Traumatic Cerebral Aneurysm Associated with a Contralateral Traumatic Carotid Cavernous Fistula A Case Report

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Summary

This report documents the management of a traumatic carotid aneurysm (TCA) with a traumatic carotid-cavernous fistula (T-CCF) of the contralateral internal carotid artery (ICA) following a closed head injury. A 38-year-old man presented with severe traumatic subarachnoid hemorrhage and pneumocephalus due to a severe head injury.

Four months after the accident, the patient presented with clinical symptoms of exophthalmos and retroorbital bruit. Cerebral angiography showed a TCA of the IC-PC region, which coexisted with a contralateral T-CCF. Both lesions were successfully managed with an endovascular treatment using coils to isolate a fistula from the ICA, and direct surgical trapping of the intracranial ICA to eliminate a TCA.

Post-operative angiography revealed a good cross-flow through the anterior communicating artery from the contralateral ICA, which was completely obliterated by the T-CCF. No additional surgical or endovascular procedure for traumatic vascular injuries was required. The patient remained asymptomatic during the clinical follow-up period of 24 months. The goal of traumatic carotid injuries is the selective elimination of the vascular pathologic injury with asymptomatic state, using direct surgery and/or an endovascular treatment.

Introduction

Traumatic carotid-cavernous fistula (T-CCF) is the common carotid vessel injury requiring endovascular treatment, whereas traumatic cerebral aneurysms (TCAs) of the internal carotid artery (ICA) are rare complications. A T-CCF presents with cranial bruit, chemosis, proptosis, or decreased visual acuity. In contrast, a TCA is frequently asymptomatic and probably an underdiagnosed condition, if it is not ruptured fatally. The most common sites for TCAs, which are usually associated with basal skull fractures, are the cavernous and petrous portions of the ICA^{1,2}. T-CCFs with a concomitant TCA of the ICA are rare: only ten cases of a T-CCF with coexistence of a TCA have been reported³⁻⁹. Furthermore, no case of a T-CCF with a TCA of the contralateral ICA has been described. In the last two decades, endovascular treatments have been improved for diseases on the extracranial and intracranial vasculature. These minimally invasive options are considered to be the feasible treatment for a number of conditions^{10,11}. An endovascular technique was used in this case with direct surgery.

Case Report

This 38-year-old man was admitted to our hospital following a fall from 11m in which he

Figure 1 Preoperative left CA angiograms. Upper left: Anteroposterior view demonstrating of a good cross-filling of the right MCA territory. Upper right: Lateral view revealing a traumatic carotid-cavernous fistula, which was mainly drained into superior orbital vein. Lower left and right: preoperative 3D left CA angiograms demonstrating the orifice of fistula clearly.

suffered a severe head injury. On arrival at the hospital, his status was 65/35 mmHg in BP, 36 rpm in RR, 35.7°C in BT. He was comatose, 3(E1V1M1) in GCS, but isocoric, and his light reflexes were prompt on both sides. Initial cranial CT demonstrated traumatic subarachnoid hemorrhage and pneumocephalus. Computed tomography scans in other regions obtained on admission revealed lung contusions on both sides and rib fractures from 2nd to 7th on the right side, a pelvic fracture, a scapula fracture on the right side. He was in shock, transarterial embolization was performed to arteries in the pelvis with monitoring of intracranial pressure. BP was stabilized after the procedure with blood transfusion. One month after injury, his level of consciousness had improved

to 14 (E4V4M6) in GCS. Abducens nerve palsy on the right side remained neurologically. He was transferred to another hospital for rehabilitation.

Four months after the accident, he was noted to have proptosis of the left eye and left ocular bruit. On the days following the appearance of symptoms cerebral angiography was performed to clarify the causes of these symptoms. Angiography showed a T-CCF (figure 1) on left ICA with a contralateral aneurysm (figure 2) of IC-PC region. Good cross-filling of the right MCA territory was also revealed on the left ICA. Moderate steal through the fistula was seen in left internal carotid angiography. This aneurysm with a contralateral T-CCF was not differentiated completely as traumatic from

Figure 2 Preoperative right CA angiograms. Above: early (upper left) and late (upper right) phases of lateral view demonstrating aneurysm of internal carotid –posterior communicating artery, which showed the delayed filling of the sac. Lower left: anteroposterior view with compression of the left cervical CA revealing the orifice of T-CCF. Lower right: 3D angiogram showing the orientation of the origin of the right posterior communicating artery.

non-traumatic origin. Direct surgical treatment was selected, which had options of aneurysmal neck clipping or trapping and sacrifice of the parent artery, because of its rarity. First of all, endovascular treatment for T-CCF was performed. A No. 7 French guiding catheter (Envoy; Cordis Corp., Miami, FL) was selectively placed in the proximal right ICA through a transfemoral approach. Systemic heparinization was achieved, and the patient's activated clotting time was maintained at about twice its baseline level.

We attempted a transarterial endovascular approach to deliver detachable balloons to the CCF. A transvenous approach was made through the left facial vein (figure 3). A microcatheter (Renegade) was inserted in the cav-

ernous sinus. GDC embolization was first performed around the origin of the inferior petrosal sinus. After this sinus was confirmed to be occluded, coil embolization continued around the fistula site. A T-CCF was totally obliterated with the patency of the parent artery. Heparinization was discontinued 24 hours post-treatment. There were no episodes of distal embolization, vessel disruption/ruptures, new neurologic deficits, or puncture site complications during the procedures. The presenting symptoms and signs of T-CCF dramatically improved during the postprocedural period. One week after endovascular treatment, direct surgery was performed for the aneurysm of the right IC-PC region. The arterial wall around the aneurysmal neck seemed to be dissected

Figure 3 Left: A microcatheter (Renegade) through the left facial vein delivering GDC. Center and Right: anteroposterior (center) and lateral (right) view of left CA angiograms obtained after GDC embolization, demonstrating complete obliteration of T-CCF.

with a crack (figure 4). This wall was so fragile that direct repair of the wall was impossible. We checked a good cross-flow from the left ICA by Doppler, and trapping of left ICA was performed.

After the intervention, the patient underwent a complete neurologic examination, after which his neurologic status was monitored closely for 24 hours. Angiography performed at 21 days after these procedures revealed complete obliteration of the CCF, with preservation of the parent artery, as well as a good cross-filling of right MCA branches from the left ICA through anterior communicating artery. No additional surgical procedures for vascular lesions were required. There were no delayed neurologic or vascular complications, and no lesions recurred during the follow-up, the patient was assessed as functionally improved or clinically stable. Angiography performed one year post-operatively revealed persistent obliteration of the CCF, with a patent parent artery (figure 5). The patient was neurologically intact at the last clinical follow-up 24 months after these procedures.

Discussion

Traumatic intracranial aneurysms are rare, accounting for less than 1% of intracranial aneurysms^{2,12}, although vascular injuries were found to be 25% in patients carefully screened with facial bone fracture, closed head injury, or a history of serious hyperextension-rotation mechanism¹³. The concomitant appearance of a traumatic intracranial aneurysm and a CCF secondary to head trauma is rare^{4,5,15,16,7,17}. Furthermore, no case of T-CCF associated with a contralateral TCA due to head injury has been

reported. A TCA may be clinically silent because most TCAs are asymptomatic until fatal rupture, and most patients are referred for treatment of the T-CCF and its related visual problems. Missed diagnoses and treatment may result in life-threatening SAH, followed by severe disability and death³. In particular, TCAs of the supraclinoid ICA may follow a poor natural course, with high mortality and morbidity rates^{18,5,19,20,2,21,12}. Aggressive management is required to prevent fatal hemorrhage or thromboembolic cerebral ischemia, but it is difficult to manage.

Several angiographic features suggesting TCAs have been reported, such as an irregular shape, delayed filling and emptying of the sac, an orifice which is not located at the common arterial branching points, and no visible neck^{20,22,5,23,19,20,2,21,12}. In the present case, although there were a history of trauma and pneumocephalus on initial CT, which indicated a suspect a basal skull fracture, the co-existence of a T-CCF, the delayed filling of the sac, an irregularity of the parent arterial wall near the aneurysm, TCA could not be diagnosed definitively before treatment because of a clearly visible neck, the common location of the aneurysm, internal carotid-posterior communicating artery region and, furthermore, no case had been reported as TCA in association with a contralateral T-CCF.

Traumatic cerebral aneurysms should be obviously treated because they are life-threatening lesions⁴. Most TCAs have a broad orifice, pseudo-wall, and are fragile, their neck is often difficult to confirm in the acute phase. Coil embolization into the TCA is dangerous because of the tendency of aneurysmal rupture during the procedure. The first five of ten reported

Figure 4 Above: Intraoperative photographs showing the crack of the right CA after clipping (upper left), followed by trapping (upper right). Below: Photomicrographs showing no collagenous fiber, without arterial wall component (hematoxylin and eosin stain, original magnification x100 (lower left) and x400 (lower right)).

Figure 5 Postoperative left CA angiograms. Early (left) and late (right) phase of anteroposterior view with compression of the right cervical CA revealing a good cross-filling of the right MCA territory.

cases of traumatic aneurysms of the supraclinoid ICA associated with CCF were operated by clipping the aneurysm neck through a direct surgical approach⁷, or by trapping the aneurysm and sacrificing the parent artery^{5,6,8}. However, if at least four to six weeks has passed from the onset, the traumatic aneurysmal wall may withstand safe delivery of an embolic device. The latter five of these ten cases were treated by endovascular techniques: a stent placement⁴, and a coil embolization into TCAs^{3,4}. In our case, which could not be eliminated preoperatively as a non-traumatic aneurysm, direct surgery was selected. The operations was initially designed to clip an aneurysmal neck, but finally an internal trapping of the ICA was performed because of the fragility of the arterial wall, and an EC-IC bypass was not required because a good collateral blood supply was expected.

In direct CCFs, transarterial embolization with detachable balloons has been recognized as the first-line treatment³³, which is not always fortunate because of a mismatch of size of the orifice and balloon size or the existence of a sharp angle^{34,35}. Various techniques can be used to treat the CCF with preservation of the ICA: transarterial embolization with microcoils, transvenous embolization through the inferior petrosal sinus or through the ophthalmic vein³⁴, through which is not difficult to advance coils into the cavernous sinus. A stent placement within the ICA allows coils to be deposit-

ed around the ICA safely, because the stent within the arterial wall during coil embolization facilitates the recognition of the arterial lumen margin and the prevention of a protrusion by the coils into the artery^{36,4}. In our case, transarterial embolization with detachable balloons failed and was followed by transvenous embolization with coils, which successfully isolated vascular pathologic injury with the preservation of the ICA without a stent.

We performed the treatment of T-CCF first because it seemed to be a general consensus of the treatment for T-CCF, which allowed the preservation of the patency of ICA. Direct surgery to the contra-lateral aneurysm, which could not be eliminated as non-traumatic, was performed after treatment of the T-CCF, which resulted in the trapping of the ICA. Pathologic findings proved this aneurysm to be traumatic.

If there is an aneurysm in association with a contralateral T-CCF, clinical history and angiographic findings should be checked carefully to identify whether the aneurysm is traumatic or non-traumatic. If the aneurysm seems to be traumatic, the order of treatments should be considered according to the situation, in which the treatment of T-CCF was performed first, followed by trapping of the ICA in our case. Less invasive techniques are indicated for the management of traumatic intracranial internal carotid injuries to keep the distal flow from injuries or preserve the patency of the parent artery.

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