Traumatic Aneurysm of the Basilar Artery

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Summary: We report a case in which a "false" aneurysm of the basilar artery developed after an assault on a patient resulting in head injuries. Diagnostic imaging and endovascular treatment are described. Formation mechanisms of traumatic intracranial aneurysms are discussed, and the literature is reviewed.

Index terms: Aneurysm, intracranial; Head, injuries; Interventional neuroradiology

Traumatic lesions of the basilar artery are a very rare and often fatal. Loop et al (1) describe a traumatic occlusion of the basilar artery caused by a clivus fracture. Bank et al (2) described a case of traumatic aneurysm of the basilar artery, and their review of intracranial traumatic aneurysms summarized only 41 cases in the world literature. Feldges et al (3) describe an aneurysm of the basilar artery, but the diagnosis was made by necropsy. We report a case of traumatic aneurysm of the basilar artery—a young man with a clivus fracture caused by assault. The patient was treated by endovascular occlusion of the pseudoaneurysm and basilar artery using a Guglielmi detachable coil system (Target Therapeutics, Fremont, Calif).

Case Report

A 27-year-old man was admitted to our hospital with head injuries resulting from assault. It is not known if consciousness was lost. He presented with multiple skin erosions and left eyelid hematoma. He was awake but with a tendency to fall asleep, and his language was repetitive and inappropriate. Severe left-side hemiparesis with brachial predominancy was detected. A computed tomographic scan showed a diffuse subarachnoid hemorrhage with ambiens cistern predominance, occipital horn hemorrhage, ventricular dilation, left sphenoidal sinus hemorrhage, and air bubbles within the right ambien cistern. Thin computed tomographic sections with bone reconstruction allowed identification of multiple left ethmoidosphenoidal sinus fractures and a clivus fracture (Fig 1A).

The neurologic defects stabilized, and over the next 3 days the patient returned to normal consciousness. On the third day of admission, angiography showed an irregular sesil aneurysm at the point between the upper third and lower two thirds of the basilar artery proximal to the cerebellar arteries. The aneurysm was situated at the anterolateral right wall of the basilar artery. The parent vessel had several adjacent irregularities (Fig 1B). The clivus in front of the aneurysm had a fracture that was projecting backward against the basilar artery. Computed tomographic control at this time showed a hypodense pontine lesion that suggested contusion or traumatic infarction. No clinical changes were noted, and surgical clipping, vertebral ligature, and endovascular aneurysm occlusion were considered. It was decided to follow the patient clinically and angiographically and, if necessary, perform an endovascular treatment.

A first angiographic control on the 10th day showed no changes in form and size of the aneurysm, and the irregularity of the basilar artery was similar to the initial study. The patient started rehabilitation treatment. A second angiographic control, 3 weeks after admittance, showed no aneurysm growth, and the basilar artery appearance was normal. An attempt to embolize the aneurysm was made, but its size was insufficient to accept our smallest coil, which was retrieved into the microcatheter. The patient was discharged with a left hemiparesis. Control angiograms were made every 2 months.

The aneurysm grew, and by the fourth month its size was sufficient for endovascular treatment. At this time, catheterization and occlusion with the Guglielmi detachable coil system was carried out and the aneurysm was occluded. Three Guglielmi detachable coils ($6 \times 20 \times 10$) were detached into the aneurysm without difficulties or complications. Four months later, angiography showed the aneurysm to be occluded and the basilar artery to be intact. The mild left hemiparesis was stabilized.

Fourteen months after the endosacular occlusion, the aneurysm had grown and a "water hammer effect" was noted (Fig 1C). We decided to occlude the pseudoaneurysm and the parent vessel. After a test occlusion of the basilar artery for 30 minutes, three Guglielmi detachable
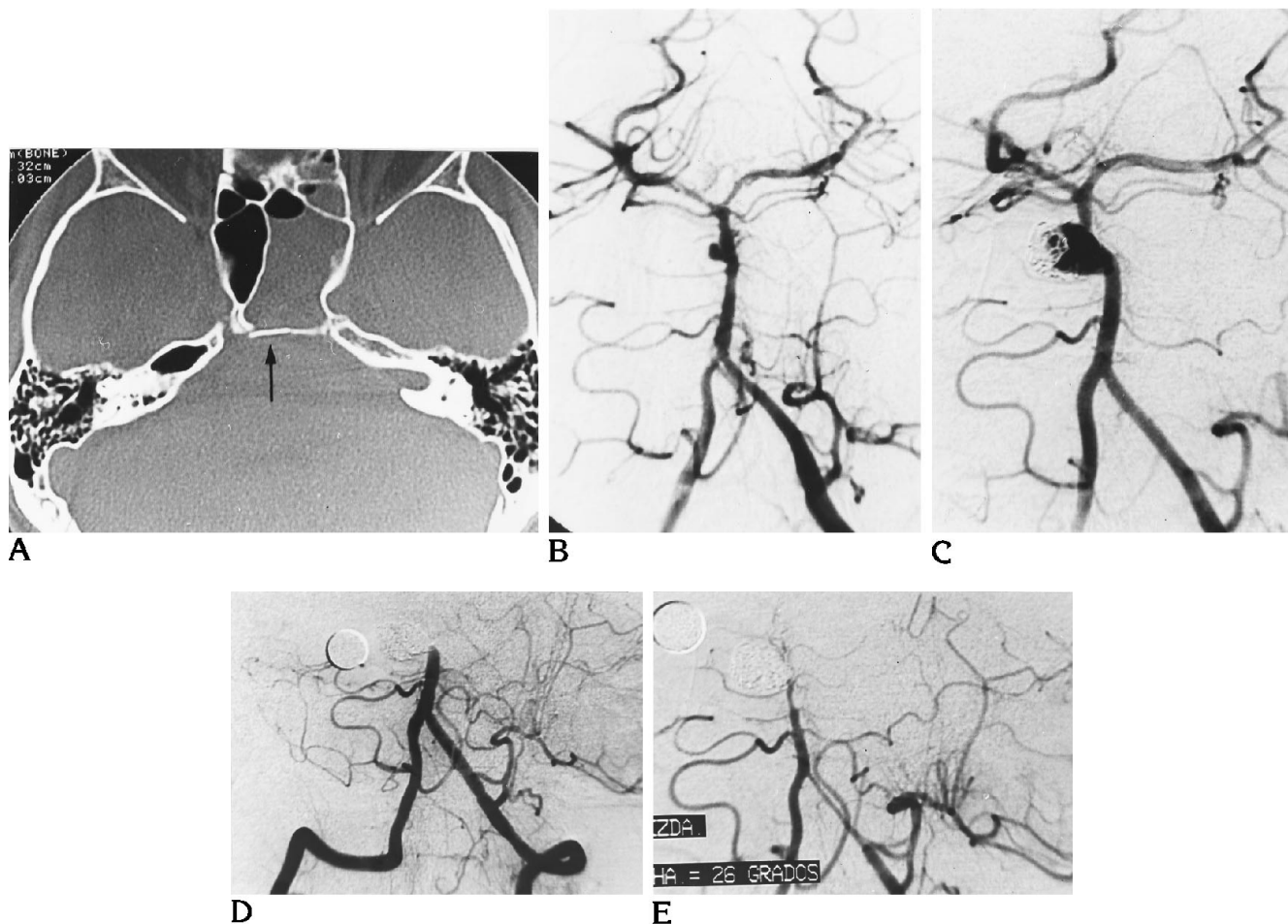
Fig 1. *A*, Computed tomographic 1-mm axial section: clivus fracture and posterior displacement of fracture (*arrow*).

B, Posteroanterior left vertebral arteriogram: 3-mm-diameter aneurysm and irregular walls of the basilar artery.

C, Posteroanterior left vertebral arteriogram, 14 months after Guglielmi detachable coil treatment. The aneurysm is not occluded; the basilar artery is preserved. Coils are compressed to the top of the pseudoaneurysm.

D, Posteroanterior left vertebral arteriogram after complete occlusion of the aneurysm and basilar artery.

E, Posteroanterior left vertebral arteriogram, 6 months after total occlusion: complete occlusion of the pseudoaneurysm and parent vessel. Reduction in size of both vertebral arteries and development of the circumferential arteries of the basilar artery is noted.

coils (8 × 20 × 10, 6 × 20 × 10, and 5 × 15 × 10) were introduced, causing total occlusion of the basilar artery at the base of the pseudoaneurysm (Fig 1D). The basilar artery distal to the aneurysm was perfused by the carotid artery. No clinical changes were detected.

Six months later, the angiographic control showed permanent occlusion of the basilar artery and complete exclusion of the aneurysm (Fig 1E). The hemiparesis is stable, and the patient leads a normal life and can work.

Discussion

Injury of intracranial vessels is a rare complication of intracranial trauma. The majority of cases affect the carotid artery, and the most frequent complication is carotid-cavernous fistulas. The location of arterial damage depends on the proximity of the skull fracture (2, 3), and the type of lesion depends on the extent of wall damage (4): the rupture of all three layers accompanied by an organized hematoma results in a false aneurysm. If the adventitia is preserved, the traumatic result is a true aneurysm (3, 5).

Injuries of the basilar artery (1–3, 6) are exceptional because of its protected location. Occlusion (1, 6) and false (2, 3) and/or dissecting (4) aneurysms are the anatomic lesions reported. Traumatic wall rupture causes subarachnoid hemmorhage, subarachnoid clots, and parenchimal hemorrhage or ischemia. Morbility and mortality are high in basilar artery lesions. False aneurysms grow faster than true

aneurysms, and their risk of rebleeding is higher.

The diagnosis of traumatic aneurysm can be definitive if a previous study demonstrates no lesion. Previous trauma is not a sufficient reason to consider it as the indisputable cause of the pseudoaneurysm. In practice the diagnosis is based on additional information: history of the trauma, vessel location, and histopathologic findings (5). Our case had: (*a*) a nontypical location (anterior and right lateral wall of the basilar artery without nearby emergent branches or bifurcations); (*b*) irregularity of the basilar artery wall; and (*c*) a clivus fracture close to the aneurysm.

Clinical features were explained by occlusion of perforating arteries, probably because of direct trauma or indirect (subarachnoid hemmorhage and vasospasm) effects. Because spontaneous thrombosis of traumatic aneurysms can occur, follow-up is usually suggested. Progressive growth of the aneurysm in staged angiograms determined our therapeutic decision in this case.

Occlusion of the parent vessel may be needed to treat intracranial pseudoaneurysms (7). Endovascular treatment of posterior circulation aneurysms has been done using balloons (8) and coils (9). Today, the Guglielmi detachable coil system is most frequently used to treat intracranial aneurysms. Conservation of the parent vessel in pseudoaneurysms is often not possible because of anatomic characteristics. We attempted to conserve the basilar artery in this case, but the growth after aneurysmal sac occlusion required us to occlude the basilar artery.

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