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Case Report

Obstructive Hydrocephalus Produced by Venous Ectasia of Dural AV Fistula

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Abstract

Dural arteriovenous fistula (Dural AVF) is an anomalous shunt between dural arterial and venous channels. About 10–15% of all intracranial vascular malformation are dural fistulas which represent anomalous shunts among arterial branches and dural venous sinuses, meningeal, or cortical veins. We are presenting a rare case of dural AVF treated by transarterial embolization and complicated by hydrocephalus.

Key words: Dural arteriovenous fistula, Hydrocephalus, Transarterial embolization

INTRODUCTION

A dural arteriovenous fistula is an abnormal direct connection between a meningeal artery, meningeal vein, or dural venous sinus. Dural AVF is approximately 10–15% of all cerebral vascular malformation which represent anomalous shunts among arterial branches and dural venous sinuses, meningeal or cortical veins.[1-4]

The common predisposing factor for dural AVF appears to be venous sinus thrombosis which causes the development of venous hypertension, lead to opening up of the microvascular connection within the dura. These channels become hypertrophied and result in direct shunting between arteries and veins.

CASE REPORT

A 52-year-old male admitted with chief complaints of headache, weakness of the left half of body, and slurring of speech. Computed tomography (CT) brain was suggestive of small to moderate size, enhancing vascular structure seen at ambient cistern on the right side [Figure 1].

Magnetic resonance imaging brain revealed a large lobulated lesion in the right side of midbrain extending into ambient cistern showing vascular flow voids [Figure 2].

On the day of the intervention, the patient had a Glasgow Coma Score of 15. Cerebral digital subtraction angiography (DSA) was done through the right femoral artery which showed dural AVF at the junction of the right transverse and sigmoid sinus. Arterial supply of dural AVF is from the right middle meningeal, right ascending pharyngeal artery, and right tentorial artery [Figure 3]. There was marked venous ectasia with cortical venous reflex (Cognard Type IIb). After 3 days, transarterial embolization was done with glue (NBCA) mixed with lipiodol. At the end of the intervention, the patient was awake, and no new neurological deficit was noted. CT angiography brain was performed, which showed a complete resolution of fistula. The patient was discharged with no neurological deficit.

After 1 month of embolization, the patient was again admitted with complaints of irrelevant talking, ataxia, and slurring of speech. The plain CT brain showed dilatation of bilateral lateral ventricles and third ventricles [Figure 4]. The right ventriculoperitoneal shunting was done and the patient was shifted to the intensive care unit. The patient improved neurologically and he was discharged [Figure 5].

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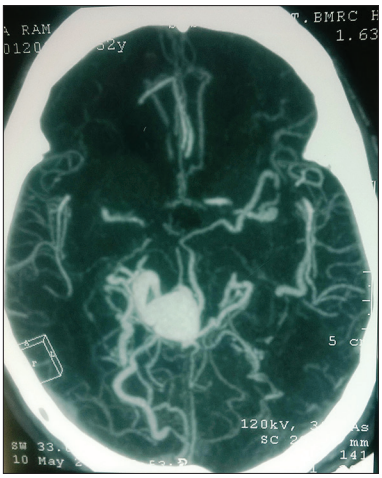

Figure 1: Computed tomography pre-surgrey showed enhancing vascular structure seen at ambient cistern on the right side

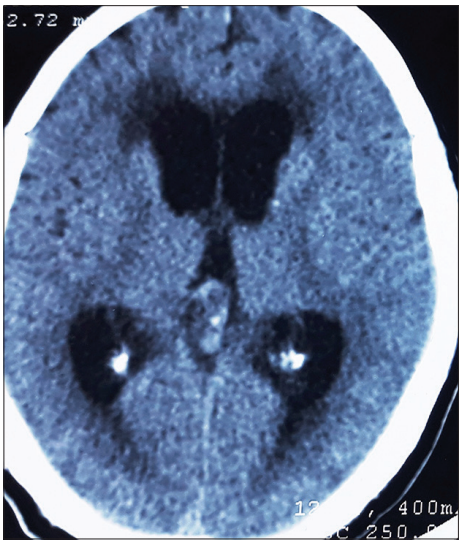

Figure 4: Post-embolization (1 month later) and non-contrast computed tomography brain show dilated ventricles

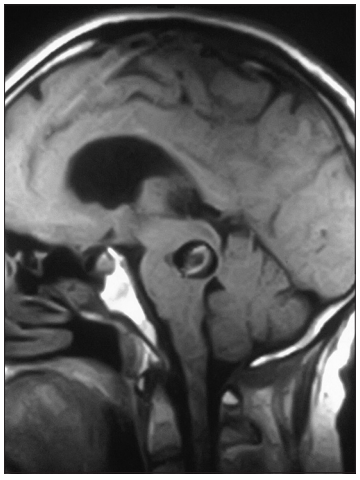

Figure 2: T1 sagittal show predominant hypointense lesion with laminated hyperintensities within due to flow signal

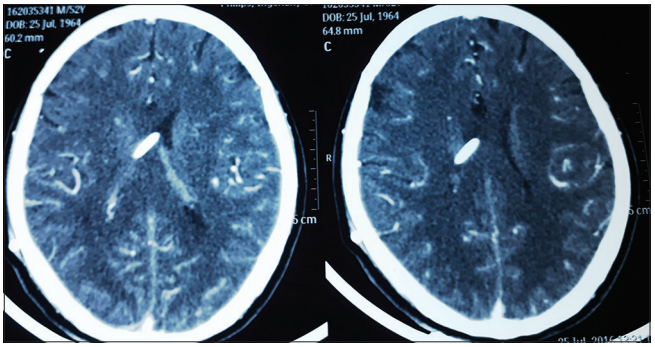

Figure 5: Contrast-enhanced computed tomography after shunt surgery, ventricles has returned to normal size

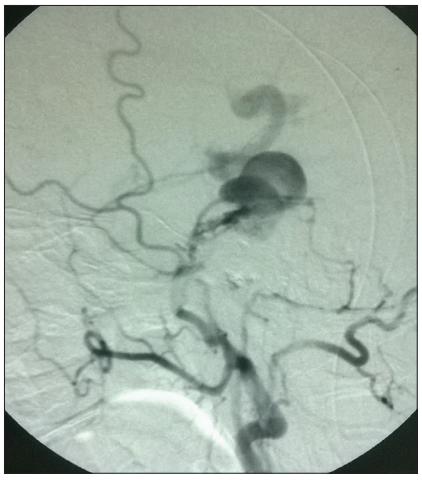

Figure 3: During pre-embolization digital subtraction angiography, RECA injection shows dural arteriovenous fistula filling through middle meningeal artery and ascending pharyngeal artery with marked venous ectasia

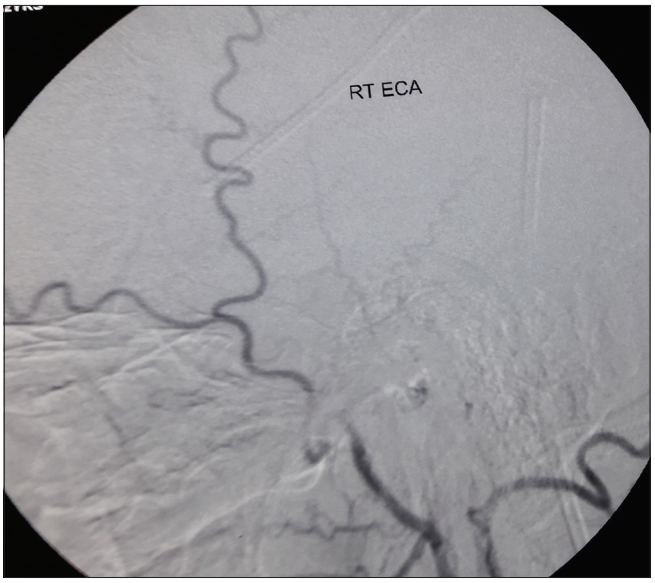

Figure 6: During post-embolization digital substraction angiography, the right external carotid artery injection shows no filling of dural arteriovenous fistula

After 6 months of embolization, the patient admitted for follow-up DSA. DSA showed no filling of AVF [Figure 6].

DISCUSSION

Hydrocephalus after the treatment of dural AVF is rare. No case of hydrocephalus after treatment of dural AVF by glue has been reported. Several reports have described the detection of hydrocephalus after coiling with hydrogel coil.[5-8]

Ozaki *et al.* reported two cases of delayed hydrocephalus after embolization of unruptured aneurysm using bare platinum coils. Chaves *et al.* reported a case of intraventricular hemorrhage after dural fistula embolization. Maimon *et al.* described morbidity of 6% (1/17 patients) related to a transient trochlear nerve palsy and in a retrospective study by Rangel-Castilla *et al.*, a complication rate of 9.7% (7 of 72 patients) was described, of which the only one corresponded to intraparenchymal hemorrhage.[9,10]

Dural embolization with ONYX (a non-adhesive embolic agent composed by ethylene vinyl alcohol dissolved in dimethyl sulfoxide) has grown to become one of the major approaches to dural AVF, which is linked to its remarkable cure rates and low morbidity. In fact, there are few complications noted after this technique.

After a systematic review of the international literature using the PubMed database, we were not able to find any article describing the occurrence of obstructive hydrocephalus after dural AVF embolization, which corroborate the relevance of this case report.

The phenomena behind the development of hydrocephalus remain unclear. Different hypotheses have been proposed to relate the phenomena and materials used for embolization. With bare platinum coils, mechanical obstruction of the CSF pathway due to the expansion of the embolized coil mass can be the cause of post-embolization hydrocephalus. Another theory of the obstructive hydrocephalus was due to IVH which was related to iatrogenic injury to a microperforator while retrieving the microcatheter used for arterial cannulation. Obstructive hydrocephalus, in this case, seems to be due to compression of aqueduct by venous ectasia of dural AVF.

In summary, we report a case of unusually delayed hydrocephalus after treatment of dural AVF with glue which has not been reported until now.

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