Posttraumatic Intradural Internal Carotid Artery-Cavernous Sinus Fistula Associated with Ipsilateral Carotid Dissection

Transarterial Embolization with Detachable Coils

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Key words: fistula, carotidocavernous, dissection, arterial, embolization, cavernous sinus detachable coils

Summary

We describe a relatively unusual case of traumatic direct carotid-cavernous fistula in association with a giant intradural venous pouch and ipsilateral carotid dissection, related to carotid artery fistula located in the supraclinoid segment just below the origin of posterior communicating artery. Endovascular therapy could be accomplished by use of detachable coils transarterially. Awareness of an unusual intradural origin of a carotid-cavernous sinus fistula and the possibility of an embolization should be kept in mind.

Introduction

Arteriovenous fistulas of the cavernous sinus can be classified into direct carotidocavernous fistula (CCF) with high-flow shunting of blood from the internal carotid artery (ICA) to the cavernous sinus, or indirect CCF, when there is a dural arteriovenous connection¹. The direct CCF is usually due to trauma or to rupture of a cavernous carotid artery aneurysm. Traumatic CCF is almost always direct caused by laceration of the ICA segment lying within the cavernous sinus. We described an unusual case of posttraumatic CCF arising from the ICA at or near the level of posterior communicating artery (PCoMA), and draining into the

right cavernous sinus. A large venous pouch of CCF was also present, receiving arterial flow from the ICA and spilling out into the cavernous sinus.

Case Report

A 30-year-old man who sustained a severe craniofacial injury in a high-velocity accident, was admitted to the emergency room in a local hospital unconscious. Initially, he was noted to have extensive soft tissue lacerations involving the face and scalp, and right mandible fracture. CT revealed a fracture line around the sella turcica and anterior cranial base without any evidence of intracranial haemorrhage. He regained consciousness 24 hours later and was transferred to another hospital on the second day. The mandible fracture was screwed here and the patient was discharged from the hospital on the tenth day after the accident. He was neurologically intact at one-month follow-up. After three months, the metal fixator placed over the mandibular fracture was removed successfully. About a year and a half later, he experienced slowly progressive eyelids swelling and slight chemosis of the right eye.

Magnetic resonance imaging obtained upon persistence of the eye symptoms showed a large venous pouch of the cavernous sinus and evidence of right-sided CCF. As the regression

Figure 1 A 30-year-old man with a giant venous pouch and an ipsilateral intradural ICA-cavernous sinus fistula through this venous pouch accompanied by ipsilateral ICA dissection. A) Lateral right common carotid angiogram shows narrowed and irregular ICA lumen that loses its integrity in the cavernous segment. Note faintly opacified fistula originating from the supraclinoid portion of ICA, and large venous pouch of the cavernous sinus. B) Anteroposterior left ICA injection shows the cavernous sinus fistula arising from the supraclinoid right ICA segment, and the large venous pouch. C) Lateral left vertebral angiogram clearly demonstrates the patent right PComA filling the fistula. The fistula is located between the supraclinoid portion of ICA and the right cavernous sinus through the giant venous pouch which drains eventually into the right superior ophthalmic vein. D) Slightly oblique lateral view after concomitant injection of the right vertebral artery and the left ICA. The angiogram shows the exact fistula site which is located in the right ICA at or just below the origin of the right PComA. Note jet flow into the venous pouch at the fistula site (arrow). E) Roadmap image in the same projection with the figure 1D shows the microcatheter-microguidewire combination coming from the right vertebral artery, passing through the right PComA, and reaching proximal portion of the right middle cerebral artery. F) Lateral right vertebral angiogram showing occlusion of the fistula after coil embolization of the right PComA orifice joining the fistula. G) Anteroposterior left ICA injection after coil embolization of the right supraclinoid ICA segment. Retrograde opacification of the right-sided fistula is no longer seen. H) Non-subtracted lateral right ICA injection after recanalization of the previously dissected cavernous segment shows the microguidewire just inside the giant venous pouch. Note previously deposited coil pack laying in the junction of the right PComA and the right ICA supraclinoid segment. Also note contrast material stagnation in the dependent portion of the venous pouch after embolization of the main feeders of the fistula. I) Final lateral right common carotid angiogram shows complete elimination of the venous pouch and the fistula after coil occlusion of the right ICA at the level of the ophthalmic artery. Note reconstruction of the distal ophthalmic artery from the branches of the internal maxillary artery.

occurred in signs and symptoms of the right eye, the patient refused further diagnostic and therapeutic interventions. Six months later, however, he was admitted to the hospital because of uncontrollable headache. He had exophthalmos and mild chemosis of the right eye without visual loss, and distended facial veins on physical examination.

Angiography demonstrated right-sided di-

rect CCF. Selective injection of the right common carotid artery showed narrowed and irregular ICA lumen. The artery had lost its integrity in the cavernous segment, however it regained its usual calibre above the clinoid segment. It could carry very slow flow which opacified faintly the large venous pouch and the fistula subsequently (figure 1A). Selective injection of the left ICA showed a patent ante-

rior communicating artery with a good cross-filling. There was a flow steal through the right distal ICA retrogradely due to sump effect of the right direct CCF (figure 1B). After opacification of the large CCF pouch and the cavernous sinus, venous drainage was carried outside the cranium through the ipsilateral superior ophthalmic vein as well as a small cortical vein draining into the superior sagittal sinus. Selective injection of the left vertebral artery showed patent ipsilateral PComA feeding the fistula through the venous pouch (figure 1C).

Transarterial embolization of these three feeders appeared to be a most feasible option. Under general anaesthesia, a 5F guiding catheter was placed first in the right cervical vertebral artery, and 5F diagnostic catheter in the left ICA. Selective injection through both catheters concomitantly showed that exact fistula site was on the right distal ICA around the level of PComA origin (figure 1D). Activated clotting time (ACT) was checked preprocedurally and the heparin was given to keep the ACT 2.5 times baseline level (10,000 U IV bolus initially, 3000 U IV bolus one hour later). An Excelsior microcatheter (Boston Scientific, Fremont, USA) was advanced into the right vertebral artery, basilar artery, right posterior cerebral artery, right PComA and reached the fistula. By using standard manoeuvres, 0.014 inches Transend guidewire (Boston Scientific, Fremont, USA) could be advanced beyond the fistula into the distal ICA and right middle cerebral artery (figure 1E). The microcatheter was readily advanced into the distal right ICA over the guidewire.

After multiple attempts, it was possible to deploy a 3D GDC18 coil (4 mm x 8 cm) in the distal ICA stump. A second 3D GDC18 coil (3 mm x 6 cm) was anchored the first coil and then deployed mainly in the fistula orifice. Selective left ICA injection at that time showed cessation of retrograde opacification of the fistula. Finally, the right PComA was sealed with a third 3D GDC18 coil (3 mm x 4 cm) (figure 1F,G). The guiding catheter was withdrawn from the vertebral artery and placed in the right common carotid artery. Faint opacification of the fistula was still present with the right carotid artery injection. Another 3D GDC18 coil (6 mm x 15 cm) was deployed in the right ICA just before the dissected segment. Control angiogram obtained injection

through the guiding catheter showed occluded ICA at the coil site and retrograde opacification of the right supraclinoid ICA and the fistula subsequently mainly through the ophthalmic artery, therefore the coil was withdrawn. After multiple attempts without success, Transend guidewire was exchanged to 0.011 inches Terumo guidewire (Terumo Corp. Tokyo, Japan). First the guidewire then the microcatheter could pass through a thin recanalization lumen and reached the supraclinoid ICA segment (figure 1H). The artery was plugged with two GDC18 coils at the level of ophthalmic artery origin. The final right common carotid artery angiogram demonstrated closure of the fistula as well as reconstruction of the distal ophthalmic artery branches from the ipsilateral internal maxillary artery with well-opacified choroidal crescent (figure 1I).

The patient awoke well without any problem. Congestion of the facial veins disappeared immediately. IV heparin infusion (500-1000 IU/h) was continued for three days after the procedure for a target PTT of 40 seconds. He was kept on subcutaneous fractionated heparin for another four days and oral acetylsalicylic acid for one month. The chemosis of the right eye subsided within one week. At two-month follow-up, both eyes looked nearly symmetrical.

Discussion

Posttraumatic direct CCF is caused by injury to the cavernous segment of ICA¹. Trauma may also cause ICA aneurysm on the cavernous, petrous or even intradural segment, usually accompanied by basal skull fracture. In the present case, there were a large cavernous sinus pouch and a CCF arising from the ICA just below the origin of the PComA which drains into the ipsilateral cavernous sinus through the venous pouch. Ipsilateral ICA dissection below the cavernous segment was another associated finding. This form of traumatic intradural direct CCF, indeed, is very rare and only a few similar cases have been reported previously²⁻⁸. The mechanism of formation of this arteriovenous communication is speculative, but laceration of the superior dural wall of the cavernous sinus and aneurysm formation from the injured wall of the ICA with subsequent penetration and rupture into the cavernous sinus is probably the best way of explanation⁷.

Treatment of direct CCF is today via the endovascular approach. The strategy of the endovascular therapy depends heavily on the placement of embolic agents at the fistula site. Transarterial embolization is usually indicated in patients with direct CCF where there is a tear in the cavernous segment of ICA which allows the balloon or the microcatheter passing through it. An alternative would have been a venous approach in such cases with small tear or difficulty in arterial access. Usually occlusion of fistula through a patent ipsilateral ICA can be performed with detachable balloons, or with coils. In case of occlusion of the ipsilateral ICA, it can be accomplished by using collateral arc of the circle of Willis⁹, or after recanalization of occluded ICA¹⁰.

The ipsilateral carotid dissection, the high carotid artery fistula at the level of PComA, and the giant venous pouch between the fistula site and the cavernous sinus are unusual angiographic features of the present patient which preclude classical endovascular approaches of direct CCF treatment such as the transarterial or transvenous detachable balloon embolization of cavernous sinus or coil packing of cavernous sinus harbouring the CCF either transarterially or transvenously. Selective plugging of all three arterial pedicles (i.e., distal ICA segment retrogradely, PComA, and supraclinoid ICA segment) appeared to be a most appropriate endovascular way. We prefer detachable coils since they can be placed and replaced several times until an ideal position has been achieved. Electrolytically detachable coils offer an adequate, safe way of treating various types of arteriovenous fistula on brain-supplying vessels by the endovascular route¹¹. We could plug two arterial pedicles (the distal ICA and PComA) simultaneously with the same microcatheter coming from the vertebro-basilar system, passing through the PComA, and reaching in the distal ICA.

The microcatheter navigation described above is known as retrograde approach and should only be considered for those cases in which the antegrade route through the parent artery will not provide adequate access. Debrun et Al⁹ used this technique successfully in a subgroup of three patients with fast-flow direct CCF associated with ipsilateral ICA occlusion. With this technique we were able to occlude two arterial pedicles of the patient at

the same time. The plugging of last one (i.e., the supraclinoid ICA) necessitated catheter navigation through the previously occluded and recanalized ICA lumen, since proximal plugging of the artery did not eliminate the CCF because of the retrograde filling of ICA through the ophthalmic artery. Wilms et Al¹⁰ have described a case of direct CCF and ipsilateral dissection in which the cavernous sinus was catheterized through the occluded ICA, and embolization performed successfully with coils. In our patient, the ICA had a few thin and irregular recanalization vessels on the previously occluded segment. Carefully selected microcatheter and microguidewire allowed us to pass through this segment and reach desired position. After occlusion of supraclinoid ICA at the origin of ophthalmic artery, we obtained complete elimination of this unusually high direct CCF.

Open surgery was the treatment option in some reported patients with posttraumatic intradural ICA-cavernous sinus fistula with accompanying intradural aneurysm^{2,4,6,7}. Komiya et Al³ have first tried to occlude the CCF with endovascular route by using detachable balloons, but the embolization was complicated with early aneurysm rupture and subarachnoid haemorrhage. All these authors, including Komiya et Al who prefer endovascular therapy initially, have concluded that a direct CCF associated with a posttraumatic intradural ICA aneurysm should be treated by direct surgery (either trapping of ICA or neck clipping of aneurysm) rather than the use of a detachable balloon^{3,4,6,7}. Similar to these authors, we also think that especially in early clinical period, intraaneurysmal balloon inflation may cause aneurysm rupture in such cases. The first successful endovascular therapy has been accomplished by transvenous coil deposition of cavernous sinus in a patient with a fistula of the PComA and cavernous sinus which was reported by Kinugasa et Al⁵.

The recent article by Weaver et Al⁸ described a case with a traumatic fistula between the PComA and the cavernous sinus accompanying a PComA aneurysm that was treated successfully with transarterial coil embolization. They occluded the aneurysm first by transarterial coil packing followed by coil embolization of the parent artery (i.e., the PComA) which excluded the lesion completely from the circu-

lation. Both the transvenous and transarterial coil packing of the cavernous sinus were not feasible in the present patient since there was a large venous pouch in the cavernous sinus. For this reason, we focused our attention on the arterial pedicles of the CCF, and we were able to occlude all three pedicles successfully by using the retrograde microcatheter manipulation through the circle of Willis, the microcatheter navigation through the previously occluded ICA, and the coil deposition of the arterial pedicles, respectively.

Considering the embolization technique used in the present patient, the bifemoral approach and the anticoagulation scheme may be a matter of discussion. The bifemoral approach allows us to perform embolization under continuous angiographic control especially in patients having AV fistula with multiple arterial pedicles, as in the present case. The bifemoral approach may also delineate more precisely the angiographic features of the vascular lesion and help understanding the complex anatomy. Anticoagulation is used traditionally in most (if not all) neurovascular interventions. After occlu-

sion of an intracranial AV fistula with large venous pouch and/or tortuous and dilated drainage veins as in the present case, retrograde propagation of a thrombus in the sluggish venous flow is one of the dangerous condition which may result in venous stasis, ischemia, or even intracranial haemorrhage¹². Strict and extended anticoagulation scheme plays, therefore, an important role to prevent this untoward clinical condition, and must be kept in mind in all patients with similar features.

Conclusions

In conclusion, careful angiographic examination is mandatory in all cases with direct CCF. According to the architecture and haemodynamic features of the fistula as well as the characteristics of intracranial arterial and venous anatomy, endovascular route may be safe and alternative to surgery even in such very rare cases with posttraumatic intradural ICA-cavernous sinus fistula associated with a large venous pouch and ipsilateral carotid artery dissection.

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