Traumatic Carotid-Cavernous Fistula with Fatal Epistaxis
Report of a Case*

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Damage to the intracavernous segment of the internal carotid artery may be caused by penetrating objects,4 or the vessel wall may be torn indirectly by basilar skull fractures. A common consequence of the arterial injury is the immediate or delayed development of a carotid-cavernous fistula. Bleeding into the air-containing sphenoid sinus occurs less frequently but this massive epistaxis is an immediate threat to life in contrast to the more benign behavior of carotid-cavernous fistulae.7 The purpose of this paper is to report the occurrence of a traumatic carotid-cavernous fistula with massive epistaxis, a coincidence reported in only one previous case.1

Case Report

A 31-year-old male was admitted to the Veterans Administration Hospital on February 13, 1964, following a self-inflicted gunshot wound of the head. Eight hours earlier he had been found lying in a pool of blood with a .22 caliber bullet-wound beneath his chin. A tracheostomy was performed at a local hospital and he was transferred for definitive treatment.

Examination. The patient was comatose at the time of admission. Vital signs included a pulse of 120; blood pressure, 80/40; and temperature, 98°. There was brisk arterial bleeding from the nose and mouth. The wound of entry was beneath the chin in the midline. The bullet was palpated beneath the intact scalp overlying the left parietal bone. The left pupil was fully dilated and did not react to light. The left eyeball was proposed and pulsed faintly. A loud systolic bruit was heard over the left eye. The hematocrit was 26 per cent.

Plain radiographs of the skull indicated the course of the missile (Fig. 1). An air-fluid level was seen within the sphenoid sinus, and the cisterna interpeduncularis contained air.

Operation. The patient was transported directly to the operating room where a cuffed endotracheal tube was passed through the tracheostomy after fresh bloodclots had been removed from the trachea. Bright red blood was seen coming from the nasopharynx when the mouth and pharynx were inspected with a laryngoscope.

The wound of the exit was debrided through a parietal craniectomy. The craniectomy was extended inferiorly because of persistent extradural bleeding from a lacerated branch of the middle meningeal artery beneath a low parietal fracture. This vessel was coagulated and the dural defect was repaired with temporalis fascia.

Brisk bleeding from the nasopharynx continued throughout the operative procedure. The patient's blood pressure was maintained above 80 mm. Hg by the rapid administration of 10 units of whole blood. A left carotid angiogram, performed preliminary to ligation of the internal carotid artery, demonstrated total diversion of carotid flow into the left cavernous sinus (Fig. 2).

As the left internal carotid artery was exposed in the neck, the blood pressure and pulse became unobtainable and cardiac action ceased. Twelve units of blood had been administered at the time of death. Permission for autopsy was not granted.

Discussion

Massive epistaxis may originate from the cavernous segment of the internal carotid artery because of the anatomic proximity of this vessel to the sphenoid sinus. Communications between a carotid artery and the sphenoid sinus may be classified as follows:

1. Direct: communication between a ruptured aneurysm and the sphenoid sinus:
   a. traumatic (or false) aneurysm, or
   b. non-traumatic aneurysm.

2. Indirect: communication between a carotid-cavernous fistula and the sphenoid sinus.

The initial formation of an aneurysm, either true or false, is essential to the development of a

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direct communication between the carotid artery and the sphenoid sinus. The artery has adventitial attachments to the dorsum sellae and to the anterior clinoid process but at no point does the artery normally lie in direct apposition to the sphenoid bone. An expanding aneurysm may erode through the thin bony wall of the sphenoid sinus or it may rupture through a traumatic defect in the roof of the sphenoid sinus giving rise to massive epistaxis.

An indirect communication between the carotid artery and the sphenoid sinus occurs when a carotid-cavernous fistula gains access to the sphenoid sinus through a defect in the structures separating the cavernous and sphenoid sinuses. A traumatic tear in the carotid artery results in a carotid-cavernous fistula unless the cavernous sinus becomes thrombosed since the normal carotid artery is completely surrounded by an intercommunicating network of venous plexuses within the cavernous sinus.

Traumatic carotid-cavernous fistulas may appear immediately or manifestations of the fistulous communication may be delayed for days or weeks. In the first instance an opening has usually been produced in the arterial wall at the time of injury. The late appearance of a fistula may be explained either by rupture of a true aneurysm originating from an area weakened by the initial injury or by the development and eventual rupture of a false aneurysm. In this case rupture of a pre-existing congenital aneurysm as a result of trauma seems unlikely since there is neither pathological nor clinical evidence for such an occurrence. The early appearance of the fistula is consistent with a perforation in the wall of the carotid artery caused by the missile, although the possibility of a traumatic rent produced indirectly by a basilar fracture cannot be dismissed completely.

Maurer et al., reporting the 9th case of massive epistaxis following closed head injury, concluded that a direct communication between a traumatic carotid aneurysm and the sphenoid sinus existed in every case. Hemorrhage was delayed up to 3 months in 5 instances and none developed a carotid-cavernous fistula. McCormick and Beals reported the 20th case in which there was acceptable radiological, surgical, or autopsy demonstration of a nontraumatic carotid aneurysm as the cause of severe epistaxis by this mechanism.

Fatal hemorrhage is reported to occur in 3 per cent of patients with carotid-cavernous fistulas following rupture either into the intracranial cavity or into the sphenoid sinus. The only documented case of fatal bleeding into the sphenoid sinus from a traumatic carotid-cavernous fistula was reported by Cairns (Case VIII). This patient sustained multiple fractures of the skull and within half an hour exophthalmos was noted. Initially severe, epistaxis ceased spontaneously.

A carotid-cavernous fistula was treated by ligation of the internal carotid artery 7 weeks following the injury. Fatal epistaxis occurred 40 days later. Autopsy revealed an "aneurysmal false sac" projecting into the sphenoid sinus. The carotid artery opened into the false sac and into the cavernous sinus through 2 small holes in the arterial wall.

Clinical and radiographic findings in the present case established the diagnosis of traumatic carotid-cavernous fistula. The course of the missile through the cavernous sinus is consistent with