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Case Reports & Case Series

A case of cranial vault intraosseous arteriovenous fistula treated with transarterial embolization

Tomohiro Iida[a,b,*], Yukiko Enomoto[a], Yusuke Egashira[a], Keita Yamauchi[b], Toru Iwama[a]

[a] Department of Neurological Surgery, Gifu Medical University, 1-1 Yanagido, Gifu City, Gifu 5011194, Japan
[b] Department of Neurological Surgery, National Hospital Organization Toyohashi Medical Center, 50 Hamamichiue, Imure, Toyohashi City, Aichi 4408510, Japan

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1. Background

Intracranial dural arteriovenous fistula (dAVF) is caused by abnormal anastomosis on the dura, and accounted for 10 to 15% of intracranial arteriovenous malformation. Usually, the arteriovenous shunt is fed by meningeal arteries and drained to meningeal veins, venous sinuses and cortical veins. An arteriovenous fistula with a shunt point located in the bone (what called “intraosseous AVF”) is rare, especially at supratentorial location. We report a case of good clinical course after transarterial embolization (TAE) for intraosseous AVF at supratentrial location with literature consideration.

2. Case presentation

An 88-year-old woman with a history of deep vein thrombosis (DVT) was transferred to previous hospital because she could not get up after falling. She has noticed tendency to falling several weeks ago. Because the edema in right frontal lobe was detected in the head plain computed tomography (CT) taken at previous hospital, she was referred to our hospital. On admission, her consciousness level was alert, she had mild paresis of her left lower limb. No abnormal coagulability was observed in blood sampling.

3. Investigation

Head magnetic resonance imaging (MRI) revealed extensive edema with flow voids which thought to be dilated medullary veins in deep white matter (Fig. 1). Digital subtraction angiography (DSA) shows an arteriovenous shunt in left cranial vault fed from middle meningeal artery (MMA) and anterior deep temporal artery (ADTA) and drained into the right frontal cortical vein via the diploic vein (Fig. 2). This cortical venous reflex was attributed to the occlusion of the superior sagittal sinus (SSS) (Fig. 2). We diagnosed as TypeIII dAVF as Borden classification and TypeIII as Cognard classification.

4. Treatment

Under local anesthesia, an 8 Fr balloon guide catheter (Flowgate 2, Stryker) was placed at right common carotid artery. Flow directed microcatheter (Marathon, Medtronic) was induced to the anterior branch of MMA. The anterior branch of MMA was embolized with 33% n-butyl-2-cyanoacrylate (NBCA, Cordis) (Fig. 3A). The external carotid angiogram revealed the residual shunt from ADTA (Fig. 3B), then ADTA was embolized with 17% n-butyl-2-cianoacrylate (NBCA) (Fig. 3C). Although the faint shunt from the small branches of MMA remained (Fig. 3D), the sufficient embolization was obtained.

Abbreviations: ADTA, anterior deep temporal artery; ATDV, anterior temporal diploic vein; CT, computed tomography; dAVF, dural arteriovenous fistula; DSA, digital subtraction angiography; DVT, deep vein thrombosis; FDV, frontal diploic vein; TAE, transarterial embolization; MMA, middle meningeal artery; MRI, magnetic resonance imaging; NBCA, n-butyl-2-cianoacrylate; PTDV, posterior temporal diploic vein; SSS, superior sagittal sinus

* Corresponding author.
E-mail address: tomohiro.iida0107@gmail.com (T. Iida).

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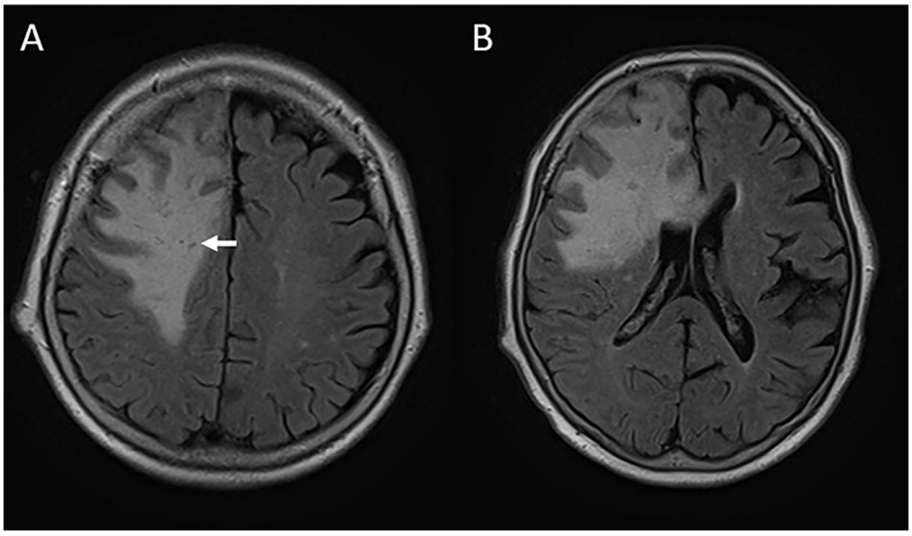

Fig. 1. Non-contrast MRI on admission. Axial FLAIR image shows brain edema with expanded medullary vein (white arrow) in right frontal lobe.

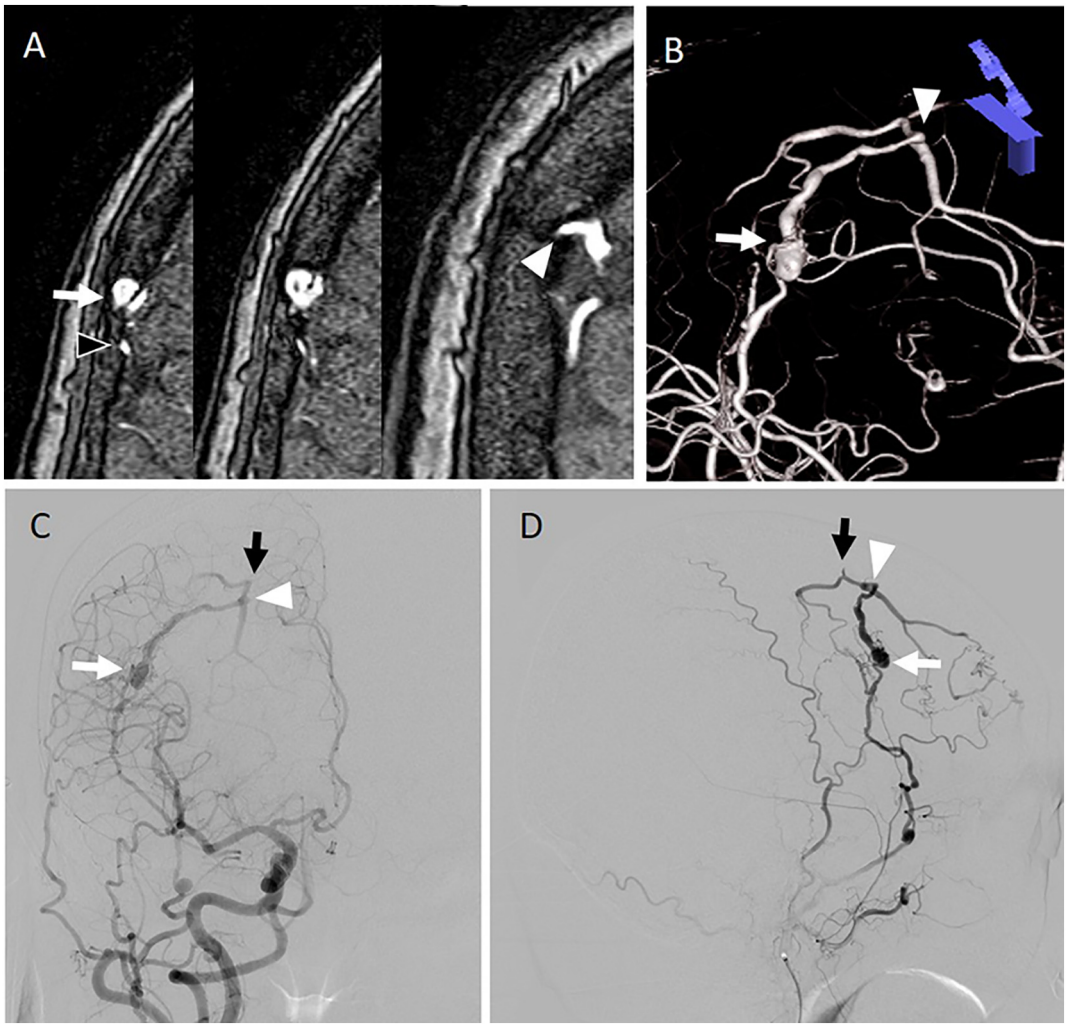

Fig. 2. MR angiography demonstrating a shunt point and the diploic vein (A). Three-dimensional view (B), anteroposterior view of digital subtraction angiogram (DSA) from right common carotid artery (C) and lateral view of right external carotid artery (D) demonstrating an arteriovenous shunt (white arrow) from MMA (black arrow head) and ADTA which is drained into the right frontal cortical vein via the diploic vein (white arrow to white arrow head). The outflow to the superior sagittal sinus (SSS) was occluded (black arrow).

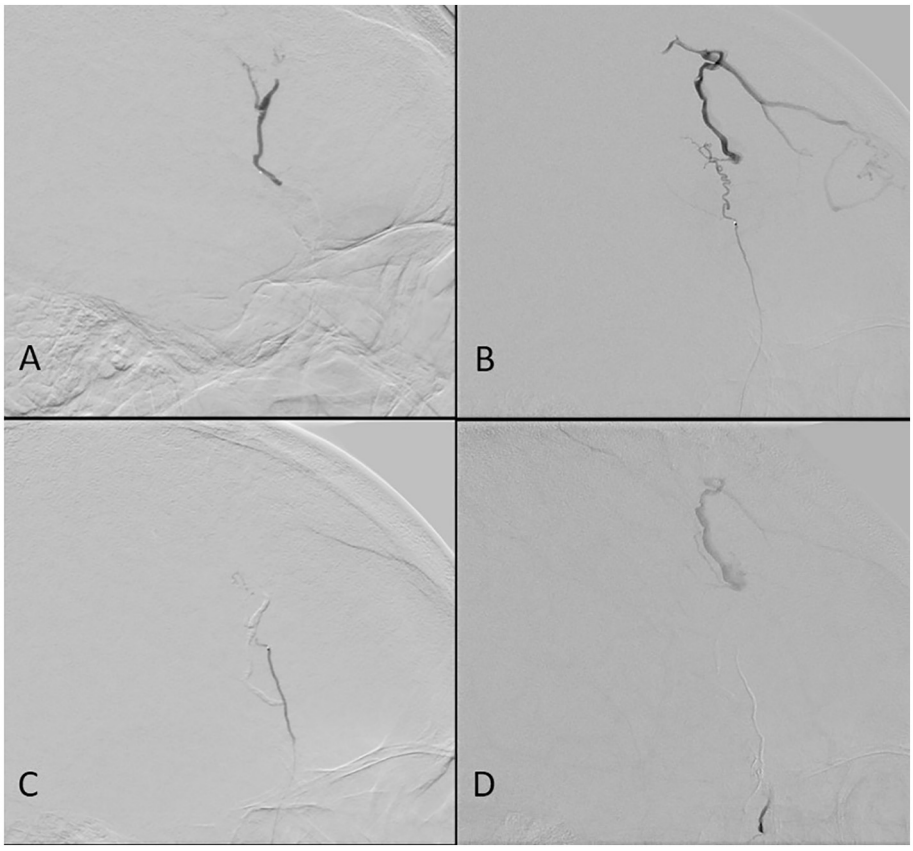

Fig. 3. Digital subtraction angiogram (DSA) during trans-arterial embolization. 33% NBCA was injected via microcatheter placed at right middle meningeal artery (A). DSA after embolization from MMA demonstrating shunt flow from anterior deep temporal artery (ADTA) (B). ADTA was embolized using 17% NBCA (C). DSA after embolization demonstrating slight shunt flow from small branches from MMA (D).

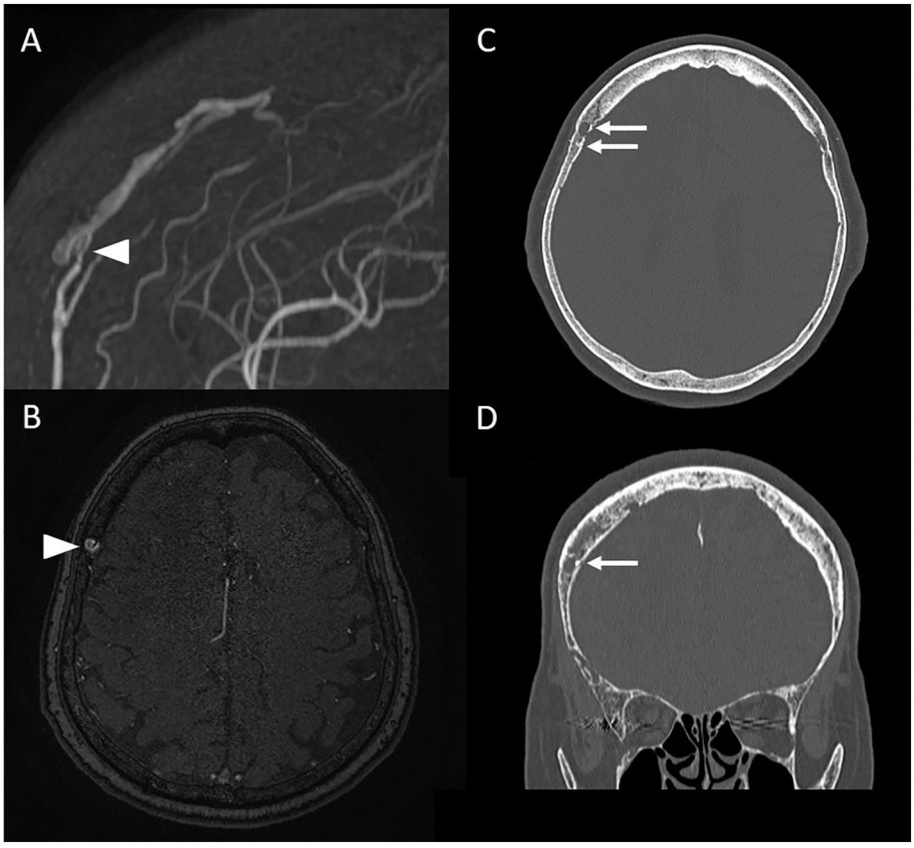

Fig. 4. Comparing with MRA before embolization (A and B), axial view (C) and coronal view (D) of the non-contrast CT after embolization demonstrating the distribution of NBCA in the diploic space (white arrow).

Fig. 5. Non-contrast MRI demonstrating the reduction of the brain edema in right frontal lobe.

5. Outcome and follow up

We confirmed no hemorrhagic complication with cone-beam CT just after embolization. Postoperative CT showed the NBCA-cast distributed in diploic space of skull (Fig. 4). MRI shows remarkable improvement of the brain edema in right frontal lobe (Fig. 5). After embolization, left lower limb paralysis gradually improved in a week. At 6 month after embolization, her modified Rankin Scale improved 2 points from 4.

6. Discussion

Intraosseous AVF is relatively rare, and furthermore there are far fewer reports about the supratentorial location like our case because almost reported cases are infratentorial location, which have relationship between many emissary veins [1–3].

Diploic veins are classified into frontal diploic vein (FDV), anterior temporal diploic vein (ATDV), posterior temporal diploic vein (PTDV) and occipital diploic vein. The FDV and ATDV form an anterior diploic venous subsystem converging in the pterional area, which connects the SSS with the sphenoparietal sinus. The posterior subsystem converging in the asterion is formed by the PTDV and occipital diploic veins, and connects the posterior SSS with the transverse and sigmoid sinuses [4].

It is believed that diploic vein has potentially channels between dural venous sinuses and pachymeningeal veins and also in pericranial veins through emissary veins. These channels open under pathological condition such as the sinus thrombosis, venous thrombosis, trauma, meningioma, etc. [4]. In general, diploic vein has no direct anastomosis with cortical veins, so it thought to be rare and interesting that shunt flow via diploic vein leads the focal sign due to venous congestion.

A case of supratentorial intraosseous AVF with focal sign related to venous congestion is, in addition to this case, only one case reported by Yako et al. [1]. The mechanism of the case was proposed as follow. The enlarged DV might have connections not with the SSS itself but with the venous lacuna via emissary veins. The falcine vein might have communicated with both the venous lacuna and the SSS originally.

Similar to general dAVF, due to venous hypertension caused by AV shunt, the occlusive change of drainage site might occur at the channel between the venous lacuna and the SSS and consequently the shunt flow might be directed from the venous lacuna to the cortical veins via the falcine vein. Eventually, these venous drainage changes involving the occlusive process might result in intracerebral hemorrhage.

In the present case we speculate the mechanisms of our case as follows. At first, the dilated diploic vein connect with bridging vein via an emissary vein. Second, this connection leads the influx of shunt flow into cortical veins and SSS. Finally, the occlusive change at outflow to SSS leads the cortical venous reflex into the medullary vein and resulted in extensive edema in right frontal lobe.

7. Conclusions

We experienced a rare case of dAVF involving diploic vein. TAE using NBCA successfully improved the brain edema and neurological symptoms. Like the aggressive type of dAVF, this type has a high risk of intracranial hemorrhage and neurological deficit depending on the pattern of outflow veins. We think this disease is important as a differentiation of vascular disease exhibiting nonspecific onset patterns and image findings.

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