

Case Report

Dural Arteriovenous Fistula of Jugular Foramen with Subarachnoid Hemorrhage : Selective Transarterial Embolization

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We report the case of a 64-year-old man with dural arteriovenous fistula (DAVF) at right jugular foramen, presented as subarachnoid and intraventricular hemorrhage. The malformation was fed by only the neuromeningeal trunk of the right ascending pharyngeal artery and drained into the right lateral medullary veins craniopetally. Complete embolization was attained by selective transarterial glue injection, but patient showed lower cranial neuropathies. A 3-month follow-up angiogram still showed persistent fistula occlusion. Transarterial glue embolization is a feasible method, only if a transvenous access is not possible in case of single channel fistula.

KEY WORDS : Dural arteriovenous fistula · Glue · Intra arterial injection · Subarachnoid hemorrhage.

INTRODUCTION

The most common cause of nontraumatic subarachnoid hemorrhage (SAH) is a rupture of the intracranial aneurysm. Rupture of the arteriovenous malformations or tumors, and blood dyscrasias are also remote culprits. We report the case of a dural arteriovenous fistula (DAVF) of the jugular foramen presented as SAH, and was treated by intraarterial embolization, in which the fistulous location was assessed by computed tomography (CT) obtained after embolization.

CASE REPORT

A 63-year-old man presented with sudden headache, dyspnea, and hyperventilation. He had no prior history of head trauma or cerebrovascular disease. Neurologic examinations showed no abnormal findings except for drowsy mental status. Noncontrast computed tomography (CT) scan showed SAH in right prepontine cistern and intraven-

tricular hemorrhage in fourth ventricle. Thin section CT angiography showed abnormal vascular structure, suggesting an arteriovenous fistula (AVF) locating at the right perimedullary cistern (Fig. 1). Catheter angiography demonstrated a single channel AVF which was fed by the right neuromeningeal branch of ascending pharyngeal artery (Fig. 2A, B). The venous outflow was drained into the lateral medullary veins with reflux into the superior petrosal



Fig. 1. A computed tomographic angiography shows tortuous vascular structure in the right perimedullary cistern.

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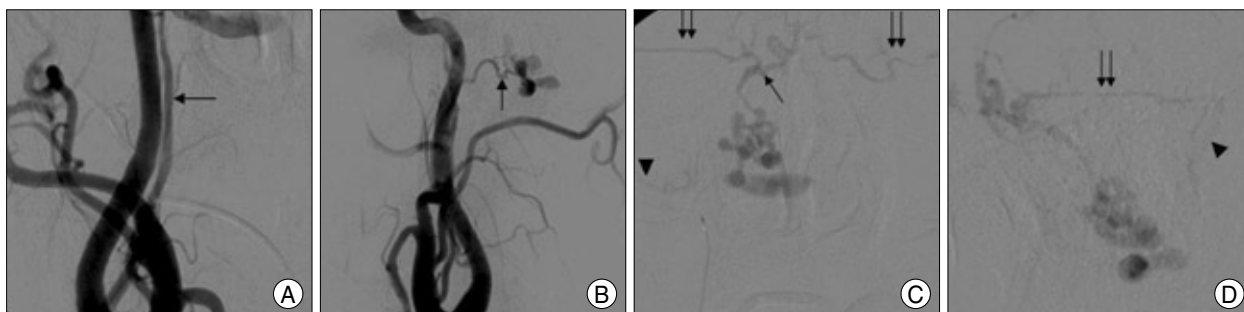


Fig. 2. Frontal (A) and lateral (B) views of initial common carotid arteriography. The dural arteriovenous fistula is fed by neuromeningeal trunk of the ascending pharyngeal artery (A, arrow) and is drained into congestive lateral medullary veins. Irregular fistulous point is noted (B, arrow). Selective injection of the right neuromeningeal trunk in frontal (C) and lateral (D) view. Reflux into transverse pontine vein (C, arrow) is seen with opacification of superior petrosal sinus (double arrow) and the sigmoid sinus (arrow head).

sinuses bilaterally which drained subsequently into the sigmoid sinuses (Fig. 2C, D).

Transfemoral transvenous coil embolization has been recognized as the treatment of choice for an intracranial DAVF^{4,5,11}. However, this was not suitable for the current case because a transvenous approach was inaccessible. Therefore, we had no choice but transarterial embolization through the right femoral artery route. A microcatheter (Excelsior; Boston Scientific, Fremont, USA) was advanced into the far distal neuromeningeal trunk of ascending pharyngeal artery through a 5F guiding catheter. And, 25% N-butyl cyanoacrylate (NBCA) in lipiodol, 0.6 mL was injected (Fig. 3). Migrations of NBCA are noted at the transverse pontine vein and right superior petrosal sinus during intraarterial injection. Immediately after the embolization, complete occlusion of the AVF was seen (Fig. 4). However, 3 days after the procedure, the patient showed diminished gag reflex and taste sense, vertigo, nausea, dysphagia and hoarseness. Magnetic resonance imaging (MRI) obtained at 5 days after the treatment showed high signal intensity in the right lateral medulla (Fig. 5). Temporal bone and angiographic CT after embolization showed glue cast in right perimedullary subarachnoid space and jugular foramen (Fig. 6). The patient underwent gastrostomy against swallowing difficulty. Selective angiogram of the ascending pharyngeal artery obtained at 3 months after treatment persistently showed complete occlusion of fistula (Fig. 7). After improvement of dysphagia, gastric tube was removed 7 months after embolization. Other lower cranial neuropathies were also improved except hoarseness.

DISCUSSION

The majority of DAVFs are known to develop in association with an adjacent dural sinus. In this case, the fistula was located at the margin of jugular foramen and was not directly associated with the sigmoid sinus or jugular bulb.

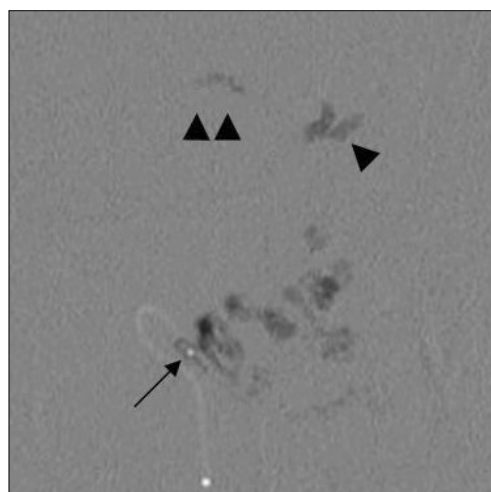


Fig. 3. Frontal view of 25% glue-lipiodol mixture injection in right distal neuromeningeal trunk show filling of dilated lateral medullary veins and migration to the transverse pontine vein (arrow head) and right superior petrosal sinus (double arrow head). A catheter tip (arrow) is located at just before fistula which lies within the cranium.



Fig. 4. Lateral view of the right common carotid artery angiogram after embolization. Venous congestion is no longer opacified.

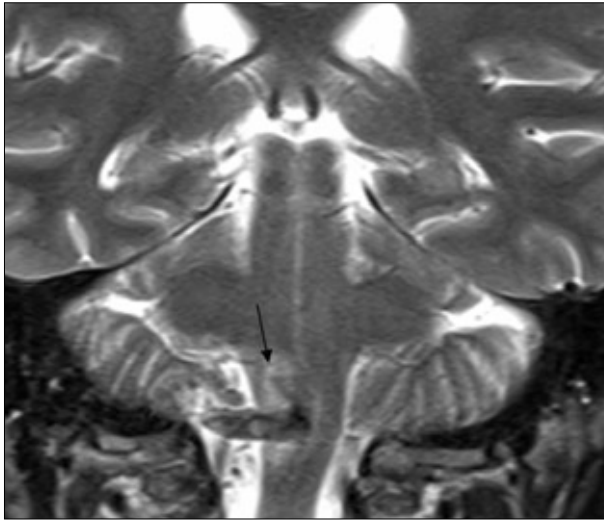


Fig. 5. Magnetic resonance imaging of the brain in T2WI coronal view. High signal intensity is noted in right lateral medulla (arrow). Flow is arrested in dilated venous structure.

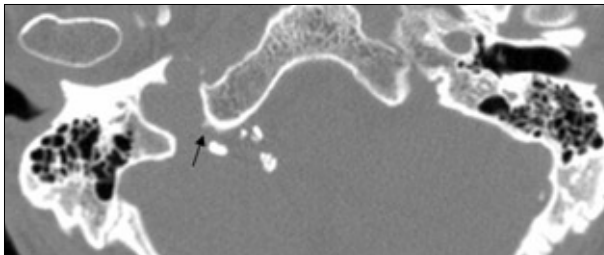


Fig. 6. Temporal bone computed tomography in axial view. Glue cast in serpentine structures is detected in the subarachnoid space on the right side of the medulla oblongata after embolization. A fistulous point is supposed to be at the margin of jugular foramen (arrow).



Fig. 7. Selective injection of the right ascending pharyngeal artery at 3 months post-embolization in frontal view. A fistula is completely occluded (arrow), but jugular branch is intact. The venous pouches are not opacified, and dural channel is noted which is not seen on initial angiogram (double arrow).

According to Lasjaunias and Moret⁸⁾, the jugular rami of ascending pharyngeal artery course around the jugular foramen. Lateral medullary vein often gives rise to an

inferior petrosal bridging vein near the foramen of Luschka, which courses along the rootlets of the nerves entering the jugular foramen to join the venous sinuses near the jugular bulb¹⁰⁾. Analysis of the postembolization temporal bone CT shows that it is likely that this fistula developed in association with the bridging vein. Glue cast was not noted in the inferior petrosal sinus, anterior condylar vein, or marginal sinus.

Noncontrast thin section CT scan such as temporal bone CT is a useful modality when we want to evaluate position of previously injected glue cast. Coil shows beam hardening artifact on CT, but glue does not.

Drainage into the cortical or perimedullary spinal cord veins results in increased pial venous pressure with a high rate of venous infarction, hemorrhage in the brain, and congestive venous myelopathy in the cord^{1,13)}. In this case report, patient showed single channel DAVF with lateral medullary drainage, which was thought to be the cause of SAH.

DAVF with localized fistula can be obliterated by passing the fistula with a small amount of liquid embolic material such as NBCA^{6,9)}. However, transarterial embolization of neuromeningeal trunk of ascending pharyngeal artery using NBCA has a risk of cranial nerve palsy^{2,3)}. Jugular branch supplies the ninth, tenth, and eleventh nerves at jugular foramen⁷⁾. Despite the risks of cranial nerve palsy, we mainly focused on occluding the fistula due to the high risk of rehemorrhage.

Transarterial coil embolization can be another choice of treatment in single channel DAVF. But, it is only feasible when coil can pass through the fistula. If fistulous portion of fistula is narrow and irregular, coil embolization is not possible. In the present case, we tried coil embolization first, but failed.

In the present case, patient developed typical lower cranial nerve symptoms (diminished gag reflex, vertigo, nausea, dysphagia, diminished taste) after embolization. Causes of these symptoms are thought to be multifactorial. First, penetration of the liquid embolic agent into the vasa vasorum of the cranial nerves IX, X, and XI arising from the jugular ramus of ascending pharyngeal artery seemed to cause infarction of the lower cranial nerves¹²⁾. Second, migration of the liquid embolic agent into the prepontine venous structures was deemed cause secondary venous infarction or congestive venous myelopathy in the lateral medulla. Even if coil embolization was not feasible in current case, occlusion of the vasa vasorum can be prevented when we use coil instead of glue for a single channel fistula. And, if we use a higher density NBCA localized to the fistulous site, migration of glue cast may have been avoided.

CONCLUSION

Transarterial glue embolization is a feasible method, only if a transvenous access is not possible in case of single channel fistula. However, we should be careful if feeding artery is neuromeningeal trunk of ascending pharyngeal artery, because of potential risk of cranial nerve palsy.

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