pseudoaneurysm formation at the superior sagittal sinus (arrow).*

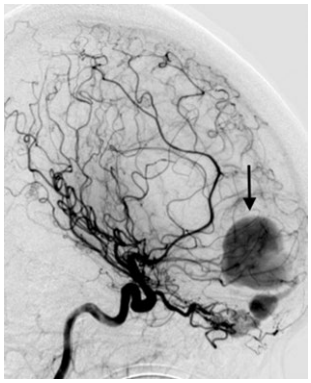

***Figure 4b.** Cerebral angiogram prior to second embolization showing pseudoaneurysm formation at the superior sagittal sinus (arrow).*

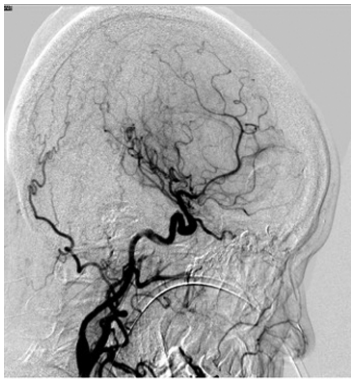

***Figure 5**. Cerebral angiogram post trans-arterial embolization showing complete occlusion of the DAVF*

.

REFERENCES

1. Gupta A K, Periakaruppan A L. Intracranial dural arteriovenous fistulas: A Review. Indian J Radiol Imaging 2009; 19: 43-8.
2. Kiyosue H, Hori Y, Okahara M, Tanoue S, Sagara Y, Matsumoto S, et al. Treatment of intracranial dural arteriovenous fistulas: current strategies based on location and hemodynamics, and alternative techniques of transcatheter embolization. Radiographics 2004; 24(6): 1637-53.
3. Im SH, Oh CW, Han DH. Surgical management of an unruptured dural arteriovenous fistula of the anterior cranial fossa: natural history for 7 years. Surg Neurol 2004; 62(1): 72-5.
4. Lewis AI, Tomsick TA, Tew JM Jr. Management of tentorial dural arteriovenous malformations: transarterial embolization combined with stereotactic radiation or surgery. J Neurosurg 1994; 81(6): 851-9.
5. Rai SPV, Bele K, Reddy H. Endovascular Approach for Treatment of Frontal Dural Arteriovenous Fistula through the Ophthalmic Arteries using Glue: A Case Presentation with Review of Literature. Indian J Vasc Endovasc Surg 2015; 2: 125-9.
6. Kim DJ, Kim DI, Suh SH, Kim J, Lee S, Kim EY, et al. Results of transvenous embolization of cavernous dural arteriovenous fistula: a single-center experience with emphasis on complications and management. AJNR Am J Neuroradiol 2006; 27(10): 2078-82.
7. Choi HJ, Cho CW. Anterior cranial fossa dural arteriovenous fistulae presenting as subdural hematoma. J Korean Neurosurg Soc 2010 Feb; 47(2): 155–157.