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Giant Vein of Galen Malformation as a Cause of TIA- A Case Report

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Abstract

Introduction: Vein of Galen malformations (VOGMs) are rare vascular entities originating from persistent shunting of primitive choroidal vessels into the median prosencephalic vein of Markowski. They usually present in infancy or childhood as seizures, hydrocephalus or cardiac failure; adult presentations are uncommon. In addition, giant VOGM in an adult is extremely rare to find.
Case description: We present an exceedingly rare case of a giant VOGM measuring 3.4 x 3.7 x 3.2 cm in a 20-year-old female presenting as episodes of transient ischemic attack (TIA) which was obliterated successfully using endovascular embolization.
Conclusion: VOGMs are associated with poor clinical outcome if left untreated. With advancement in endovascular techniques of obliteration, proper timely management can lead to good outcomes.

Key-words: Vein of Galen malformation; Vascular malformation; Digital subtraction angiography; Neurosurgery; Embolization; Endovascular

Key Messages: We report a rare case of giant vein of Galen malformation in an adult presenting as TIA. The malformation was successfully obliterated via endovascular embolization. This emphasizes the fact that with proper patient selection and timely intervention, good outcomes are possible in such lesions.

Introduction

Vein of Galen malformations (VOGM) are rare vascular malformations which originate from persistent shunting of primitive choroidal vessels into the median prosencephalic vein of Markowski. Usually the presenting symptoms are high-output cardiac failure and hydrocephalus during infancy, with other delayed sequelae like developmental delay and seizures.[1] f left untreated it has been associated with poor clinical outcome with a high mortality rate of up to 76.7%.2We report a rare case of a giant, untreated VOGM in a 20-year-old woman presenting as transient ischemic attack (TIA). Case

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History:
A 20-year-old apparently healthy female presented to the OPD with history of two episodes of transient loss of consciousness 3 months apart. On both the occasions patient recovered completely within 2-3 minutes with mild to moderate headache which persisted for a day and resolved with analgesics. There was no focal deficit as reported by the patient. There was no other significant history.
On examination, patient was conscious, oriented and comfortable. There was no cranial nerve involvement and motor and sensory examination was essentially normal.
Considering a diagnosis of a TIA, an MRI was advised which showed multiple dilated tortuous vessels extending from the left medial temporal lobe up to the quadrigeminal cistern suggestive of a vascular malformation measuring 3.4 x 3.7 x 3.2 cm (Figure 1)

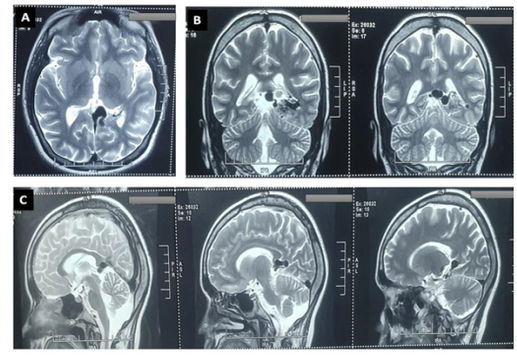

Figure 1: Pre operative MRI images

After all routine blood investigations and a pre anaesthetic checkup, patient was posted for an endovascular embolization. Preoperative digital subtraction angiography (DSA) showed a giant vein of Galen malformation with feeders from the left posterior cerebral artery (PCA) P2 segment and the left middle cerebral artery (MCA) (Figure 2).

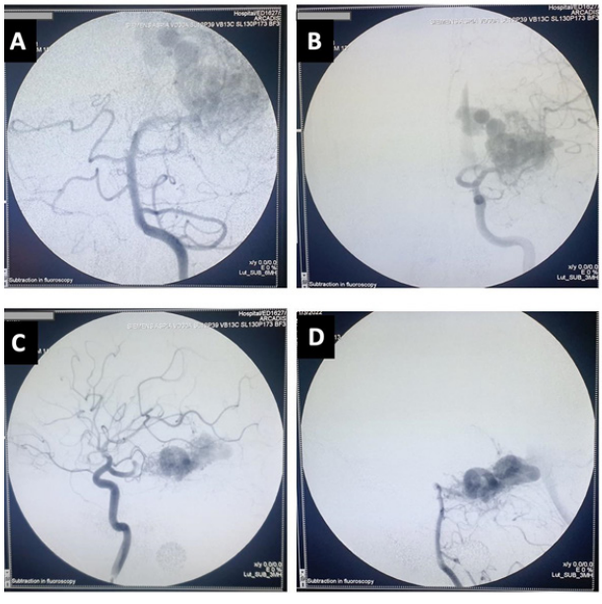

Figure 2: Pre embolization DSA images; (A) Feeders from left PCA; (B) Feeders from the left MCA; (C) Lateral view showing feeders from left MCA; (D) Lateral view showing the giant VOGM with feeders from left PCA

Embolization was carried out with the patient under general anaesthesia. The right femoral artery was cannulated. 6 Fr envoy multipurpose catheter (MPC) was used as a guiding catheter. Embolization was done using 0.10 microcatheter (Prowler 10) and Lipiodal and n cyanobutylacrylate (NCBA) as the embolizing agents (Figure 3).

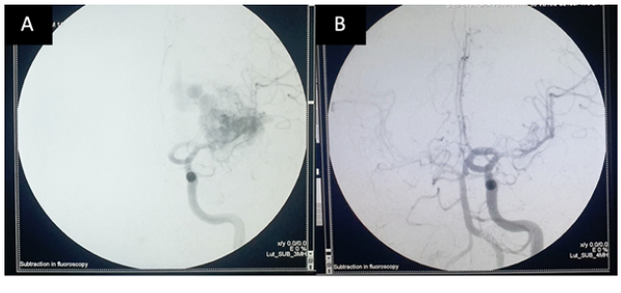

Figure 3: (A) Pre embolization DSA showing steal phenomenon resulting in no flow in the ACA and reduced flow in MCA. (B) Post embolization DSA showing reestablishment of flow in the ACA, MCA with good cross flow to contralateral side

Post procedure patient was extubated and shifted to the neurosurgery ICU where she was observed for one day. Patient developed mild weakness of the right upper and lower limb which resolved with intravenous infusion of nimodipine. Rest of the post operative course was uneventful and patient was discharged on the fifth day.At two months follow up, patient is doing well and has joined back her studies.

Discussion

Ours was a case of giant VOGM in an adult patient which has rarely been reported in literature. Initially VOGMs were classified into four types by Yasargil. Later Lasjuanias divided them into two main types- choroidal and mural depending upon the location and nature of the shunt.[3]The choroidal type, more common in newborns, consists of multiple high-flow fistulas draining into the tela choroidea, ultimately into the anterior portion of the median vein of prosencephalic vein of Markowski. On the other hand, the mural type has a single arteriovenous fistula that feeds directly into the inferolateral wall of the median vein of Markowski and present in later childhood.

Neonates usually present with severe congestive heart failure, infants with macrocephaly or hydrocephalus, and older children or adults typically manifest seizures, headaches, or cranial neuropathies secondary to mass effect. However, the only clinical finding in our patient was two episodes of TIA. As per the American Heart Association and American Stroke Association TIA is defined as transient episode of neurological dysfunction caused by focal brain, spinal cord, or retinal ischemia, without acute infarction. The common causes are occlusion of small perforating vessels, embolism from a cerebral artery or from an extracranial source.[4] In our case, there was a significant steal of flow from the ACAs and the MCA which may have led to the episodes of TIA. The flow was reestablished after endovascular embolization of the sac. A similar case of TIA presenting as left sided weakness revealed a large left-sided parietal-occipital arteriovenous malformation (AVM) with the cause attributed to AVM related steal phenomenon. [5,6] This emphasizes the fact that cerebrovascular malformations can lead to steal phenomenon and present as stroke like features or TIA.

Indications for treatment depends upon the severity of the patient's symptoms or the presence of angiographic features that indicates an increased risk of future rupture.[7] Currently, the natural history of VOGMs in adults is unknown. In adults VOGM detected incidentally without evidence of hemorrhage or thrombosis are likely to remain stable, whereas patients presenting with hemorrhage, thrombosis, or mass effect or raised intracranial pressure have a malignant course and warrant immediate treatment.[7] Kerolus et al also reported a case of giant VOGM in an adult.[2] However in their case no intervention was done and the patient was kept on observation due to the patient's poor clinical status at baseline. Whereas, in our case the VOGM was successfully obliterated via endovascular approach with good outcome. Hence our case highlights that endovascular intervention if done at the earliest, has good chances of successful outcome even if it is a giant VOGM.

The management of VOGMs continue to be challenging. The treatment modalities include microsurgery, stereotactic radiosurgery, and endovascular surgery. However, the favored treatment strategy for VOGMs has traditionally been endovascular embolization.[3] As newer improved management strategies have evolved, the overall survival and technical success of endovascular treatment for VOGMs are improving. Although the goal is to completely obliterate the malformation, partial occlusion may halt the progression of cardiac sequelae and promote self-thrombosis preventing the need for further embolization.

There is paucity of literature reporting giant VOGMs in adults. Given the progress in the field of endovascular techniques in the recent years, endovascular treatment has become the convenient first choice and offers good clinical outcome in properly selected patients

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