Lethal Subarachnoid Hemorrhage after Successful Endovascular Embolization of a Traumatic Carotid Cavernous Fistula: a complication report

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ABSTRACT

A 35-year-old male experienced the rapid development of left pulsatile exophthalmos and chemosis in the early course after craniofacial trauma. A high-flow direct carotid-cavernous fistula was found at diagnostic carotid angiography. After successful endovascular embolization of the fistula by balloon and coils, the patient developed a diffuse subarachnoid hemorrhage leading to death. We postulate that re-damage of a previously injured intradural cerebral artery by increased cerebral blood flow after obliteration of the fistula resulted in the lethal diffuse subarachnoid hemorrhage.

CASE REPORT

A 35-year-old male was sent to emergency department after a traffic accident. He was emergently intubated and stabilized at the scene. Initial cranial computer tomography (CT) showed basal skull fractures, acute subarachnoid hemorrhage (SAH), intraventricular hemorrhage (IVH), and hydrocephalus (Fig. 1). The patient was taken to the operating room and an emergent external ventricular drainage was placed. The next day he began to develop exophthalmos and red eye, OS, but visual acuity and ocular motility could not be objectively assessed. Selective left carotid digital subtraction angiography confirmed the presence of a high-flow direct CCF, and narrowing of the peripheral branches of the left middle cerebral artery (MCA) was noted, indicating vasospasm (Fig. 2). Repeat cranial CT on day 9 after injury revealed significant shrinkage of the SAH and IVH, and there was neither deterioration of the cerebral edema nor intracranial herniation.

Endovascular treatment for the TCCF was performed under general anesthesia on day 10 of admission. Percutaneous access was obtained via the right femoral artery. A bolus of 3,000 IU of heparin was administered intravenously after placement of a 8F femoral sheath, and an
additional bolus of 1,000 IU was injected hourly. An 8F guiding catheter was placed in the left distal cervical ICA and a #9 detachable balloon (Golden Valve Balloon; Nycome Ingenor, Paris, France) threaded on a delivery catheter was coaxially introduced into the left cavernous sinus through the fistula hole. Repeated inflations of balloon and follow-up arteriograms were performed until the fistula was obliterated. However, a recurrent CCF occurred after detachment of the balloon, and progressive vasospasm of the supraclinoid ICA, M1 segment of the MCA, A1 segment of anterior cerebral artery (ACA), and peripheral branches of ipsilateral cerebral arteries was noted (Fig. 3). A Fast-Tracker-10 microcatheter (Boston Scientific/Target, Fremont, CA) was introduced coaxially, and was navigated into the left cavernous sinus via the recurrent fistula. Continuous embolization was performed with two Guglielmi detachable coils and one Matrix coil with varying length (Boston Scientific Co., USA), which were detached entirely in the cavernous sinus. A final left carotid angiogram demonstrated complete obliteration of the fistula. The left ICA was preserved, but persistent narrowing at the supraclinoid ICA, M1 segment of the MCA, A1 segment of the ACA, and peripheral branches of the cerebral arteries was still noted (Fig. 4). There was no microcatheter, microwire, or coil induced vascular perforation or dissection during procedure.

The infused heparin was reversed by suitable dose of protamine after completing the embolization. After awakening from anesthesia, he was sent back to ward in stable condition. That night he experienced loss of consciousness and urgent brain CT (Fig. 5) showed a diffuse SAH and cerebral edema. He was against advice discharge 3 days later.

**DISCUSSION**

Traumatic carotid-cavernous fistulae (TCCFs) are potential complications of craniofacial trauma, which usually produce a tear in the wall or a complete transection [2] of the cavernous ICA as a result of bony spikes from adjacent skull base fractures. Acceleration-deceleration type injuries [3] and penetrating injuries such as gun shots [4] can also result in TCCFs. True bilateral fistulae are rare [5].

The clinical symptoms of CCFs depend upon their angiarchitectural and hemodynamic characteristics [6]. CCFs mostly present with ocular symptoms. A life threatening intracranial hemorrhage occurs in only 3% of cases [7]. An untreated TCCF may results in neurologic deficiencies and life-threatening symptoms such as severe recurring epistaxis, brainstem congestion, hemiplegia, and subarachnoid or intracranial hemorrhages may be found [8]. Pre-treatment angiography should be examined for the patterns of the circle of Willis and intracranial collateral flows, available venous channels for possible endovascular navigation, and associated vascular lesions including arterial aneurysms or pseudoaneurysms, posttraumatic venous thrombosis or stricture, traumatic dissection, and underlying arterial disease [9].

The goal of treatment is occlusion of the fistula while maintaining carotid artery patency, which is important when collateral blood flow via the contralateral ICA is insufficient. Arterial detachable balloon embolization had become the treatment of choice for TCCFs; however, the detachable balloon treatment device was removed from the market in the US by the FDA in 2003. An alternative for balloon embolization are detachable platinum coils via transarterial or/and transvenous approaches, either as a primary agent or in combination with balloons [10].

Complications of endovascular embolization of a TCCF are not uncommon, and include transient cranial nerve palsies, orbital congestion, increased venous stasis, cerebral ischemia and infarction, intracranial hemorrhage, and permanent neurological damage. Once deployed, the inflated balloon or coils may cause serious neurologic damage by two different mechanisms [11, 12]. First, alternative venous drainage can increase the possibility of cerebral hemorrhage and edema and second, the embolized
Figure 2. Left carotid angiogram in anterioposterior a. and lateral b. projections on next day of admission revealed a moderate flow TCCF with draining to ophthalmic vein, inferior petrosal sinus and petrosal vein. The flow into cerebral arteries was seen. There were no evidence of pseudoaneurysm/aneurysm and venous outflow thrombosis/stricture, but vasospasm was discovered at peripheral branches of middle cerebral artery.

Figure 3. Left carotid angiogram in anterioposterior a. and lateral b. projections showed recurrent TCCF after detachment of balloon. The left ICA was preserved, but narrowing of supraclinoid ICA, M1 segment of middle cerebral artery, A1 segment of anterior cerebral artery and peripheral branches of cerebral arteries indicating vasospasms.
materials may directly compress regional structures such as cranial nerves, bony structures, and the injured vessels. Etiologies of therapy-related hemorrhage are directly iatrogenic rupture of a vessel, rupture of pre-existing associated vascular lesions, and blockage of venous outflow by migrated embolized materials [13]. Another complication, known as “normal perfusion pressure breakthrough” [14], is related to the reestablishment of normal cerebral perfusion after abrupt closure of the fistula. Rapid restoration of cerebral blood flow can result in malignant cerebral edema and hemorrhage [15].

In our case, there was no angiographic evidence of traumatic associated vascular lesions; however, the initial brain CT showed a diffuse SAH. This may have implied underlying vascular injury with extension to intradural or/and intrasubarachnoid portions. The symptoms of our patient occurred rapidly, about 1 day after head injury, implying that the injured intradural cerebral artery was still damaged. Progressive vasospasm of supraclinoid ICA, M1 segment of the MCA, A1 segment of ACA, and peripheral branches of the ipsilateral cerebral arteries was found in intraprocedural angiograms. The narrowing was more severe in the distal intracranial ICA to M1 segment of the MCA, which could indicate vasospasm or early development of dissection. Therefore, early endovascular obliteration of the acute TCCF could induce re-damage to injured vessels by a sudden increase of cerebral blood flow and pressure after acute closure of the TCCF. Endovascular

**Figure 4.** Final carotid angiogram in lateral projection revealed total occlusion of TCCF after following coils embolization. The left ICA was preserved, but vasospasm at supraclinoid ICA, proximal segments and distal branches of cerebral arteries was still seen.

**Figure 5.** Urgent brain CT on the next day after embolization therapy revealed metallic artifact from coils **a.** and new diffuse subarachnoid hemorrhage and brain swelling **b.**
procedure in an unhealed artery which may be near the bony spikes resulting from adjacent skull base fractures may be the additional factor to injury this unhealed artery, although iatrogenic perforation was not happened in our procedure. Early deflation or mild migration of the balloon after detachment occurred in our case, and lead to recurrence of the fistula. The deflated balloon and following coils were possible to development of a pseudoaneurysm, vascular dissection or laceration, or occluded venous outflow, although the post-procedural angiography could not discover any angiographic abnormality. Debrun et al. [6] reported that a venous or false aneurysm can occur if the balloon deflated too quickly. However, a TCCF-related pseudoaneurysm will not result in a diffuse acute SAH due to the extradural anatomic location of cavernous segment of internal carotid artery. Other possible etiologies of intracranial hemorrhage following cerebral revascularization of a TCCF include hypertension or the use of anticoagulants. In our case, the procedure was uncomplicated; the patient was normotensive during and after procedure and systemic heparinization was reversed at the end of the procedure. The intracranial subarachnoid occurred the day after the endovascular procedure, so these potential causes could be excluded. We postulate that re-damage of a previously injured carotid artery by reperfusion of cerebral blood flow lead to the lethal diffuse SAH.

Most direct CCFs can be treated electively. First, excluding cases with urgent condition, endovascular treatment of acute TCCFs should be delayed or staged to achieve stability and healing of injured vessels, especially the cases associated with initially diffuse traumatic subarachnoid hemorrhage. Second, parent artery occlusion of an injured carotid artery may be an alternative option when a patient passes an occlusion test in the case of a life threatening TCCF, but we should be careful about delayed ischemia due to vasospasm.

CONCLUSIONS

Endovascular embolization is a safe, useful, and first choice method for the treatment of TCCFs. The timing and treatment strategy of embolization should be carefully evaluated, especially in cases associated with an initial diffuse traumatic SAH.

REFERENCES
