Diagnosis and Treatment of Traumatic Internal Carotid Artery Pseudoaneurysm Primarily Manifested by Repeated Epistaxis

[Introduction]

Traumatic internal carotid artery pseudoaneurysm (TICAP) is a rare complication of vascular injury after craniocerebral injury, and accounts for approximately 0.5% of intracranial aneurysms; TICAP frequently occurs at the internal carotid cavernous sinus and petrous bone segments and takes cranial base fracture as the main cause (8,14). Patients with TICAP present repeated epistaxis that is difficult to control, a condition that often leads to hemorrhagic shock or asphyxia and thus seriously threatens life. Endovascular interventional therapy is the main treatment method for TICAP nowadays, which involves the use of detachable balloons, microcoils, endovascular stents, and so on (13,19,20). To explore the diagnosis and treatment of TICAP primarily manifested by repeated epistaxis, we retrospectively studied 31 cases with TICAP, who were cared for over a 12-year period from 2000 to 2012.

[Data and Methods]

In this study, the diagnosis and treatment of TICAP for 31 patients between 2000 and 2012 were retrospectively analyzed.
**General Data**
A total of 31 patients with TICAP, including 25 males and 6 females, were enrolled. Their ages ranged from 15 years to 52 years with an average of 37 years. This study was conducted in accordance with the declaration of Helsinki. This study was conducted with approval from the Ethics Committee of Wuhan General Hospital, Guangzhou Command of PLA. Written informed consent was obtained from all participants.

**Clinical Manifestations**
All the patients had a definite history of craniocerebral injury: 24 had traffic accident-caused trauma and 7 had fall from height-caused trauma. They all presented with delayed repeated epistaxis clinically: Massive epistaxis occurred 3 days to 3 months after trauma and varied from 2 times to 6 times with a varying volume between 300 ml and 1000 ml per time. All epistaxis was characterized by spurting. 19 patients were subjected to hemorrhagic shock due to repeated bleeding. All the patients were referred from a local hospital after receiving anterior and posterior nasal packing and blood transfusion. Seven patients also had intracranial murmur, blepharoedema, and ectropion. Three had monocular deprivation as a consequence of cranial base fracture-caused optic nerve injury.

**Imaging Examination**
Cranial computed tomography showed cranial base fracture in all the patients, as well as hydrops and liquid-air planes in paranasal sinuses. Digital subtraction angiography (DSA) was performed: The patients all had a pseudoaneurysm at the internal carotid artery siphon and cavernous sinus; the pseudoaneurysms were irregular in shape with a diameter of 4–20 mm; 7 patients also had internal carotid cavernous fistula; and 18 had pseudoaneurysm protruding into the sphenoid sinus according to three-dimensional (3-D) DSA image reconstruction.

**Treatment**
All the patients received endovascular interventional therapy after DSA under local anaesthesia. Intraoperative burst of nasal hemorrhage occurred in one patient. For this patient, local anaesthesia was converted to a general anaesthesia after nasal packing, followed by an endovascular interventional therapy. Operation was performed 3 days to 1 week after the last time of bleeding. Internal carotid artery occlusion on the affected side and controlled hypotension were performed. When the mean arterial pressure was controlled between 60 mmHg and 70 mmHg, consciousness, speech, vision, as well as limb muscle strength and activities on the healthy side were observed for 30 min. Femoral arteriopuncture on the opposite side and internal carotid and vertebral arteriography on the healthy side were performed simultaneously to understand anterior and posterior communicating artery collateral circulations. Embolization with detachable balloons for a direct occlusion of the parent artery was adopted for 20 patients (a typical case is shown in Figure 1A-D). For the remaining 11 patients who received treatment after 2008, covered stent (Willis stents; Shanghai MicroPort Medical Co., Ltd., China) implantation was adopted to cover the parent arterial tear; meanwhile, the internal carotid artery was maintained unobstructed (a typical case is shown in Figure 2A-D).

**RESULTS**
The interventional therapies for all the patients in this study were successful: Postoperative immediate arteriography did not show redeveloped aneurysm; the parent artery was occluded in 20 patients and maintained unobstructed in 11. No death occurred. No epistaxis recurred. All the patients were discharged from the hospital 2 weeks after operation. They did not present neurological deficits. 18 patients were followed up for 6 months to 2 years with an average of 11 months. No repeated epistaxis or neurological deficit occurred.

**DISCUSSION**
TICAP refers to a medical condition formed through the following processes: After a trauma ruptures the internal carotid artery wall, a small hematoma is formed at the tear; later, this hematoma is enclosed by its surrounding connective tissues to form a temporary tubal wall; then, this tubal wall develops into an aneurysm which continuously bulges because of the impact of high pressure and blood flow in the artery. Since the wall of TICAP does not contain arterial wall components in nature, it may rupture when there is a sudden arterial pressure increase or thrombotic shedding, thereby leading to massive hemorrhage. If the tear is small, a timely-formed thrombus can seal it and stop hemorrhage temporarily; if the tear is large or rupture occurs repeatedly so that a collapsing large tear is formed, however, life-threatening massive hemorrhage can be resulted in (16,21). TICAP, principally characterized by repeated epistaxis, commonly occurs at the internal carotid siphon and cavernous sinus. The main cause for TICAP occurrence is as follows. After craniocerebral injury causes cranial base fracture, the bone fragment of the lateral wall of the sphenoid sinus can directly damage the internal carotid artery tightly close to the sphenoid sinus, thereby resulting in hematoma in the sinus; the hematoma gradually develops into a TICAP with the continuous impact of the arterial blood flow. TICAP can also be caused by injured internal carotid artery at the cavernous sinus in trans-nose-and-sphenoid sinus operation (4,11,12). Yet, in this study, all TICAPs were caused by traffic accidents and falling from height.

Delayed repeated epistaxis after head and facial trauma is the most distinctive manifestation of TICAP: Each time the hemorrhage is violent and can reach hundreds of or even more than one thousand milliliters. Although hemorrhage stops after blood pressure decreases or shock occurs, patients may die of hemorrhagic shock or asphyxia due to airway blockage caused by coagulated blood. DSA is the gold standard for TICAP diagnosis. This technique can clearly display the site, size, and morphology of the lesion, which
Figure 1: A typical case: A 43-year-old male suffered from repeated epistaxis three times after 1 month of a traffic accident-caused injury, and he also had left exophthalmos. A, B) Front and lateral DSA shows pseudoaneurysm concurrent with cavernous fistula at the internal carotid arterial siphon and a thickened vein above the left eye; C) Two balloons occlude the tear and the internal carotid artery; and D) Opposite internal carotid arteriography shows a good compensatory function of the Willis circle collateral circulation after the occlusion.

Figure 2: A typical case: A 38-year-old male suffered from repeated epistaxis three times after 2 months of a traffic accident-caused injury. A) Front DSA shows an irregularly-morphological pseudoaneurysm at the internal carotid arterial siphon but without an apparent neck; B) 3-D DSA reconstruction more clearly displays the vascular tear and the aneurysm; and C, D) Front and lateral DSA after the stenting shows that the blood flow in the parent artery is unobstructed, no aneurysm is developed, and the stent has good adherence.
is manifested by an irregularly-mass-like contrast agent concentration area protruding into the sphenoid or ethmoid sinus at the internal carotid artery cavernous sinus segment, with a distinct boundary. Cranial base fracture, epistaxis, and monocular deprivation were previously considered as the “three primary manifestations” of TICAP (5,9,18). Recent literature reviews, however, have revealed that monocular deprivation is the consequence of cranial base fracture-caused optic injury and that most patients with TICAP do not have monocular deprivation. Therefore, monocular deprivation is no longer considered as a necessary diagnostic condition of TICAP nowadays. In this study, only 3 out of the 31 cases were complicated with monocular deprivation. Based on literatures as well as the retrospective analysis of 31 patients in this study, if regular anterior and posterior nasal packing cannot achieve a satisfactory effect on repeated massive nasal hemorrhage after trauma, DSA should be performed as early as possible whereby to determine whether TICAP exists. The diagnostic criteria of TICAP include: 1) There is a definite history of head injury; 2) Delayed and serious repeated life-threatening epistaxis occurs; and 3) DSA shows an irregular-mass-like contrast agent concentration area protruding into the sphenoid or ethmoid sinus at the internal carotid cavernous sinus segment with a distinct boundary.

Once TICAP is diagnosed, treatment should be given as early as possible. Traditional approaches to TICAP include surgery, ligation of the common carotid artery or the internal carotid artery, as well as intra- and extra-cranial combined ligation of the internal carotid artery; however, all these methods have the drawbacks of unsatisfactory effect, serious surgical trauma, multiple complications, and so on (6,10). With development and gradual perfection in recent years, endovascular interventional therapy has been gradually taking the place of the traditional treatment methods for TICAP by virtue of its microtrauma and high recovery rate. This technique is the treatment of choice for TICAP; balloons and covered stents are frequently-used embolising materials (1-3,13,19,20).

Because pseudoaneurysm does not contain normal vascular wall components, balloons can pose a risk of breaking pseudoaneurysm after entering and dilating fully, thus leading to fatal massive hemorrhage. Therefore, intervention by occluding the internal carotid artery with a balloon at the pseudoaneurysm tear site should be adopted. In addition, considering that a prerequisite for internal carotid artery occlusion with balloons is that the circle of Willis must have good lateral circulation compensation function so that the patient can tolerate the occlusion, a temporary balloon occlusion test must be performed before internal carotid artery occlusion. Specifically, a detachable balloon is used to occlude the internal carotid artery on the affected side when the patient is in a conscious state, and controlled hypotension is performed in the meantime to decrease his mean arterial pressure to 60–70 mmHg. The patient’s consciousness, speech, vision, as well as his limb muscle strength and activities on the healthy side are observed for 30 min. Femoral arteriopuncture on the opposite side and internal carotid artery and vertebral arteriography on the healthy side are performed simultaneously to understand the anterior and posterior communicating artery collateral circulations. If the patient can tolerate the occlusion and the circle of Willis has a good compensation function, internal carotid artery occlusion can be performed immediately. However, if the patient cannot tolerate the test, carotid arterial compression training should be taken: Generally, the carotid artery on the affected side is compressed 3–5 times per day (30 min each) for one week. After that, balloon embolization is performed. After embolization, patient’s nervous system and vital signs should be closely observed and a high blood pressure should be maintained by administering blood volume-enlarging drugs to improve the blood supply for local brain tissues in case of brain ischemia. In this study, 20 of the 31 patients were subjected to direct parent arterial occlusion using embolization with detachable balloons. All of them were strictly tested before operation using the temporary balloon occlusion method. After operation, they all recovered and no brain ischemia occurred.

Covered stent implantation, as a fruit of the development of materials science in recent years, is a newly-emerged interventional therapy for TICAP. This technique makes use of the biomembrane on the stent surface to directly cover the distal and proximal ends of the aneurismal neck to isolate aneurysm and reconstruct an arterial lumen, thereby restoring the hemodynamics in the diseased region and decreasing intra-aneurysmal pressure; as time prolongs, intra-aneurysm thrombosis occurs and the aneurysm then occludes automatically (7,15,17). In this study, Willis stent implantation was performed for 11 patients who received treatment after 2008 to cover the rupture site of the parent artery. Good effect was achieved: Postoperative immediate DSA showed that aneurysm did not redevelop and that blood flow in the parent artery as well as in the surrounding branches was unobstructed. Covered stent implantation is an interventional treatment method most adaptive to human physiological characteristics, and it does not change blood flow circulation paths. However, some points should be paid great attention to after stenting: 1) As covered stents have poor compliance, they are not feasible for patients with an apparently-circuitous diseased blood vessel; 2) Covered stents should be ensured to completely cover the distal and proximal segments of the aneurismal neck, and meanwhile the openings of the important branch blood vessels of the parent artery should be avoided; and 3) Antiplatelet drugs such as aspirin and the Polivy should be administered during and after operation in case of vascular occlusion due to thrombosis in the stent.

**CONCLUSION**

To summarize, patients with posttraumatic repeated massive epistaxis may have TICAP. For these patients, DSA should be performed as early as possible. Once TICAP is confirmed,
endovascular interventional embolization should be performed. Compared with internal carotid artery occlusion with balloons, covered stenting does not change blood flow circulation paths and maintains the smoothness of the affected internal carotid artery. Thus, it may be more valuable than internal carotid artery occlusion with balloons in treating TICAP.

REFERENCES