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. 8, 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; hypesthesia 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
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.
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Fig. 2. Base of skull demonstrating defects in left orbital plate (1) left jugum and atrophy of left optic nerve (3). The aneurysm lies immediately below the enlarged left foramen ovale (3).

On March 26, 1962 he had a sudden onset of severe epistaxis and went into shock. Blood pressure was 90/0 and hematocrit was 24 per cent. Neurological findings were normal except for total blindness of the left eye. The pupils were equal and both reacted sluggishly to light and the extraocular muscles were intact. Bleeding, clotting and prothrombin times were all normal. Bilateral carotid arteriogram revealed the previously described aneurysm which had not changed significantly except for a formation of a small “teat-like” projection extending for 2 mm. anteriorly. The aneurysm, however, filled only via an arteriogram needle inserted in the left carotid bulb above the point of previous common carotid ligation. The right common carotid artery was compressed for a few seconds and the patient had a grand mal seizure, from which he recovered. He was then transfused to a hematocrit of 32 per cent. Several days later right carotid arteriography was performed with compression of the left internal and external carotid arteries. After about 15 sec. of compression early seizure activity developed. Roentgenograms showed fair cross-filling (right to left), but in view of his intolerance to occlusion of the left internal and external carotid arteries the neurosurgeons were hesitant to operate to trap the aneurysm intracranially which would stop any blood flow which he might be receiving via anastomoses to the left carotid system. However, in view of the recurrence of severe epistaxis it was decided to transfuse the patient to a high hematocrit and proceed with operation. During the night prior to operation he again had severe bleeding from his nose and expired.

Autopsy was limited to the head. There was (1) a linear fracture with nonunion of portions of its edges located in the orbital plate of the anterior cranial fossa on the left, beginning at a distance of 2.0 cm. from the anterior pole of the plate. The lateral borders of this defect were 4 mm. from each other at their widest point. A small amount of viscid, yellow, mucoid material was present within the defect, and extended out onto the orbital plate. There was an old craniotomy defect, 2×1.8 cm. in size, in the squamous portion of the left temporal bone 2.5 cm. caudal to the posterior border of the lesser wing of the sphenoid. This bony defect was covered by dura mater, fibrous tissue, and the body of the temporalis muscle. There was (2) a roughly rounded, 4 mm. defect in the jugum, just anterior and lateral to the left anterior clinoid process. (3) A smooth, glistening, dark red mass could be seen through an enlarged left foramen ovale (Fig. 2). There was atrophy of the left optic nerve, which measured 2.5 mm. in diameter compared to a diameter of 4.0 mm. for the right optic nerve.

The majority of the right and left orbital plates, the anterior clinoid processes, the posterior clinoid processes, and the petrous portions of the temporal bones were removed to expose both internal carotid arteries from their upper cervical portion and including their petrous and cavernous segments. The left orbital plate was markedly thinned. The sphenoidal and ethmoidal sinuses were unroofed and were seen to be filled with fresh, clotted blood (Fig. 3). There was a firm, multilobulated mass, 2.4×1.8×1 cm. in size, arising from, and attached to, the anterior and inferior portion of the cavernous portion of the left internal carotid artery (Fig. 4).

Following the unroofing of the orbits, the peri-orbita were incised bilaterally and the extrinsic muscles of the eye and the periorbital fat were removed to expose the optic nerves in their entirety. These nerves were sectioned immediately posterior to the globes and submitted for microscopic examination. The measurements of the right and left optic nerve, as stated above, were taken at a point 1.5 cm. posterior to their insertion in the globes. No dilatation or aneurysmal formation were noted in the ophthalmic artery on the left.

The pituitary gland was removed and appeared normal grossly with no evidence of compression by the aneurysm of the left internal carotid artery. Fig. 3 shows the anatomic relationship between the left internal carotid artery, the aneurysm, and the optic nerves, and demonstrates the marked
Epistaxis Caused by Ruptured Aneurysm

FIG. 3. (Left) Aneurysm in situ. The paranasal sinuses are filled with fresh hemorrhage. Note marked atrophy of left optic nerve. (Right) Artist’s overlay of same area.

atrophy of the left optic nerve caused by compression by the aneurysm.

Sections of the 2.8 cm. aneurysm revealed the lumen to be almost completely filled with laminated thrombus (Fig. 5). The aneurysmal wall varied in thickness from less than 0.5 to 1.5 mm. No gross calcification or large atheromata were encountered in the wall. The lumen of the internal carotid artery was patent throughout, and there was a small, clot-free area in the aneurysm adjacent to the lumen of the vessel. The point of rupture of the aneurysm could be identified in its dome, and measured 1.5 mm. This point corresponded to the “teat” noted on the angiograms done during his last hospitalization.

The brain revealed areas of yellowish-orange discoloration, softening, and depression of the cortex of the left orbital gyri, gyrus rectus, and of the right inferior temporal gyrus. The vessels of the circle of Willis had only mild, patchy atherosclerosis. No aneurysms were encountered on the vessels of the circle of Willis.

Serial sections of the brain at 0.5 cm. intervals revealed no abnormalities except for softening, yellow-orange discoloration, and atrophy in the left orbital gyri and the right inferior temporal gyrus corresponding to the areas of encephalomalacia noted on the uncut brain.

Microscopic sections of the left orbital and right inferior temporal gyri revealed loss of neurons from the cortex and degeneration and fennulation of the convolutional and central white matter in the area of involvement. There was a marked increase in perivascular glia in both cortex and white matter in the areas of encephalomalacia, together with large numbers of macrophages containing abundant, foamy cytoplasm.

Longitudinal and cross-sections of the left optic nerve demonstrated increased numbers of astroglial and fibroblastic nuclei within the nerves, a decrease in the cross-sectional diameter of the nerve, a marked increased prominence of the fibrous tissue septae separating the nerve bundles, and a marked pallor of staining of the nerve with the myelin-sheath preparations.

Multiple sections taken at various levels through the saccular aneurysm of the cavernous portions of the left internal carotid artery revealed the lumen to be filled with laminated antemortem thrombus. The erythrocytes comprising the thrombus were relatively well-preserved, suggesting a recent origin for most of the thrombus. The walls of the aneurysm were increased in thickness, with rather prominent hyalinization and thickening of the intima, and fragmentation and virtual dissolution of muscle in a number of areas. There were small numbers of lipid-filled macrophages in the subintima and intima and a
only one with an aneurysm in this location among our 118 patients. Two of the largest series of aneurysms of the cavernous portion of the carotid artery were reported by Meadows (15 cases) and by Jefferson (17 cases). Hamby reported the highest incidence among aneurysms of the carotid-vertebral systems, having encountered 10 cases of "subclinoid" aneurysms in 86 patients with aneurysms. Walton discussed Hamby's series and concluded, as do we, that his high incidence of aneurysms of the internal carotid artery reflect their relative ease of diagnosis by the neurosurgeon and otolaryngologist. Aneurysms of the cavernous portion of the

Fig. 4. Left internal carotid artery with the large, multinucleated aneurysm arising from its cavernous portion.

Fig. 5. Cross-sections of aneurysm. The lumen of the aneurysm is almost completely filled with laminated thrombus.

Discussion

The rarity of aneurysms of the petrous and cavernous portions of the internal carotid arteries is attested to by numerous reports, and by our own experience with 118 patients with 162 aneurysms coming to autopsy during the 10 years 1952–1962. The patient reported here was the
internal carotid arteries would appear to comprise less than 1 per cent of all aneurysms of the carotid-vertebral systems.

The etiology of aneurysms of the carotid-vertebral systems is still not settled. Such statements as “evidence accumulated over a long period of time by many workers has now established beyond doubt that arteriosclerosis plays no direct part in the pathogenesis of the saccular aneurysm” are probably too dogmatic. Housepian and Pool\textsuperscript{24} stressed some of the difficulties in classifying many of the saccular aneurysms, and Courville\textsuperscript{10} has illustrated large, saccular aneurysms that he considered to be arteriosclerotic. Hamby\textsuperscript{21} considered that both arteriosclerotic and congenital aneurysms occur in the cavernous portion of the internal carotid arteries. The age of our patient, the absence of the aneurysm at a demonstrable vascular bifurcation, and the marked thickening of the aneurysmal wall would tend to suggest the possibility of an arteriosclerotic origin, but would by no means prove it. In general, we tend to consider saccular aneurysms as congenital. The possibility that this, and the other reported aneurysms, arose from the vestibial artery of Padget must be considered.\textsuperscript{14} It cannot be proven. Instructive in regard to the etiology of these aneurysms are the findings reported by Meadows\textsuperscript{32} of 2 autopsies in which aneurysms of the vessels of the circle of Willis were also present. This is, in our opinion, strong agreement for a congenital origin, as multiple aneurysms have been found in 25 per cent of our patients with aneurysms seen at autopsy.

Finally, the relation of the aneurysm to known trauma must be considered. As noted in Table 1, trauma is a feature in many of the reported cases. Trauma, alone without a cavernous-carotid aneurysm, may produce many of the findings that occur in cases of aneurysm, as has been well summarized recently by Maurer \textit{et al.}\textsuperscript{32} Weaver \textit{et al.}\textsuperscript{49} attributed such hemorrhage to the production of a “false” aneurysm following a tear in the internal carotid artery. As several of the reported cases had no history of trauma, trauma alone cannot be evoked to explain all of the aneurysms. Courville\textsuperscript{9,35} has stressed that the incidence of “probable traumatic aneurysms” of the carotid-vertebral system of vessels is “extremely low”, and has specifically mentioned aneurysms located within the cavernous sinus as examples of trauma rupturing “pre-existing” saccular aneurysms. We concur with this statement. Brihaye \textit{et al.}\textsuperscript{5} have described what they considered to be a traumatic supraclinoidal aneurysm. Moritz\textsuperscript{29} has described saccular aneurysms of extracranial vessels resulting from trauma, but cited no examples of carotid or intracranial aneurysms. The type of head trauma sustained in our case corresponds to what Courville\textsuperscript{21} has called a “group C” injury, or one that was sustained when the head was in motion and struck an immovable object. The coup-contrecoup lesions of the brain in our case were quite clear.

Our review of the literature on epistaxis secondary to cavernous-carotid aneurysms reveals a rather widespread acceptance on the part of many authors of patients in whom the existence of an aneurysm has not been proven. Table 2 summarizes these cases. We have required clear-cut radiologic, surgical, or autopsy demonstration of an aneurysm as the only proof of its existence. All other reported cases are considered as either possible or probable, depending on the clarity of the clinical syndrome. The syndrome of the cavernous sinus is sufficiently familiar\textsuperscript{2,15,21,27,33,42} so that details need not be given here.

It is somewhat surprising that aneurysms of the cavernous portion of the carotid artery do not give rise to epistaxis more often. Indeed, it is not even mentioned as a possible cause in some reviews of severe epistaxis.\textsuperscript{29} Blackwood\textsuperscript{17} illustrated such an aneurysm which had eroded into the sphenoid sinus and had bled into the sinus, apparently without epistaxis. Two very common findings in saccular aneurysms of the cavernous portion of the internal carotid artery are their large size and their partial thrombosis.\textsuperscript{1,2,17,19,27,33} Our case illustrates both of these two features very well. The presence of intra-aneurysmal thrombus may well be in part responsible for the relative infrequency of their bleedings.
Another factor in the infrequency of bleeding may be the relatively thick wall possessed by many of these large aneurysms.

Summary

We have reported the twentieth "proven" case of a ruptured saccular aneurysm of the cavernous portion of the internal carotid artery associated with epistaxis. After careful review of the literature and our own case we have concluded that saccular aneurysms of this portion of the carotid system are quite uncommon, are either congenital or atherosclerotic in type, and may erode into the paranasal sinuses. Rupture of such an aneurysm which has eroded into the sphe-
noidal air cells may give rise to severe, and often fatal, epistaxis. Trauma with fracture of the skull appears to be important in causing these saccular aneurysms to rupture, but is not thought to be the cause of the aneurysm per se.

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