

Posttraumatic Intracavernous Aneurysm

Epistaxis With Monocular Blindness Preceded by Chromatopsia

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Two and a half months after blunt head injury, a 19-year-old man complained of headaches, repeated nosebleeds, unilateral blindness, and numbness below the eye. Angiography revealed an aneurysm arising from the carotid artery in its intracavernous course and extending into the sphenoid sinus. This clinical presentation is typical of the rare *traumatic intracavernous*

carotid aneurysm and contrasts with the symptoms produced by a slowly expanding *spontaneous* aneurysm at this site. Recognition of this posttraumatic clinical syndrome can lead to prompt and effective surgery and should reduce the present mortality of approximately 45% for these lesions.

ANEURYSMS OF the carotid artery within the cavernous sinus comprise less than 1% of all intracranial aneurysms encountered at postmortem¹ and represent 1.9% of aneurysms reported in a recent clinical study.²

Despite this rarity, the intracavernous aneurysm is generally familiar to the ophthalmologist because of its tendency to act as a slowly-expanding mass causing progressive unilateral ophthalmoplegia often followed by visual failure and facial numbness.³

A small percentage of aneurysms at this site occur following blunt and, less commonly, penetrating head injury. These posttraumatic intracavernous aneurysms do not act as slowly-expanding masses but tend to expand medially and downwards into the sphenoid sinus usually presenting with progressively severe, often fatal, epistaxis.

As unilateral visual loss is almost always

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present, the ophthalmologist is frequently consulted and should be aware of this unusual but eminently treatable condition.

Our experience with a recent case illustrates some of the clinical problems involved in diagnosing a traumatic intracavernous aneurysm, and this report is offered with a view towards increasing the awareness of its typical clinical presentation.

Report of a Case

History.—A 19-year-old man was admitted to the hospital after sustaining a head injury when the bicycle he was riding was struck by an automobile. He was confused and agitated but responded appropriately to verbal commands. His blood pressure was 140/90 mm Hg and the pulse rate was 80 beats per minute. A laceration was present over the left zygoma. He had ecchymoses about both eyes, more so on the left, and dried blood was present in both nostrils. The left pupil was 1 mm larger than the right but both responded briskly to light. The optic fundi were normal and eye movements were full. His sense of smell was not tested but the remainder of his nervous system was normal on clinical examination. X-ray films of the skull and facial bones demonstrated no fracture but showed sphenoid sinus clouding, probably representing blood or cerebrospinal fluid in association with a subtle fracture through the wall of the sphenoid sinus. (Skull

x-ray films taken the year before were completely normal.)

He was discharged from the hospital a week later, symptom free aside from noting a decrease in his sense of smell and "taste."

Ten days after the injury, he first noticed numbness below the left eye and inside his mouth on the left.

Seventeen days after the accident, the numbness was joined by a left frontal and retro-orbital headache and he experienced his first nosebleed.

Three weeks after the injury while watching television, the patient suddenly noted a "blue-

ness" in his vision. He made the observation that objects looked normal with the right eye whereas with the left eye objects appeared distinct but were blue-tinged and surrounded by a blue halo. During the next ten days, he noted progressive impairment of vision in the left eye and increasingly intense headaches. The nosebleeds became so severe that he finally sought medical advice ten weeks after his accident.

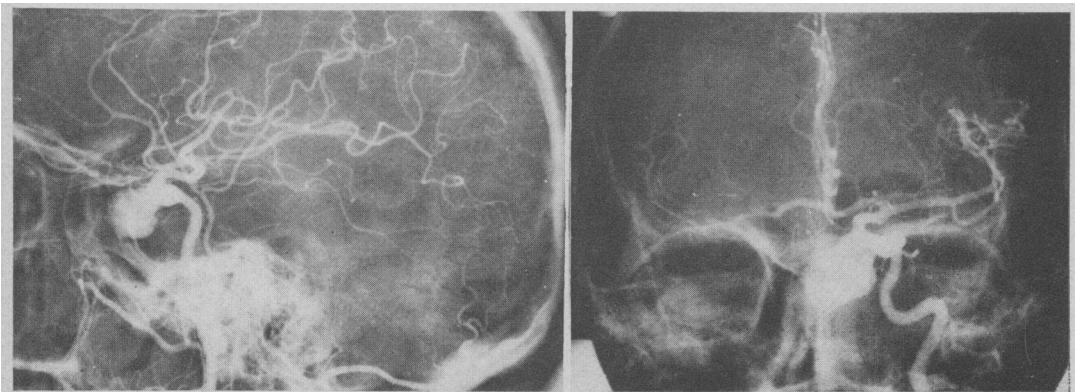
Examination.—The patient was fully conscious and alert but apprehensive, pale, and looked ill. Fresh blood exuded from both nostrils and a faint hemorrhagic discoloration encircled the left eye. His blood pressure was 120/80 mm Hg and the pulse rate was 60 beats per minute. Visual acuity and the field of vision were normal in the right eye. There was no perception of light in the left eye, but he was able to detect accurately and repeatedly the beam of a bright flashlight directed into the blind eye as a sensation of pain in this eye. The left pupil was 0.5 mm larger than the right and exhibited a brisk consensual but no direct light reaction under careful observation. The left optic disc was moderately pale and the right was normal. Extraocular movements were full. A moderate blunting of sensation to a pin in an area surrounding the left eye and extending to the left upper lip was found. The sense of smell could not be tested but the other cranial nerves as well as the remainder of the nervous system were normal (Fig 1).

Radiographic Examination.—Conventional and polytomographic studies of the orbits demonstrated opacity of the ethmoid and sphenoid sinuses, with erosion of the sphenoid bone involving the floor of the sphenoid sinus and the left superior orbital fissure and optic foramen. An old fracture involved the left lateral extremity of the brow. A soft tissue mass filled

Fig 1.—Preoperative photograph showing discoloration about the left eye and ineffective nasal packing.



Fig 2.—Left carotid angiogram views demonstrating intracavernous carotid aneurysm extending into sphenoid sinus.



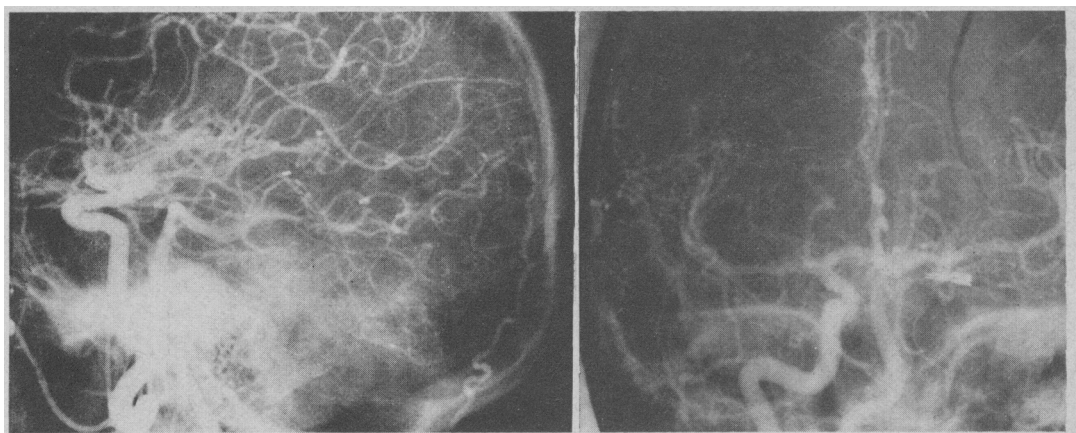


Fig 3.—Immediate postoperative right carotid angiogram views showing lack of aneurysm filling.

two thirds of the nasopharynx, suggesting a nasopharyngeal tumor.

Progress.—His epistaxis became more severe and persisted despite nasal packing, and he required a transfusion of four units of blood. Rhinoscopy revealed a deep left intranasal mass which was thought to be a highly-vascular malignant tumor. Biopsy was recommended but because of the uncertain nature of the destructive mass, cerebral angiography was performed.

A left carotid angiogram showed a large multilobular aneurysm arising from the intracavernous portion of the internal carotid artery and extending into the sphenoid sinus (Fig 2). The aneurysm was thought to be partially filled with blood clot and its true size much larger than the contrast-opacified mass because of the extensive bony destruction seen on the plain x-ray films and the presence of the large nasopharyngeal mass. Immediate surgery was performed.

Operation.—Through a frontal craniotomy, a silver clip was placed across the left internal carotid artery to occlude it as it emerged from the cavernous sinus. The left internal carotid artery in the neck was occluded with ligatures and a transfixing silk suture. The aneurysm was thereby isolated or "trapped."

Subsequent Course.—He made an uneventful recovery from the operation; the epistaxis promptly ceased and his headache was gone. An immediate postoperative angiogram showed that the aneurysm failed to fill with contrast. The vascularization of the left cerebral hemisphere was normal and had been taken over by the right carotid artery (Fig 3).

When seen one year later, there had been no recurrence of headache or nosebleeds. The left eye remained blind with marked optic atrophy and normal retinal vessels, the ophthalmic ar-

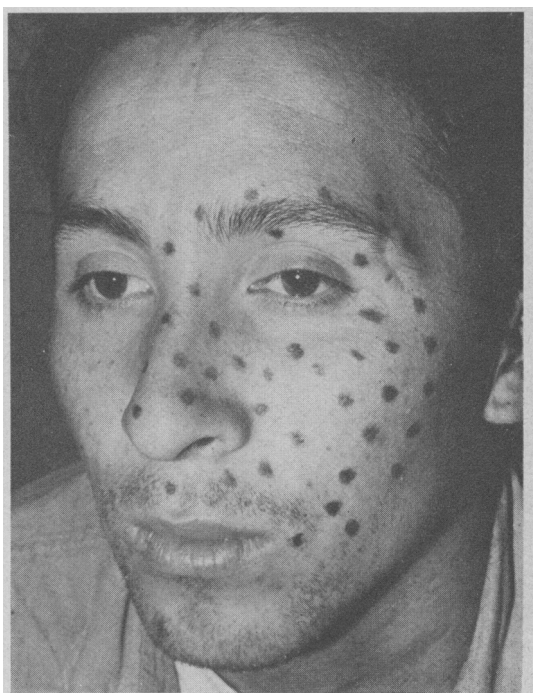


Fig 4.—One year postoperative photograph outlining area of sensory decrease.

tery being supplied through external carotid anastomoses. Sensation was diminished over parts of the ophthalmic and maxillary divisions of the left trigeminal nerve (Fig 4).

Comment

The monocular cyanopsia our patient experienced provided good documentation of the course of his visual loss. Chromatopsia is most often binocular and a result of drug

intoxication, but has been reported occasionally in a wide variety of diseases of the retina and optic nerve.⁴ Monocular chromatopsia is uncommon, and is a most unusual manifestation of optic nerve compression.

Carotid aneurysms within the cavernous sinus present in one of three ways. The commonest presentation is that of a slowly expanding mass with compression of the structures within the cavernous sinus. Once an aneurysm becomes sufficiently enlarged, rupture is less common, presumably because of the protection afforded by the laminated thrombus lining its walls. Aneurysms in the cavernous sinus receive additional support from the surrounding dura. All carotid aneurysm sites favor women,² but these spontaneous intracavernous aneurysms are particularly prone to affect middle-aged and elderly women.³ The typical clinical syndrome is that of headache associated with slowly progressive unilateral ophthalmoplegia, often followed by ipsilateral visual loss and sensory impairment in the trigeminal distribution.³ Very rarely, medial and downward expansion into the sphenoid sinus will produce epistaxis.⁵⁻⁷

Undoubtedly, many of the intracavernous aneurysms rupture when still small and asymptomatic to produce a carotid-cavernous fistula which usually manifests itself as unilateral proptosis with an orbital bruit. Only rarely can an aneurysm be identified in studies of such fistulae, but presumably this is a major mechanism for the development of both spontaneous and delayed traumatic carotid-cavernous fistulae.

Our patient represents the least common type—the posttraumatic intracavernous aneurysm. The clinical evolution in reported instances of this uncommon aneurysm is remarkably constant and differs from that of patients with spontaneous intracavernous aneurysms. The typical history is that of a young man (the age and sex correlate with the incidence of head trauma) who awakens from coma secondary to major, usually blunt, head trauma in a traffic accident. Unilateral blindness is noted by the patient when he regains his senses although occasionally delayed blindness occurs as in our case. Although the third, fourth, fifth, and sixth cranial nerves are often affected, either ipsilaterally or bilaterally at the time of

trauma, less frequently these cranial nerves are gradually involved by the expanding aneurysm. X-ray films nearly always reveal evidence of an anterior basal skull fracture.

Within one or two weeks to many months, he experiences the onset of repeated nosebleeds of increasing frequency and severity. The number and pattern of the epistaxes vary but the initial nosebleed is rarely if ever fatal. In nearly half the cases reported, the patient dies as a result of the profuse hemorrhage before diagnosis, or, as in several cases, despite carotid ligation alone.^{5,6,8} If the diagnosis is suspected, angiography reveals the aneurysm arising from the carotid artery in the cavernous sinus and extending into the sphenoid sinus.

Epistaxis from a traumatic intracavernous aneurysm has been documented for at least 100 years,⁹ but the exact incidence of these aneurysms remains unknown. Van Beusekom et al in 1966,⁵ in a careful review of the literature, found 27 cases of epistaxis from proven posttraumatic carotid aneurysms. Since then, reports of 11 additional cases following blunt trauma^{8,10-16} and two cases following penetrating injuries^{17,18} have been made known to us. This apparent increase in incidence probably reflects a mounting clinical awareness of the posttraumatic intracavernous aneurysms, the proliferation of automobiles with the associated increase in blunt head trauma, and the more frequent resort to cerebral angiography in the management of such injured patients.

The relationship of these aneurysms to trauma is convincing in nearly all reported cases. Indeed, a fracture through the adjacent wall of the sphenoid sinus would seem the most likely way to explain the preference of posttraumatic aneurysms to expand medially from the cavernous sinus into the sphenoid sinus. McCormick and Beals¹ performed a thorough clinical and pathological study of an intracavernous aneurysm in a 70-year-old man who presented with epistaxis following head trauma. They suggested that trauma acts to encourage rupture of a preexisting carotid aneurysm rather than to damage the normal carotid artery in the sinus to initiate aneurysm formation. This is undoubtedly true in some instances (certainly in those instances where the trauma was surgical biopsy⁵), but the theory of

trauma to a previously normal carotid artery is easier for us to reconcile with the striking difference in age, sex, and clinical course of patients with a traumatic carotid aneurysm as compared to those with the spontaneous variety.

In contrast to current discussion about the timing and techniques of treatment of most intracranial aneurysms, there is no debate about the immediate need and, we feel, little choice about the type of surgical intervention when a traumatic aneurysm is actively bleeding into the sphenoid sinus. Only one example of a spontaneous cessation of epistaxis from an aneurysm is known to us, and this involved a resourceful man who occlud-

ed his own carotid artery at each bleed.¹⁵ Several fatal exsanguinations have occurred despite carotid ligation alone. Immediate single stage occlusion of the carotid artery above and below the aneurysm appears to be the minimal procedure required to stop the hemorrhage. Fortunately, the youth of most of the patients minimizes the morbidity of such a procedure.

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Key Words.—Trauma; aneurysm; carotid artery; cavernous sinus; monocular blindness; epistaxis; carotid ligation; facial numbness; chromatopsia.

References

1. McCormick WF, Beals JD: Severe epistaxis caused by ruptured aneurysm of the internal carotid artery. *J Neurosurg* 21:678-686, 1964.
2. Sahs AC, Perret GE, Locksley HB, et al (eds): *Intracranial Aneurysms and Subarachnoid Hemorrhage*. Philadelphia, JB Lippincott Co, 1969.
3. Meadows SP: Intracavernous aneurysms of the internal carotid artery. *Arch Ophthalmol* 62:566-574, 1959.
4. François J, Verriest G: On acquired deficiency of colour vision. *Vision Res* 1:201-219, 1961.
5. Van Beusekom GT, Luyendijk W, Huizing EH: Severe epistaxis caused by rupture of a non-traumatic infraclinoid aneurysm of the internal carotid artery. *Acta Neurochir* 15:269-283, 1966.
6. Polcyn JL, Roth GR Jr: Epistaxis from rupture of aneurysm of internal carotid artery. *JAMA* 213:876, 1970.
7. Poffenbarger AL, Velasquez AC, Church J: Recurrent epistaxis secondary to aneurysm of internal carotid artery. *JAMA* 216:1644, 1971.
8. Handa J, Kikuchi H, Iwayama K, et al: Traumatic aneurysm of the internal carotid artery. *Acta Neurochir* 17:161-177, 1967.
9. Hamby WB: *Intracranial Aneurysms*. Springfield, Ill, Charles C Thomas Publisher, 1952, p 225.
10. Maurer JJ, Mitchell M, German WJ: Triad of unilateral blindness, orbital fractures and massive epistaxis after head injury. *J Neurosurg* 18:837-840, 1961.
11. Bonnal J, Stevenaert A, Beaujean M, et al: Epistaxis répétées graves, parfois mortelles, secondaires à des lésions de la carotide interne. *Neurochirurgie* 13:417-430, 1967.
12. Arseni C, Ionesco S: Hémorragie nasale grave provoquée par la rupture d'un anévrysme carotidien intracranien posttraumatique. *Rev Otoneuroophthal* 41:149-154, 1969.
13. Loiseau G, Marchand J, Moncade J, et al: Epistaxis grave par rupture d'anévrysme de la carotide interne dans son segment infraclinoidien. *Ann Otolaryng* 84:472-475, 1967.
14. Malecki J: Do kazuistyki krwotokow nosowych z tetniaka tetnicy szyjnej wewnetrznej. *Otolaryng Pol* 21:475-478, 1967.
15. Zakrzewski A, Konopacki K, Kwaskowski A, et al: Rozdarcie tetnicy szyjnej wewnetrznej w przebiegu zlamanania podstawy czaski. *Otolaryng Pol* 23:685-692, 1969.
16. Philippides D, Buchhest F, Roth A, et al: Anévrysme post-traumatique de la carotide intracaverneuse. *Rev Otoneuroophthal* 41:345-349, 1969.
17. Petty M: Epistaxis from aneurysm of the internal carotid artery due to a gunshot wound. *J Neurosurg* 30:741-743, 1969.
18. Jackson FE, Augusta FA, Sazima H, et al: Head injury and delayed epistaxis: Report of case of rupture of traumatic aneurysm of internal carotid artery due to grenade fragment wound received in Vietnam. *J Trauma* 10:1158-1167, 1970.