Transarterial Embolization of Traumatic Carotid-Superior Hypophyseal Arterial Cavernous Fistula

A Case Report

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Summary

A 26-year-old man presented with symptoms of progressive bilateral exophthalmos and swelling of the eyelids after a severe head injury. Angiography confirmed a direct carotid-superior hypophyseal arterial (SHA) cavernous fistula with petrosal sinus and intracavernous sinus drainage. Successful transarterial coil embolization of the fistula was performed with resolution of the patient's symptoms. To our knowledge, post-traumatic arteriovenous fistula between SHA and the cavernous sinus has not been previously reported. We hereby demonstrate an effective, minimally invasive method of occluding a rare fistula by transarterial embolization.

Introduction

Carotid cavernous fistulas (CCF) are rare complications of cranial base trauma. We report a case of carotid-superior hypophyseal artery cavernous fistula, which was successfully obliterated by transarterial coil embolization.

Case Report

A 26-year-old man sustained a severe head injury in a motor vehicle accident. He was admitted to hospital in coma. There was periorbital soft tissue swelling and ecchymosis of both eyes. The patient awoke 17 days after the accident, with progressive bilateral exophthalmos and swelling of the eyelids especially on the left

side. Physical examination revealed chemosis and prominent conjunctival vessels of both eyes, a left temporal bruit, and elevation of left intraorbital pressure. His visual acuity was impaired. CT revealed a dilated left superior ophthalmic vein and left orbital fracture.

Endovascular Therapy

Twenty days after the accident an initial angiogram of the left internal carotid artery verified the rapid opacification of the left cavernous sinus in the arterial phase (Figure 1A,B). The left cavernous sinus was distended, with enlarged drainage into the left inferior petrosal sinus and intracavernous sinus. Right ICA angiogram with compression of the left common carotid artery revealed not enough compensation flow through the anterior communicating artery (Figure 1C,D).

Transarterial embolization was performed soon after the cerebral angiogram. Under general anesthesia, through 6F guiding catheter, a Tracker 10 microcatheter (Boston Scientific/Target, Fremont, CA, USA) was used for superselective catheterization of the fistula. The origin of the fistula was difficult to visualize despite the acquisition of multiple projection angiograms. To try our best again and again, with the use of rapid digital subtraction angiography, the fistula was found to originate from SHA. With standard manoeuvres, the microcatheter was subsequently advanced into the fistula. Superselective angiography was performed to verify the location of the fistula (Fig-

Figure 1 Angiograms obtained in a 26-year-old man who sustained severe head injuries in a motor vehicle accident. Original lateral view left internal carotid injection demonstrates the direct left carotid-cavernous fistula (arrow) with opacification of the cavernous sinus (A). Oblique views also cannot show the exact position of the fistula (arrow) (B). Compression of the left common carotid artery, left VA angiogram arterial phase lateral view (early arterial phase C,D) demonstrates the possible fistula position (arrow) and not enough cross-filling by the posterior communicating artery.

ure 2A,B). Then we embolized left SHA with four Guglielmi detachable coils with 2 mm diameters (GDC; Boston Scientific/Target). No coils were detached within the cavernous sinus, resulting in complete obliteration.

A left carotid angiogram demonstrated complete obliteration of the fistula with a normal caliber and contour of the cavernous internal carotid artery segment (Figure 2C,D) after occlusion of the SHA at the origin of the fistula.

Outcome and Follow-up

The patient was observed in the intensive care unit overnight without complication. The next day, the intraocular pressure remained normal and the proptosis, eyelid fullness, and dilated conjunctival vessels were improved. There was no change in the vision and endo-

crine presentation of the patient. The patient was discharged on the fourth day after embolization, with relief of his conjunctival injection and chemosis. Two weeks later, ocular examination remained stable. Three months after therapy, the patient's exophthalmos and chemosis had completely resolved, with no symptoms of headache, eye pain, or blurred vision.

Discussion

Direct fistulas between the intracavernous carotid artery and the cavernous sinus are uncommon but well-described complications of cranial base trauma that occur in 0.2 to 0.3% of patients with craniofacial trauma¹. Based on angiographic studies, direct CCF are classified as type A or TCCF, which occur three times as often as the indirect types (B,C,D)².

Figure 2 Superselective injection of the (L) SHA anteroposterior (A) and lateral (B) views show the fistula (arrow) draining directly into left inferior petrosal sinus and intracavernous sinus. Postembolization internal carotid anteroposterior (C) and lateral (D) angiograms show complete occlusion of the fistula and restoration of blood flow to the internal carotid artery and the middle and anterior cerebral arteries (arrow, embolization side).

Anatomy Basis of CCF

An arterial-venous fistula can occur at five specific areas along the course of the ICA in the cavernous sinus^{3,4}. These include the anterior ascending segment of the ICA, the junction of the anterior ascending and horizontal segments, the horizontal segment of the ICA, the junction of the horizontal and posterior segments, and the posterior ascending segment.

SHA-cavernous sinus fistula may occur at the anterior ascending segment of the ICA, which has not been reported in the literature. The blood supply of the pituitary stalk came from superior hypophyseal artery, while the blood supply of the neurohypophysis came from inferior hypophyseal artery. The ventromedial aspect (the medial or medial inferior wall) of the supraclinoid internal carotid artery

is thought to arise in relationship to the perforating vessels supplying the optic chiasm and hypophysis. The largest of these vessels is referred to as the superior hypophyseal artery (SHA)⁵⁻⁷. The arterial stem went into pituitary stalk and some small branches to optic nerve and chiasm. Day was the first to use the term “superior hypophyseal artery aneurysms” to refer to these aneurysms arising medially and inferiorly from the ophthalmic segment of the ICA, suggesting their origin in relationship to the perforating branches of this segment^{8,9}. In this patient, after fracture of cranial base trauma, a distinct arterial segment of SHA may rupture into cavernous sinus, and cavernous sinus fistula occurred.

To verify the origin of TCCF, high-speed digital subtraction angiography is essential in confirming the diagnosis and delineating exact venous drainage patterns.

Treatment

The aim of treatment of CCF is to close the abnormal communication between the feeding artery/arteries and the cavernous sinus. Because of limitations with detachable balloons¹⁰⁻¹³, we often use microcoils to achieve a relatively more precise and controlled occlusion of the fistula.

Carotid-SHA cavernous fistula may carry a higher risk than normal "direct traumatic carotid-cavernous fistula", because the fistulous communication resides in the subarachnoid space. It is unlikely that the fistulous tract itself possesses the wall integrity of the parent vessel, and the patient may be at increased risk of subarachnoid hemorrhage. The transvenous approach is an available and useful method when a transarterial approach is very difficult or not indicated. Although complete obliteration of the shunt flow was achieved, transvenously overpacked coils may impede recovery from ocular palsy and also prolong nerve compression, resulting in an irreversible change. In addition, a longer transvenous approach usually leads to an insufficient therapy of injured artery after obliterating the shunting to the cavernous sinus. The fistula remnant within the subarachnoid space might provide the substrate for future aneurysmal change, which placed him at potentially increased risk for subarachnoid hemorrhage. For these reasons, a transvenous approach may be

less desirable than a transarterial approach, because it does not allow direct access to the arterial origin of the fistula.

The proximal outlet of the fistula in SHA was treated in the same manner as the neck of an aneurysm with dense coil packing^{8,9}. Fortunately, the fistula had arisen from a distinct arterial segment of SHA. This feature allowed dense coil embolization of the arterial segment itself, resulting in its occlusion at the origin of the fistula.

In this case, no adverse consequences seem to have occurred as a result of the occlusion of a single superior hypophyseal artery, although the potential for loss of vision and hypothalamic-pituitary disturbance exists.

Conclusions

The current case is the first reported case of a traumatic fistula arising from the SHA and connecting to the cavernous sinus, resulting in ocular symptoms. We successfully treated the carotid-SHA cavernous fistula by performing endovascular coil occlusion through a transarterial route.

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