Severe Epistaxis Caused by Ruptured Aneurysm of the Internal Carotid Artery

WILLIAM F. MCCORMICK, M.D., AND JOE D. BEALS, M.D.
Departments of Pathology and Surgery (Neurosurgery), University of Tennessee, and the City of Memphis Hospitals, Memphis, Tennessee

Fatal epistaxis caused by rupture of an aneurysm of the internal carotid artery into the paranasal sinuses is a very rare and dramatic condition. Because of its rarity we have undertaken a review of the previously published cases, to which we add 1 of our own.

The first well-documented case of an aneurysm of the internal carotid artery with severe epistaxis was reported by Delens in 1870. His patient, a young man who sustained a penetrating injury of the eye, died as a result of the ruptured aneurysm and autopsy was performed. During the 93 years following that report only 18 additional proven examples have been published. The man whose findings are summarized below is the 20th “proven” case known to us, and only the 9th in which an autopsy had been performed. As he illustrates so well the natural history of this condition, his clinical and autopsy findings are given in detail.

Case Report

Clinical Summary. On March 25, 1961, L. M., a 70-year-old Negro man, fell through a second-story window to the sidewalk 14 feet below. Further details regarding this part of the history are unknown, but he was taken to the emergency room of the City of Memphis Hospitals within a few hours following the fall. He complained of severe pain upon movement of his left hip.

Examination upon admission revealed a stuporous man who responded slowly to commands. The left pupil was twice the size of the right, and neither reacted to light. Marked right central facial weakness and right upper monoparesis were present. There was marked edema of the left fronto-temporal and periorbital areas. Roentgenograms revealed several linear fractures of the skull in the left frontal region, a fracture of the neck of the left femur, and fractures of the 10th rib on the left. It was thought that the patient had either an epidural or an acute subdural hematoma.

Operation was performed immediately. Through a left temporal burr hole a subdural hygroma was found and drained of 40 cc. of pink fluid. The brain was then pulsatile.

As his sensorium cleared he complained of being unable to see with his left eye. His left pupil remained larger than the right and was nonreactive to direct light but would react to consensual light stimulation. He was mildly hypertensive, with a blood pressure of 160/100. A test for venereal disease was nonreactive. He made an otherwise uneventful recovery following open reduction of the fracture of the left hip and was discharged home to be followed in the out-patient department.

On Oct. 18, 1961, the patient was seen in the Neurosurgery Clinic 1 week after he had been in the emergency room following a “seizure.” He was placed on Dilantin. He was totally blind in his left eye and had moderate weakness of the left lateral rectus muscle.

On Nov. 22, 1961 he returned to the out-patient department with the chief complaint of a “funny feeling” in his left frontotemporal region, described at times as a “constant roaring noise” which was relieved only by pressing on the left eyeball. There had been no improvement in the vision of the left eye. He reported having had 3 periods of syncope in the 3 weeks prior to this visit.

Examination revealed paresis of the left lateral rectus muscle; dilated, fixed left pupil; 5 mm. proptosis of the left eye; a pale left optic disc; hypesthesis over the areas of the 1st and 2nd divisions of the left Vth cranial nerve; and a continuous, loud bruit in the left frontotemporal areas, maximal over the globe. The clinical impression at this time was an arteriovenous fistula, post-traumatic, involving the left internal carotid artery and the cavernous sinus.

He was admitted to the hospital on that same day. He had a slight weakness of grip in his right hand. Bilateral carotid arteriograms revealed an aneurysmal deformity of the left carotid siphon, not typical of an arteriovenous fistula (Fig. 1). During angiography the patient stated that he could no longer hear the “roaring sound” — neither could it be heard by the examiners. In

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FIG. 1. (Left) Left lateral carotid angiogram demonstrating large aneurysm arising from cavernous portion of internal carotid artery. There is no evidence of an arteriovenous fistula. (Right) Postero-anterior projection demonstrating large, saccular aneurysm.

view of the disappearance of the bruit and the absence of an arteriovenous fistula, the patient was discharged home to be followed in the out-patient department.

He did well until Dec. 18, 1961 when he was seen for recurrent "nose bleeds" of 1 week's duration. His blood pressure was 150/110. He was treated with vaseline gauze packs and followed in the out-patient department. The point of bleeding was never identified.

On Jan. 1, 1962 he had an episode of severe epistaxis. His blood pressure was 150/90 and his hematocrit was 24 per cent. The site of bleeding from the nose was not identified, but roentgenograms of the paranasal sinus revealed a membranous thickening in the right maxillary antrum. He was transfused with whole blood.

Bilateral intranasal antrostomy was performed but the sinuses appeared free of any infection. Again, the site of bleeding was not identified.

Postoperative course was uneventful except for occasional episodes of mild hemoptysis, which suggested recurrent episodes of bleeding from the nasopharynx. The patient was discharged to be followed in the out-patient department.

On Jan. 19, 1962 he reported having had 1 episode of severe epistaxis 1 week after the antrostomy. He had also noted occasional traces of blood on his handkerchief after blowing his nose.

On Feb. 1, 1962 he went into shock and was seen 7 hours after the onset of massive epistaxis, which reportedly had lasted for 2 hours. He had lost consciousness 5 min. after the onset of the nasal hemorrhage. Blood pressure was 80/60 and hematocrit was 22 per cent. He responded to the administration of 1000 ml. of Dextran. His only new symptom was left hemicranial headaches, more severe in the left orbit and left temporal area. He also stated that the severe pains in the left eye preceded every episode of epistaxis. The bruit was heard again over the left globe. The left pupil was 2 mm. larger than the right. There was proptosis of the left eye, hypesthesia of the 1st and 2nd divisions of the left trigeminal nerve and paresis of the left lateral rectus muscle. It was thought at this time that the aneurysm had eroded into the ethmoid and sphenoid sinuses with subsequent intermittent rupture of the aneurysm and bleeding into the paranasal sinuses, thus giving rise to the severe epistaxis. Repeated left carotid and right subclavian arteriograms again revealed an aneurysm of the left internal carotid artery. There was good cross-filling, right to left, when the left common carotid artery was compressed. The right vertebral artery and the basilar artery filled poorly. The left common carotid artery was ligated totally and imbricated 1 cm. proximal to its bifurcation in the neck.

The postoperative course was satisfactory, and the patient was discharged home with no recurrences of epistaxis and with no significant change in his neurological status.

On March 7, 1962 he stated that he had heard the "roaring noise" on one occasion since his previous discharge from the hospital, but had had no epistaxis.