

Traumatic Carotid Cavernous Fistula Caused by Intradural Aneurysm Rupture: A Case Report

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Traumatic carotid cavernous fistula (CCF) associated with cerebral aneurysm is a rare condition. Early detection of the traumatic cerebral aneurysm (TCA) before traumatic CCF occlusion is difficult, largely because of steal phenomenon of the fistula, masking by complex venous drains and a nearby parent artery, and a latent period of the TCA. In addition, traumatic CCF caused by a combined aneurysm rupture is an extremely rare condition. It is a dangerous condition in which treatment should be performed immediately or even on an emergency basis. A 31-year-old man developed traumatic CCF after traffic accident. Cerebral angiography revealed a direct CCF communicated with an intradural aneurysm on the origin of right posterior communicating artery (PcomA). Successful transarterial coil embolization was achieved after consecutive two trials.

Key Words : Traumatic carotid cavernous fistula; Intradural aneurysm; Traumatic rupture; Coil embolization

Direct traumatic CCF is relatively common pathology requiring endovascular treatment. Most direct CCFs are secondary to traumatic injury involving the cavernous segment of the internal carotid artery (ICA). Rupture of an intracranial aneurysm (esp. intracavernous aneurysm) leads to CCF, epistaxis, or subarachnoid hemorrhage. On the contrary, TCAs comprise less than 1% of all intracranial aneurysms and are usually associated with penetrating head injuries or contiguous skull fractures. TCAs are primarily located in the peripheral cerebral vasculature or in the skull base. The circle of Willis is a relatively uncommon location for

TCAs. In a separate lesion, a patient with CCF usually presents with Dandy's triad of pulsatile exophthalmos, chemosis, and bruit. In contrast, a TCA is frequently asymptomatic until its fatal rupture (1). Traumatic CCF caused by an intradural TCA rupture is an extremely rare condition. Surgical treatments such as trapping and sacrifice of the parent artery or direct surgical ligation of the aneurismal neck are often not possible because of their potential risks. We report a case of a traumatic CCF caused by rupture of the supraclinoid ICA aneurysm that was successfully treated by using detachable coils.

CASE REPORT

History

This 31-year-old man was transferred to our hospital for the treatment of CCF. About 8 months earlier, the patient was initially admitted to another hospital following pedestrian traffic accident in which he suffered a severe closed head injury. Initial mental

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status was stuporous. In the neurosurgical department of the hospital, emergency craniectomy for cerebral contusion, ventriculo-peritoneal shunt for hydrocephalus, and cranioplasty for skull defect were performed in regular sequence. The patient has suffered from swelling around right eye since trauma. After the condition of the patient improved to a certain extent, cerebral angiography was performed to rule out CCF and CCF was revealed.

Examination

On admission, his consciousness level was relatively clear and vital signs were stable, although he suffered from swelling around right eye and speech disturbance. There were some neurological deficits such as motor dysphasia and blindness in the right eye. There were also right ocular bruits and proptosis of the right eye. Angiography demonstrated a high-flow CCF communicated with aneurysm on the origin of right

PcomA (Fig. 1). The Mehringer-Hieshima maneuver (gentle ipsilateral ICA injection during manual compression of the ipsilateral carotid artery) (Fig. 2A) and the Heuber maneuver (ipsilateral carotid compression during vertebral artery injection) (Fig. 2B) revealed the fistula site, which was the aneurismal dome. The nature of the aneurysm was chronic-flow relate aneurysm or healed traumatic dissecting aneurysm. The fistula drains into superior and inferior ophthalmic veins, sphenoparietal sinus, superficial sylvian vein, vein of Labbe and bilateral inferior petrosal sinuses. Angiogram obtained after the right carotid compression of the left ICA (Fig. 2C) revealed the good patency of the anterior communicating artery. Right external carotid artery (ECA) angiogram (Fig. 3) reveals was another fistula, which was fed by right middle meningeal artery. Treatment strategies were direct surgical clipping of the aneurismal neck and endovascular embolization using with coils and/or

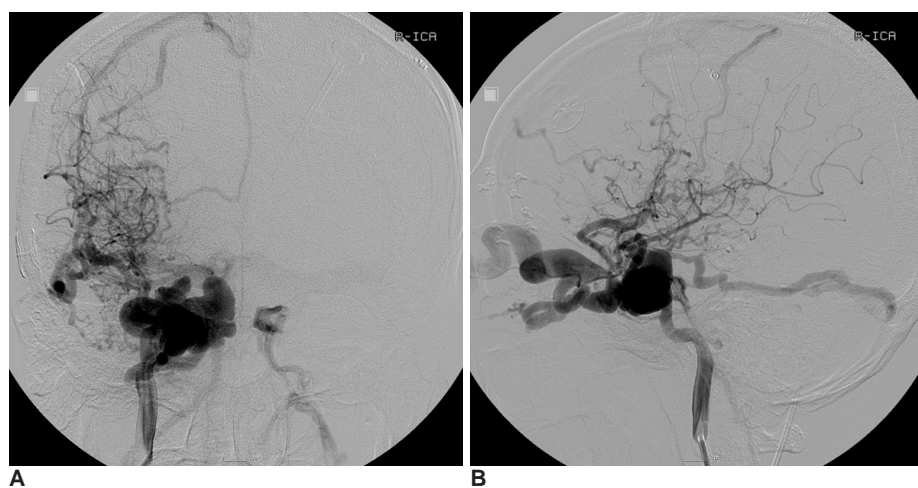


Fig. 1. Right internal carotid artery (ICA) angiograms (A: frontal view and B: lateral view) show a direct CCF with high-flow shunt with venous drainage to superior and inferior ophthalmic veins, sphenoparietal sinus, superficial sylvian vein, vein of Labbe and bilateral inferior petrosal sinuses. However, the conclusive diagnosis of the aneurysm is very difficult in these views.

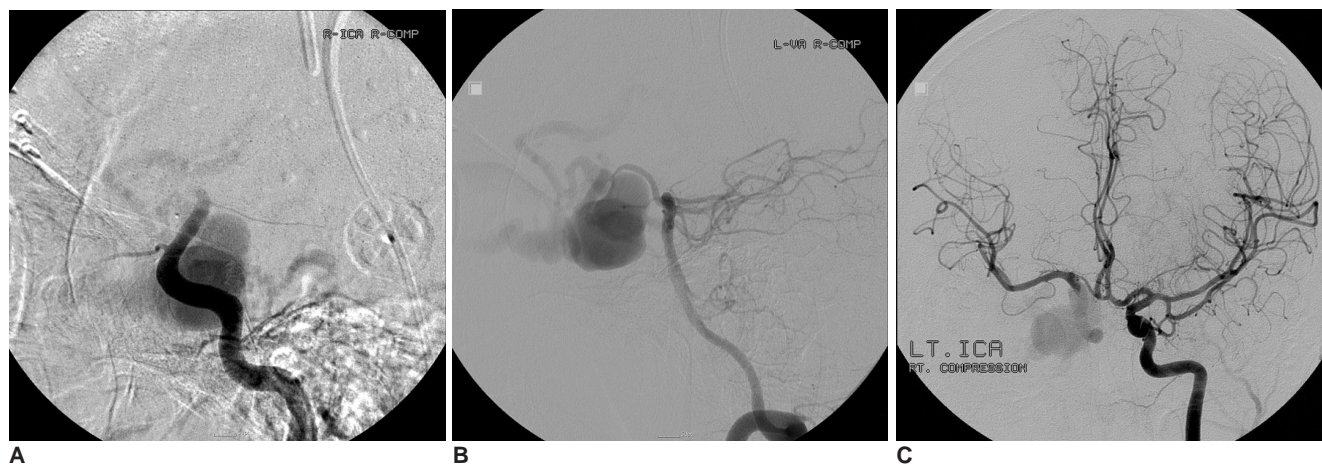


Fig. 2. Angiograms obtained after the right carotid compression of the right ICA (A), the vertebral artery (B), and the left ICA (C) reveal the accurate location of the fistula, which is the dome of the posterior communicating artery aneurysm and the good patency of the anterior communicating artery.

balloon. Direct surgical approach was thought to be difficult and risky in our patient because he had already received extensive surgery. Therefore, we choose endovascular treatment with coil embolization to occlude the fistula and aneurysm.

First embolization

Endovascular obliteration of the aneurysm was achieved by using multiple detachable coils (total length: 251cm). Relatively loose packing in the cavernous sinus and compact packing in the aneurysm was obtained through the transarterial approach. In the postembolic ECA angiography, there was continued fistula, which was seen in the initial angiography. Complete occlusion was achieved by glue casting. Angiography performed at the termination of the procedure demonstrated total occlusion of the aneurysm and the CCF (Fig. 4).

Clinical course (including second embolization)

There were no procedural complications and the

patient recovered from ocular swelling. However, he had a recurrent headache and right ocular swelling after two weeks. In the follow up angiography, there was recurred aneurysm and CCF owing to coil modification (Fig. 5A). Successful second embolization for the recurred fistula was undertaken and involved the deposition of multiple detachable coils (total length: 92 cm) (Fig. 5B). Postoperatively, the patient had no complicated events, and ocular symptoms resolved within several days.

DISCUSSION

Traumatic CCFs are the common cerebrovascular injury requiring endovascular treatment. On the contrary, TCAs of the ICA are rare complications, resulting from both penetrating and nonpenetrating head injuries. The most common sites for TCAs are the cavernous and petrous portions of the ICA; these injuries are often associated with basal skull fractures. Other sites, such as the supraclinoid ICA, are seldom

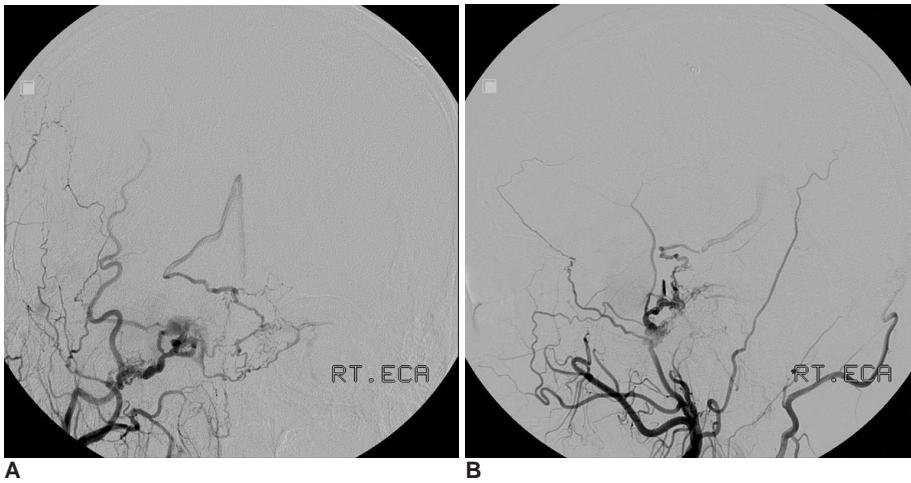


Fig. 3. Right external carotid artery (ECA) angiograms (A: frontal view and B: lateral view) show another fistula, which is fed by right middle meningeal artery.

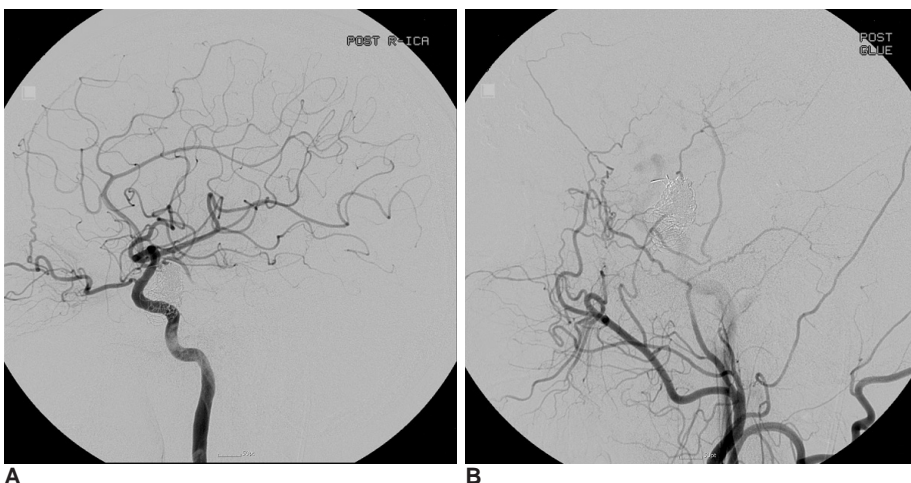


Fig. 4. Angiograms performed at the termination of the procedure (A: ICA and B: ECA) reveal total occlusion of the aneurysm and the CCF.

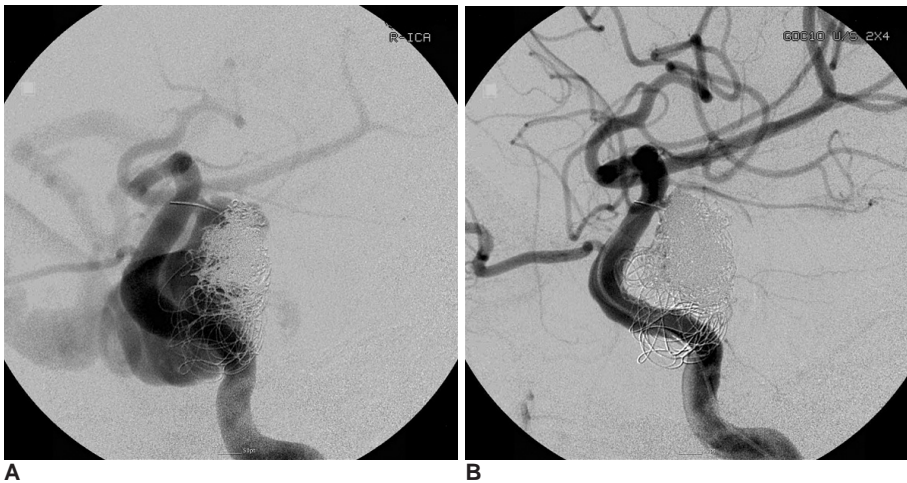


Fig. 5. Angiograms obtained after the recurrence of the symptoms reveal recurrent aneurysm and CCF (A), which was embolized with detachable coils (B).

affected (2). Of the 248 TCAs reported, 48% involved the intracranial ICA. Among them, 69% were situated in the intracavernous and petrous ICA, whereas 31% were located at the supraclinoid portion (3). Traumatic CCFs are rarely associated with a concomitant TCA.

Clinically, a traumatic CCF manifests as a cranial bruit, chemosis, proptosis, or decreased visual acuity. In contrast, a TCA is frequently asymptomatic until its fatal rupture. TCAs of the intracranial ICA and its branches are rare, constituting only 0.15 - 0.4% of all intracranial aneurysms (4).

Pathoanatomic criteria for TCAs are severely lacking. Several authors report that the following typical angiographic features may be of help in the characterization of TCAs: delayed filling and emptying of the sac, an irregular contour, no visible neck, and an ostium not located at the common arterial branching points (5, 6). However, Fox regards a history of recent trauma as the main criterion for the diagnosis of a TCA (3).

Most traumatic CCFs are benign and involve an impairment of visual acuity. However, there are some hazardous conditions in which endovascular embolization should be performed immediately. These include the followings: 1) Progressive visual loss. 2) Epistaxis. 3) Sphenoid sinus aneurysm. 4) Comatose patients in whom intracranial lesions have been excluded. 5) Rerouting of the venous drainage due to balloon migration anteriorly or posteriorly, in which rapid aggravation of ophthalmic symptoms or intradural complication such as bleeding can be appeared (7). In contrast, the prognosis of patients with a TCA is generally poor. The mortality rate is almost 50% in cases left untreated (8).

Early detection and treatment of the TCA before traumatic CCF occlusion may be difficult because of

steal phenomenon of the fistula. Therefore, thorough angiographic studies are needed. They include the following parts: 1) Assessment of the morphology of the carotid bifurcation and the origin of the ICA on both sides. 2) Ipsilateral ICA and external carotid artery angiograms to identify the location of the fistula, to assess whether the CCF is of high or low flow and to determine the steal nature. The Mehringer-Hieshima maneuver (gentle ipsilateral ICA injection during manual compression of the ipsilateral carotid artery) is also included. 3) Assessment of the patency of the anterior communicating artery, which is performed by ipsilateral carotid compression of the contralateral ICA. 4) Heuber maneuver (ipsilateral carotid compression of the vertebral artery) to opacify the fistula through a patent PcomA. 5) Assessment of venous drainage correlated with patient's symptoms (9).

If a traumatic CCF combined with a TCA is revealed in the preoperative angiography, the TCA should be occluded first, and if a TCA is revealed after a CCF is treated, it should be treated as soon as possible.

Our case was a high-flow CCF caused by the rupture of aneurysm on the origin of right PcomA. It was difficult to define the true nature of the aneurysm because first angiography was performed at 8 months later after trauma. It was either chronic-flow related aneurysm or healed traumatic dissecting aneurysm. Because sufficient time passed after trauma, the wall of the aneurysm may have matured enough to permit the safe delivery of an embolic device without fear of rupture even if the aneurysmal nature was traumatic. Lempert et al. describes the safe and effective treatment of TCAs by using GDC endosaccular embolization with preservation of the parent artery (10). The aneurysm was embolized in the usual manner with

detachable coils. In our case, relatively loose packing in the cavernous sinus and compact packing in the aneurysm was obtained through the transarterial approach. Although there was a recurrent CCF due to coil modification two weeks later, final result after second embolization was good.

CONCLUSION

Traumatic CCF caused by combined intradural aneurysmal rupture is an extremely rare condition. It is a dangerous condition in which endovascular treatment should be performed immediately or even on an emergency basis. Early detection and treatment of the aneurysm before traumatic CCF occlusion may be difficult because of steal phenomenon of the fistula. Therefore, thorough angiographic studies are needed. Endovascular embolization with detachable coils can be indicated in the aneurysm with a mature wall and a clear neck.

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