Traumatic posterior communicating artery-cavernous fistula, angioarchitecture, and possible pathogenesis: a case report and literature review

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Abstract: Carotid-cavernous fistulas (CCFs) are an abnormal vascular shunt between the carotid artery and the cavernous sinus, and were traditionally classified into four subtypes based on the detailed fistulas anatomy and arterial supply. CCFs are frequently encountered in patients with traumatic skull base fractures. In this report, we present one such case caused by two head traumas. Computed tomography and digital subtraction angiography confirmed that this CCF arose from posterior communicating artery of the internal carotid artery, which is not included in the traditional Barrow et al’s categorization. The possible mechanisms include laceration of dural mater of posterior clinoid process and laceration or pseudoaneurysm formation of posterior communicating artery. This case was successfully treated with endovascular coil embolization.

Keywords: carotid-cavernous fistulas, cavernous sinus, CCF, PComA, posterior communicating artery

Introduction
Carotid-cavernous fistulas (CCFs) are one type of abnormal vascular shunt, which allow blood to flow directly or indirectly from the carotid artery to the cavernous sinus. CCFs were first classified into four subtypes by Barrow et al in 1985 based on the detailed fistulas anatomy and arterial supply.1 Type A CCF is referred to the direct high-flow shunt from the internal carotid artery (ICA) to the cavernous sinus.2 Other three subtypes involve the shunts from meningeal branches of ICA (Type B), of the external carotid artery (Type C), or of both (Type D) to the cavernous sinus. Posterior communicating artery (PComA)-cavernous fistulas are outside the Barrow’s categorization,3 and are rare complications of craniofacial trauma. In this report, we presented one such CCF intercommunicating between the left PComA and the cavernous sinus, which was caused by two head traumas. This case was diagnosed with multiple examinations and was successfully treated with endovascular coil embolization. This study was approved by the ethics committee of Shandong University, and this patient provided signed written informed consent.

Case report
A 30-year-old male complaining of progressive conjunctival congestion and strabismus on his left eye for 6 months was admitted to our hospital. This patient suffered a severe head injury 18 months ago. This injury recovered well after decompressive craniotomy and evacuation of intracranial hematoma. This patient was also given a cranioplasty for his skull defect 3 months after his injury. However, 1 year after the head injury, he had another head injury and got expectant treatment at this time.
On admission to our hospital, physical examinations revealed chemosis, orbital bruits, and incomplete abducens dysfunction on his left eye. No visual disturbance was seen. Computed tomography and computed tomography angiography scanning revealed a high-density mass in the left cavernous sinus, which was obviously enhanced after injection of contrast material (Figure 1). Cerebral digital subtraction angiography, three-dimensional angiography, and superselective angiography via a microcatheter clarified the angiography architecture of this CCF (Figure 2). The three-dimensional angiography revealed that the supplying artery had an arterial lumen of 2 mm and originated from the C1 segment of ICA, which was identified as PComA due to its origination from the posterior-medial wall of ICA and 2 mm internal diameter.

This patient was treated with coils via transarterial approach. A 6 F Envoy (Codman, Raynham, MA, USA) guiding catheter was placed in the left cervical ICA, and then an Echalon 10 microcatheter (EV3, Plymouth, MN, USA) with the Silverspeed 0.014 microwire (EV3) was superselectively introduced into the ectasia of the cavernous fistula through the ICA and the PComA. Detachable platinum coils (Axium, EV3; 20 mm × 50 cm, 20 mm × 50 cm, 20 mm × 50 cm, 20 mm × 50 cm, 18 mm × 40 cm, 18 mm × 40 cm, 16 mm × 40 cm, 16 mm × 40 cm) were inserted into the pouch of the cavernous sinus through this microcatheter. After compact embolization of the cavernous sinus, Axium coils (2 mm × 8 cm, 2 mm × 6 cm, 1.5 mm × 2 cm) were delivered and detached to occlude the PComA. Angiography was performed subsequent to embolization and showed complete occlusion of CCF (Figure 3). Orbital bruits immediately disappeared after operation. Conjunctival congestion and strabismus also disappeared 2 days later.

**Discussion**

Traumatic CCFs most commonly occur as a result of head injury-associated skull base bone fracture or puncture injury. Traumatic CCFs most commonly occur as a result of head injury-associated skull base bone fracture or puncture injury. The damage of the posterior part of the sphenoid bone, the internal part of orbit wall, the ethmoid sinus, and the ethmoid bone is the key point of traumatic CCF formation. Dural mater covers the superior, lateral, and medial surfaces
of the cavernous sinus, whereas the inferior surface is formed by the middle cranial fossa. Umansky et al. divided the roof of the cavernous sinus into three regions: the oculomotor trigone, the carotid trigone, and the clinoid space. The cranial base fracture may lacerate the dural mater constituting of the cavernous sinus; also, sharp bone fragment can directly penetrate the ICA and the meningeal artery.

CCFs are abnormal vascular shunt between carotid artery and the cavernous sinus. Based on the different supplying arteries, Barrow et al. categorized CCFs into four subtypes. While Type A CCF is a direct communication between the cavernous sinus and ICA, the other three subtypes (Types B, C, and D) are indirect fistulas, in which dural arteriovenous fistulas are fed by the dural branches of ICA, external carotid artery, or both. Traumatic CCF with the supplying artery of PComA or intradural ICA has not been reported. Anatomically, PComA does not contact with the dural mater of the cavernous sinus. Usually, PComA runs adjacent to the dural mater of the posterior clinoid process; sometimes, it is fixed to these structures by arachnoid membrane. The arachnoid membrane immobilizes PComA to the dural mater of the skull base, especially the dural mater covering the posterior process and the cavernous sinus. PComA also ran in the bony superfi ssure on the surface of posterior process in rare case. In oculomotor triangle, PComA may cross between the supratentorial and the infratentorial spaces or between the anterior and posterior petroclinoid folds. Theoretically, fracture of the posterior process may result in laceration of dural mater of this area. If PComA or PComA aneurysm ruptures simultaneously, a shunt forms between the PComA and the cavernous sinus. In the present study, this patient suffered two severe head injuries and craniotomy. During the first head trauma or surgery, the PComA may be damaged, and a pseudoaneurysm may form and adhere to the posterior clinoid process. The shunt may not appear in the first head trauma due to the local adhesion and the capsulation of hematoma and other tissues. In the second head injury, the damaged dural mater may be lacerated again, and then the shunt may form between the PComA pseudoaneurysm and the cavernous sinus. The PComA adhered to the dural mater of the posterior process can also be lacerated, and the laceration may form a communication with the cavernous sinus.

Figure 2. DSA images before treatment.
Notes: (A) Frontal and (B) lateral angiographic projection of left ICA demonstrates CCF. (C) Microcatheter enters the pouch of the cavernous sinus through the supraclinoid ICA and the PComA. (D) Three-dimensional angiography demonstrates the relationship between the PComA and CCF.
Abbreviations: DSA, digital subtraction angiography; ICA, internal carotid artery; CCF, carotid-cavernous fistula; PComA, posterior communicating artery.
The advances of endovascular technology such as detachable balloon, detachable platinum coils, and other embolic materials have provided many options for the treatment of the cavernous fistula. Traditional surgery has been rarely used in direct CCFs due to its potential risk to the brain. As an alternate, the endovascular approach has become the major option for CCFs’ treatment. In this case, we occluded the PComA using detachable platinum coil and obtained satisfactory result. The detachable platinum coil has some advantages such as reliable and controlled deployment. Even if the placement of the coil is not satisfactory, it can be replaced or removed easily and safely. Covered stents and flow-diverting stents are being used as another alternative methods for CCFs treatment. If there is no artificial cranio-plasty history, this patient can also be treated by clipping the PComA via craniotomy.

**Conclusion**

This case is a new subtype of traumatic CCF, which is beyond the traditional Barrow’s categorization. The supplying artery is PComA. The possible mechanisms of CCF formation include two aspects: laceration of dural mater of the posterior clinoid process and laceration or pseudoaneurysm formation of PComA during head trauma. Endovascular coil embolization is an effective treatment for this selected patient.

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**Disclosure**

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