A Case of Iatrogenic Middle Meningeal Arteriovenous Fistula That Occurred during Embolization of a Feeding Artery of Dural Arteriovenous Fistula

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Objective: A case of iatrogenic dural arteriovenous fistula that occurred during embolization of a feeding artery for transverse sinus (TS) dural arteriovenous fistula is reported.

Case Presentation: A 66-year-old woman suddenly noted pain of the left temporal region and nausea and was emergently transported to our hospital. CT of the head showed left subcortical hemorrhage, and DSA demonstrated a dural arteriovenous fistula at the left TS. Although embolization of the feeding artery was attempted via the middle meningeal artery (MMA), a middle meningeal arteriovenous fistula (MMAVF) developed as the MMA was damaged by intraoperative balloon inflation. The MMA was immediately embolized together with the fistulous opening using n-butyl-2-cyanoacrylate (NBCA) and coils, and no sequelae were observed postoperatively.

Conclusion: We experienced dural arteriovenous fistula caused by injury of the feeding artery during endovascular treatment. It must be remembered that iatrogenic arteriovenous fistula is a complication of endovascular treatment that is rare but requires attention.

Keywords: ▶ dural arteriovenous fistula, middle meningeal artery, iatrogenic arteriovenous fistula

Introduction

Recently, cases of intracranial endovascular treatment are increasing, and the middle meningeal artery (MMA) is its frequent site. Injury of the MMA due to head trauma is known to cause acute epidural hematoma, but reports of complications of endovascular treatment due to MMA injury have been rare.1) In this report, we present a case that developed iatrogenic dural arteriovenous fistula (i-DAVF) due to injury of the MMA during embolization of the feeding artery of transverse sinus (TS) DAVF.

Case Presentation

The patient was a 66-year-old woman. In early August 2016, she noted pain in the left temporal region and nausea and emergently visited our hospital.

She had chronic headache and hypertension, but there was no history of head trauma. She had been treated with pitavastatin at 2 mg/day and famotidine at 20 mg/day. She had no particular familial history or history of smoking or drinking.

The blood pressure was 150/90 mmHg, and the heart rate was regular at 90/min. Neurologically, she showed a Japan Coma Scale (JCS) of I-1, Glasgow Coma Scale (GCS) grades of E4V5M6, and was restless. No particular abnormality was noted in the cranial nerves. While no paralysis, sensory disturbance, or aphasia was observed, attentional deficit to the right side was noted.

Concerning imaging findings, plain CT of the head showed a subcortical hematoma with a volume of about 25 mL in the left temporo-occipital region (Fig. 1A).
Although no abnormal blood vessels were visualized on MRA of the head (Fig. 1B), DAVF was noted near the TS on DSA. There was no direct communication with the sinus, and the cortical veins were the only drainage routes. The feeding arteries were the posterior convexity branch (PCB) of the MMA and meningeal branch of the occipital artery of the affected side, and the lesion was diagnosed as Borden type III, Lalwani type IV having a shunting point in the superior wall of the TS (Fig. 2A–2D).

Endovascular treatment
Since the lesion had no communication with the TS, transvenous embolization was considered difficult, and transarterial embolization (TAE) using low-concentration n-butyl-2-cyanoacrylate (NBCA) was selected. Under general anesthesia, a 7 Fr Roadmaster TH (Goodman, Aichi, Japan) was placed at the origin of the left external carotid artery (ECA) via the right femoral artery.

The meningeal branch of the occipital artery and MMA PCB were considered to be target vessels. Since the selection of the occipital artery was difficult at diagnostic imaging, it was expected to be considerably difficult to approach the region distal to the meningeal branch. However, as the MMA PCB arose at a sharp angle, and its origin was markedly tortuous, insertion of a microcatheter was considered difficult. In addition, there was some distance from this site to the shunting point, and penetration with NBCA was expected to be difficult. Therefore, if the microcatheter could not be navigated to the MMA PCB, we planned to deliver the embolic material more distally by temporarily occluding the proximal side in a wedged manner using a Pinnacle blue 27 (Tokai Medical Products, Aichi, Japan) as an intermediate catheter. A coaxial system consisting of a Pinnacle blue 27 and a Carnelian Marvel Non Taper (Marvel; Tokai Medical Products) was guided to the left MMA with ASAHI CHIKAI-14 200 cm (Asahi Intecc, Tokyo, Japan). The Pinnacle blue 27 was navigated to the MMA horizontal segment beyond the foramen spinosum. The Marvel could be guided only to the bifurcation of the MMA PCB due to the tortuousness at its origin. When microcatheter fluoroscopy was performed by injecting the contrast medium from this site, the shunt was clearly visualized, and no anastomosis with the ophthalmic artery or internal carotid artery was noted (Fig. 3A). Since the presence of latent dangerous anastomosis was considered possible, this site was wedged by inflating the balloon to prevent proximal reflux from the bifurcation of the MMA PCB. Also, it was decided to immediately discontinue the injection in the event of reflux of NBCA from the bifurcation of the MMA PCB to the anterior branch. After these preparations, warmed 17% NBCA was intra-arterially injected from this site.

A twofold dilution of the contrast agent was placed in a 1-mL syringe, and the balloon of the Pinnacle blue 27 was slightly dilated under fluoroscopy (Fig. 3B–3D). When NBCA was injected intra-arterially from the Marvel during DSA, it flowed into the PCB but began to flow backwards before it reached the shunting point. Although we repeated the injection with pauses of about 5 seconds, NBCA could not be delivered further, and the procedure was ended after feeder occlusion (Fig. 3E). The balloon of the Pinnacle blue 27 was deflated, and the Carnelian Marvel Non-Taper was removed.
However, when angiography was performed through the guiding catheter placed in the ECA, the condition was markedly changed. While the MMA PCB was occluded, structures including the left inferior petrosal sinus (IPS) and pterygoid venous plexus were delineated in the arterial phase. Since reflux into two veins running in parallel near the MMA was observed, a diagnosis of middle meningeal arteriovenous fistula (MMAVF) with the fistulous opening near the MMA horizontal segment was made (Fig. 4A and 4B). As DAVF was not observed on DSA before the intra-arterial injection of NBCA, and as the fistulous opening was near the balloon, the MMA was considered to have ruptured due to overinflation of the balloon. Cone beam CT scans were immediately performed, and the absence of lesions such as epidural or subdural hematoma was confirmed. The Pinnacle blue 27 was removed, and, instead, a Renegade-18 (Stryker, Kalamazoo, MI, USA) was guided to the MMA horizontal segment. From the same site, 12 pushable coils (Hilal, Cook, Bloomington, IN, USA) were inserted, and 17% NBCA was injected intra-arterially from before the coils (Fig. 4C). This resulted in complete occlusion of the iatrogenic fistula (Fig. 4D and 4E).

After this, to embolize the DAVF near the TS, which was the original target, a microcatheter was guided to the meningeal branch of the occipital artery, and 17% NBCA was intra-arterially injected. Despite proximal occlusion, the shunt flow was markedly reduced. The patient has remained free of recurrence for 6 months thereafter.

**Discussion**

MMAVF is formed as communication between the MMA and the adjacent middle meningeal vein (MMV) develops as a result of injury of the MMA running in the dura mater inside
the cranium. MMAVF has often been reported with head trauma and to complicate 1.8% of head injuries, but are rarer than acute epidural hematoma, which is also caused by injury of the MMA. Cases of iatrogenic MMAVF occurring after craniotomy and a small number of cases of naturally occurring MMAVF with unknown causes have also been reported. MMAVF reported here that occurred as a complication of endovascular treatment was a relatively rare case.

While traumatic injury of the MMA often leads to the formation of epidural hematoma, it interestingly caused arteriovenous fistula (AVF) in our patient. The MMA and MMV run in the space between the bone surface layer of the dura and inner table of the skull, and the occurrence of many traumatic epidural hematomas is reported to be associated with skull fracture. In trauma, the dura mater is partly detached from the inner table of the skull due to fracture or large external force, and hematoma develops primarily in the space created by this detachment. However, when the MMA is damaged from the luminal side as in our patient, AVF rather than hematoma is considered to develop as the contact between the dura mater and skull is maintained, and as the dura mater is likely to be intact.

Since MMAVF occurred suddenly at DSA after the first injection of NBCA, it is considered to have been caused in our patient by damage of the MMA due to overinflation of the balloon in the intracranial MMA. While we inflated the balloon carefully under fluoroscopic monitoring, the MMA is considered to have been damaged due to the extremely high compliance of the balloon and significant change in its diameter with injection of even a small volume of contrast medium, marked decrease in visibility due to overlapping with bones of the skull base, and the very small diameter of the target MMA with consequent difficulty in checking of its dilatation. We regret that we did not take preventive measures such as checking the balloon diameter by inflating it outside the body in advance and reducing the
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Due to cerebral or subarachnoid hemorrhage and reported it as a condition with a risk of intracranial hemorrhage. It has also been reported to be involved in the recurrence of chronic subdural hematoma. However, cases of spontaneous regression have also been reported, and the risk of MMA VF has not been established. In our patient, MMA VF occurred during general anesthesia, and whether it was symptomatic or not was unclear. We judged that it was an indication for treatment, considering that it was angiographically a high-flow lesion and was not expected to cure spontaneously that there was a risk of hemorrhagic complications, and that the condition was caused iatrogenically during surgery.

We could manage our patient fortunately without sequelae by performing embolization immediately after the occurrence of MMA VF. MMAVF can be treated directly or endovascularly, but the endovascular approach is employed due to cerebral or subarachnoid hemorrhage and reported it as a condition with a risk of intracranial hemorrhage. It has also been reported to be involved in the recurrence of chronic subdural hematoma. However, cases of spontaneous regression have also been reported, and the risk of MMAVF has not been established. In our patient, MMAVF occurred during general anesthesia, and whether it was symptomatic or not was unclear. We judged that it was an indication for treatment, considering that it was angiographically a high-flow lesion and was not expected to cure spontaneously that there was a risk of hemorrhagic complications, and that the condition was caused iatrogenically during surgery.

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increasingly due to its mild invasiveness. For embolization, the use of embolic agents such as Onyx and Embosphere (Merit Medical, South Jordan, UT, USA) has been reported in addition to NBCA, which was used in our patient. According to the above report, Almefty et al. achieved cure in eight of nine patients with MMAVF by embolization using Onyx.

To our knowledge, only one case of MMAVF caused by damaging the MMA during endovascular treatment has been reported, by Terada et al. In this case, MMAVF was caused by perforation of the MMA by a microguidewire during embolization of the feeding vessel of a meningioma. In their report, Terada et al. ascribed perforation of the MMA to its marked tortuosity. According to our review of the literature, there was no previous report of MMAVF caused by balloon inflation as in our patient. Shi et al. reported the effectiveness of proximal occlusion with a balloon (Hyper-Form; Medtronic, Minneapolis, MN, USA) during TAE for DAVF and suggested a decrease in the shunt flow, improvement in the control of reflux of the embolic agent, and prevention of influx of the embolic agent into so-called dangerous anastomosis as its advantages. Since there have been similar reports using Scepter C (Microvention, Tustin, CA, USA), balloon assistance is an option in treating DAVF. The MMA is a frequent target of embolization, but it has a small diameter among intracranial vessels and is known to have histologic characteristics including defect of media. Therefore, the risk of the occurrence of MMAVF due to rupture or perforation of the MMA must be remembered when a balloon is inflated in the MMA.

### Conclusion

Balloon inflation in the MMA must be performed with utmost caution due to the risk of vascular injury. It may result in MMAVF.

### Disclosure Statement

There are no conflicts of interest to disclose regarding this paper.

### References