Treatment of Carotid Cavernous Fistula Secondary to Rupture of a Giant Intracavernous Carotid Aneurysm

Transarterial Coiling of Aneurysm and Carotid Compression A Case Report

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Summary

Carotid cavernous fistula secondary to ruptured giant intracavernous aneurysm of the internal carotid artery is rare. We report a case of direct carotid cavernous fistula secondary to rupture of a giant intracavernous ICA aneurysm. The presence of mirror or twin aneurysms of bilateral ophthalmic arteries raises therapeutic challenge.

Coiling of the intracavernous aneurysm could partially occlude the fistula. Complete closure of the fistula was facilitated by secondary carotid compression.

Introduction

Rupture of intracavernous aneurysms of internal carotid artery (ICA) usually lead to direct high-flow carotid cavernous fistula (CCF). Their treatment is likely to be more complicated than ordinary post-traumatic direct CCF due to the possibility of underlying collagen vascular disease and the risk of aneurysmal rupture into sphenoid sinus. Special consideration has to be taken when there are coexisting vascular abnormalities such as multiple arterial aneurysms.

Case History

A 36-year-old lady presented with headache, hyperesthesia of the left face and pulsatile tinnitus for one week. Her past health was good and there was no family history or clinical signs of collagen vascular disease. Neurological examination showed left ptosis and left third cranial nerve palsy. Fundoscopy was normal.

Cranial computed tomography showed no subarachnoid haemorrhage or erosion into the sphenoid sinus. Digital subtraction angiogram showed a ruptured giant aneurysm of the cavernous part of the left ICA with formation of a direct CCF. The venous drainage is bilateral, towards superior and inferior petrosal sinuses. No significant drainage to superior ophthalmic vein or pial reflux is noted (figure 1). Three-dimensional reconstruction of DSA revealed the course of the left third cranial nerve surrounded and likely compressed by the arterialized cavernous sinuses (figure 1), accounting for the clinical presentation of third nerve palsy. Small aneurysms of 3 mm in size were noted at the ventral aspect of the junction of ophthalmic arteries and internal carotid arteries bilaterally. They were arising at corresponding location in both sides, with the configuration of mirror or twin aneurysms (figure 2). Further intervention was planned.

Figure 1 Frontal (A) and lateral (B) views of left internal carotid angiogram show the direct CCF secondary to ruptured giant intracavernous ICA aneurysm. 3D reconstruction of DSA (C) demonstrates the direct CCF with a small aneurysm at carotid-ophthalmic junction, and the course of the third cranial nerve near the roof of cavernous sinus (D). An anatomical specimen shows the relationship of the third cranial nerve in the cavernous sinus (E).

Subsequently, there was rapid deterioration with almost complete palsies of the left third, fourth and sixth cranial nerves. Visual acuity of the left eye was also impaired. DSA was repeated, showing more extensive drainage, involving into bilateral superior and inferior petrosal sinuses and pterygoid plexuses.

There was also drainage into the ipsilateral cortical and basal veins, raising the concern of risk of parenchymal and subarachnoid haemorrhage (figure 3). The left intracavernous carotid aneurysm was embolized with GDC18 coils transarterially. There was nearly complete exclusion of the aneurysm. However, part of the fistula was still patent after the procedure (figure 4). In view of the potential of spontaneous thrombosis of the CCF, the patient was motivated to perform carotid compression. After eight weeks, there was disappearance of tinnitus and improvement in ptosis and oculomotor palsy clinically. Cerebral angiogram was repeated and showed stable exclusion of the intracavernous carotid aneurysm and complete closure of the fistula (figure 5).

Discussion

Intracavernous aneurysm of the internal carotid artery can produce variable symptoms through compressive effects of the gradually enlarging aneurysm. Patients may present with any combination of the following: headache, pain or sensory loss in the distribution of the trigeminal nerve; ophthalmoplegia or impairment in vision. Rupture of intracavernous carotid aneurysm usually lead to direct CCF of high blood flow; though it is an uncommon etiology of CCFs. It is more likely to occur in older patients, with female predominance. Post-traumatic direct CCFs are far more common and are typically seen in young adult male victims of road accidents. Clinical features include pulsatile tinnitus, bruit, proptosis, conjunctival injection and chemosis. Rupture of intracavernous aneurysm into the sphenoid sinus can result in intractable epistaxis. Rarely, rupture of intracavernous aneurysm results in subarachnoid or subdural haemorrhage^{1,2}.

Surgical procedures for intracavernous ICA aneurysms, including direct clipping of the aneurysm and surgical ligation of the proximal ICA are infrequently performed nowadays. Because endovascular treatment is less invasive, less time-consuming for treatment and reco-

Figure 2 Frontal (A), lateral (B) views, and 3D reconstruction (C) of right internal carotid angiogram show a small aneurysm at the right carotid-ophthalmic junction.

Figure 3 Frontal (A) and lateral (B) views of pre-embolization left ICA angiogram show more extensive drainage of CCF compared to figure 1.

very, and with low complication rates, it has increasingly replaced surgical treatment³.

Transarterial balloon occlusion is effective in occlusion of direct CCFs⁵, while preserving blood flow in ICA. The detachable balloon is positioned through the tear of the arterial wall into the venous spaces of the cavernous sinus and then inflated. Small fistulae will usually be closed. For large fistulae, a larger balloon or additional balloons may be required. Transvenous balloon placement is more difficult as the balloon advances against the direction of blood flow⁶.

Coiling of a ruptured intracavernous carotid aneurysm via the transfemoral venous route with successful closure of the CCF has been described⁷. Another author has reported a case of transvenous coil embolization of a direct CCF and an ipsilateral ICA dissection⁸. In both cases, a non-detachable balloon was temporarily inflated at the level the fistula in order to prevent the migration of coils into the arterial system.

In our case, the presence of mirror aneurysms at bilateral ophthalmic arteries imposes constraint on the options of treatment.

Figure 4 Frontal (A) and lateral (B) views of post-embolization left ICA angiogram show nearly complete exclusion of the intracavernous aneurysm with decreased shunt through the direct CCF.

Occlusion of ICA, either by surgery or endovascular approach, is an aggressive and injudicious measure. There is a five percent risk of permanent, predominantly ischemic complication³. Even if the patient could tolerate the occlusion, sacrificing the ICA would add substantial risk and difficulty in the subsequent management of the ophthalmic artery mirror aneurysms.

Transarterial occlusion with detachable balloon has been a commonly used and effective method to treat direct CCFs. However, there is potential risk of compression or even occlusion of the parent artery. In the diseased part of the artery that give rise to a giant aneurysm, inflation of a balloon through the tear might further damage the vessel. Even if the fistula can be successfully occluded by a balloon, there is still risk of re-rupture of the aneurysm.

We have chosen the approach of transarterial coiling of the giant aneurysm, with the goal of treating the aneurysm and occluding the CCF at the same time. The patency of the parent artery can be preserved. Transarterial GDC embolization of a ruptured intracavernous aneurysm with successful occlusion of the CCF has been reported⁹. In our case, transarterial coiling of the intracavernous aneurysm has partially diminished the shunt from ICA to the cavernous sinus. Complete occlusion of the fistula with further coiling of the aneurysm was not attempted because of the risk of compromising the parent artery. Thrombosis of the coiled aneurysm was anticipated, which might secondarily occlude the fistula. This turned out to be the case and the process was facilitated by motivating the patient to perform carotid compression.

Carotid-jugular compression was well described as a conservative treatment of CCF, mainly of dural type^{10,11}. The compression transiently decreases the arterial flow while simultaneously increasing the venous pressure; promoting thrombosis in the fistula. The initial compressions should be observed to ensure that cerebral ischemia or a vasovagal attack does not occur. Patients should compress the involved carotid artery and jugular vein with the contralateral hand so that if the patient develops weakness from cerebral ischemia unrecognized by the patient, the hand will fall away and discontinue the therapy.

The compressions are performed with the

Figure 5 Frontal (A) and lateral (B) views of left ICA angiogram performed eight weeks after embolization shows complete closure of CCF.

patient lying or sitting to reduce the risk of injury in case of vagal attack. This therapy is contraindicated in patients with atherosclerosis involving the common carotid artery with risk of dislodging plaque or thrombus. Hypercoagulability is another contraindication. Patients with cortical venous drainage, as in our patient, should not compress their jugular veins. This would further compromise their venous drainage, which could increase the risk of haemorrhage.

Spontaneous closures of CCFs have been reported, most of them were of indirect or dural type. This is rare in direct CCF, where the shunt has a high rate of blood flow^{12,13,14}. Possible mechanisms include potential thrombotic effect of iodinated contrast medium^{15,16}; stasis due

to angiography^{15,16}; and venous stasis due to increased intracranial pressure¹⁷. In our case, apart from carotid compression, other thrombogenic factors including contrast medium, aneurysmal coiling, and venous stasis due to angiography may also contribute to the complete closure of the partially occluded CCF.

Conclusions

This case demonstrates the successful treatment of a ruptured giant intracavernous ICA aneurysm by transarterial coiling under the constraint of mirror aneurysms of ophthalmic arteries. The subsequent complete occlusion of the direct CCF is likely to be facilitated by carotid compression.

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