TRIAD OF UNILATERAL BLINDNESS, ORBITAL FRACTURES AND MASSIVE EPISTAXIS AFTER HEAD INJURY*

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(Received for publication April 11, 1961)

The occurrence of massive, life-endangering epistaxis following closed craniocerebral trauma is extremely uncommon. It was first reported in 1928 by Birley,1 with a note by Trotter. In 1958, Hamilton2 noted only 3 previous reports in the literature. We have found a total of only 9 cases, including the present one.

The onset of bleeding may be insidious and remote in time from the trauma. It may be extremely difficult to identify the location of the bleeding and the pathologic anatomy that is involved. The more common type of epistaxis following cranio-cerebral trauma is that arising from the anterior ethmoidal or sphenopalatine arteries.3 The rate and rapidity of this hemorrhage usually does not become life-endangering and it is likely to be controlled by nasal packing. The massive type of hemorrhage, from which the patient rapidly goes into shock, has its origin directly from the internal carotid artery. In all the cases reviewed, the blood reached the nasopharynx via the sphenoidal air sinus. The literature is abundant with reports of cases of fistulous communications between the internal carotid artery and the cavernous sinus. However, the vast majority of these lack the prerequisite for massive epistaxis, namely, a fracture across the floor of the anterior cranial fossa and into the sphenoidal air sinus. The purpose of this paper is to bring this interesting and challenging entity to the current literature and to discuss the clinical picture in the following case, which is remarkably similar to other cases reported.

CASE REPORT

R.A.H., an 18-year-old man enlisted in the Navy, was admitted on Jan. 5, 1960 complaining of blindness of the left eye of 3 week's duration. He had been injured in an automobile accident on Dec. 14, 1959, sustaining severe blunt trauma to the left orbit. He was taken to a hospital where the findings were a semicomatose state and bleeding from a laceration above the left eye. Within 4 days, his level of consciousness returned to normal and he was discharged. The following day, swelling about the left orbit decreased sufficiently to allow the eye to open and the patient noted that this eye was blind. Because of this, he reported to the U. S. Naval Hospital in St. Albans for evaluation.

Physical examination revealed a healed laceration below the left eyebrow and resolving ecchymosis of the left bulbar conjunctiva. The left pupil was dilated and did not react to light. There was no perception of light in the left eye. The fundi were normal. Bilateral 6th cranial nerve palsy was present, complete on the left and partial on the right. Roentgenograms of the skull revealed several fracture lines in the median aspect of the left orbit extending to the floor of the anterior fossa. The ethmoid air cells were cloudy on the left; the sphenoid sinus was clear (Fig. 1).

Course. The patient was being followed to evaluate the right 6th nerve palsy when, on Jan. 16, 1960, he suddenly had severe epistaxis from the left nostril which ceased without packing and did not require transfusion. Examination of the nose and nasopharynx revealed no bleeding point. In the following 20 days, two more minor episodes of epistaxis occurred. Then on Feb. 5, 1960, a sudden massive epistaxis began at 2 A.M. Bilateral anterior and posterior packs seemed to control the hemorrhage but four units of blood were required for the treatment of shock. No bruit was audible over the skull the following day. The packing was removed on the 2nd day and the patient did well until Feb. 13, 1960, when another massive hemorrhage from the nose and mouth occurred, requiring three units of blood. The patient now noted that he could tell when he was going to bleed by a “tickling” sensation in his nasopharynx. A bruit was heard over both eyes, louder on the right. Then, on Feb. 14, 1960, the third massive hemorrhage occurred and another three units of blood were given. Several hours later, as preparation for carotid angiography was being made, he bled again, losing blood so rapidly into the pharynx that rapid, bedside tracheotomy was required to relieve obstruction of airway. The patient was transfused with six more units of blood and the left common carotid artery was ligated under local anesthesia. The bleeding ceased immediately.

The patient demonstrated no untoward effects from the carotid ligation and was ambulatory within 48 hours postoperatively. Within 2 weeks the palsy of the left 6th nerve improved to 60 per cent of normal, the right to 90 per cent. He remained blind in the left eye. A faint bruit persisted over both eyes, louder on the right. Compression of the right carotid artery made the bruit much louder over the left eye and fainter over the right.

Right carotid angiography revealed excellent bilateral

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FIG. 1. Oblique roentgenogram of skull showing fracture of posteromedial wall of left orbit.

opacification of the anterior and middle cerebral arteries but no flow into the left internal carotid artery or the cavernous sinus. Left carotid angiograms were then performed under direct vision with cannulation of the internal carotid. The left internal carotid artery was demonstrated to be thrombosed at the level of the base of the skull, probably the result of the common carotid ligation (Fig. 2). A strong pulsatile flow to this point was shown by direct pressure recordings to come entirely from the external carotid by retrograde, collateral circulation. The external carotid artery was ligated just beyond the origin of the superior thyroid artery, allowing only a small, pulsatile flow to continue into the internal carotid.

The patient recovered uneventfully from these procedures. Re-examination disclosed no change in the intracranial bruit, the source of which is not obvious. Except for the lesions of the optic and abducens nerves he has remained asymptomatic to date, and no further therapy is planned.

DISCUSSION

As the case report illustrates, this clinical syndrome can present a challenge both in diagnosis and treatment. Unilateral blindness, a fracture of the roof of the orbit and massive epistaxis are the characteristic triad.

Table 1 illustrates several points of interest concerning the 8 cases reported previously, together with the present case. Epistaxis was delayed in 5 instances, the longest interval being 3 months. The number of hemorrhages varied from 2 to 14 and there was a tendency toward increasing severity. The optic nerve was involved in every case, usually with homolateral blindness. Injury to the oculomotor nerve was apparent in 6 instances, although pupillary dilatation was the only evidence in the present case. Impairment of the 6th nerve was present in 4 cases, being bilateral in 2 of these. Damage to the olfactory nerve also was noted in 4 patients. The other cranial nerves were involved as follows: 4th, 3; 5th, 2; and 7th, 1. A cranial bruit was noted in 5 cases, not mentioned in 3 and said to have been absent in 1. The earliest period at which the bruit was detected was 7 weeks, the latest, 6 months. Cranial fracture was identified in the anterior fossa in 6 instances, the middle fossa in 2. The usual treatment was ligation of the cervical carotid artery. There were 3 fatal results, all from hemorrhage: 1 apparently because of incomplete occlusion of the common carotid, in spite of additional intracranial clipping of the carotid; 1 in a case in which no ligation was performed, and 1 for undetermined reasons. In 1 instance, intracranial clipping of the carotid artery apparently was responsible for a successful result after cervical ligation of the common and internal carotid arteries had been followed by another epistaxis.

Intracranial hemorrhage of sufficient extent to be detected clinically has not been reported, nor is intracranial hemorrhage mentioned as a nec-
Massive Epistaxis After Head Injury

An angiogram of the left internal carotid artery 2 weeks after ligation of the left common carotid artery.

At autopsy, there must be a direct communication between the traumatic aneurysm and the air sinus. The lack of a fistula between the aneurysm and the cavernous sinus explains the absence of pulsating exophthalmos in these patients.

An aura or warning of hemorrhage may occur shortly before the onset of bleeding. This may vary from a vague feeling in the nasopharynx to facial paresthesias or a sensation of pressure on the vertex of the cranium. The proximity of the lesion to the 1st and 2nd divisions of the 5th cranial nerve may account for some of these symptoms.

In the treatment of these patients, the rapid control of life-endangering hemorrhage is of first importance. If conditions permit, carotid angiography should be carried out promptly to confirm the diagnosis. The optimum extent of surgical intervention is not established clearly in the literature. One author found it necessary to ligate the homolateral common and internal carotid arteries. Ligation of the former prevented epistaxis for 6 weeks but then massive bleeding required further ligation of the internal carotid. Schorstein has discussed extensively carotid ligation in the treatment of intracranial aneurysms but none of these was traumatic.

For a thoughtful consideration of this subject, we could not improve upon the note by Trotter, appended to the report of the first case, for which he was the surgeon.

"Note by Mr. Wilfred Trotter.—It is relevant here to state the reasons which led the surgeon to the choice of this operation. In general, it may be said that the surgical treatment of internal carotid aneurysms, intracranial or cervical, by proximal ligature, if it is unsuccessful fails through too little effect on the circulation rather than through too much. In dealing with these aneurysms the risk of doing an inadequate operation is greater than that of damaging the nutrition of the brain. There is in fact no condition which causes a more thorough dilatation of the collateral blood-supply of a part than does an aneurysm of its main artery. In this case there was a special reason for assurance that the collateral circulation was already fully prepared to nourish the brain. The primary hemiplegia was probably due to the peripheral end of the ruptured artery having been obstructed from the first, and yet it had passed off completely in a comparatively short time. It was to be presumed, therefore, that the left carotid was already chiefly answerable for the blood-supply of the anterior and middle cerebral territories on the right side.

"The traditional treatment of internal carotid aneurysms is ligature of the common carotid. It is defective because it allows blood from the external carotid—freely supplied across the middle line of the face—to pass down through the bifurcation into the internal carotid and so to reach the aneurysm. Ligature of the internal carotid itself, again, may be ineffective, because it allows blood to pass up the external carotid and through the orbit to the aneurysm. Clearly then two ligatures are necessary to give the fullest possible control of blood to the aneurysm. These may be applied to the two branches above the bifurcation, as was done in this case, or, as is sometimes more convenient, to the common and external carotids. A transverse incision..."
with longitudinal splitting of the sterno-mastoid was used. It gives direct access to the vessels and avoids the permanent depression in the contour of the neck that is apt to follow the usual displacement of the muscle. The operation in itself is trivial, and this fact was felt to be an additional justification of its being done without preliminary blood-transfusion."

Combining safety with logic, it seems best initially to ligate the common carotid rather than the internal carotid artery for control of the acute massive epistaxis. With the hemorrhage controlled and when the patient stabilizes, a contralateral carotid angiogram may be done in an attempt to demonstrate the aneurysm by cross-circulation. In addition, it is important to know if blood is still entering the aneurysm from the homolateral carotid. This information may be obtained, as in our case, by direct angiography. During this procedure, if it is found that the ipsilateral external carotid artery contributes significantly to the aneurysm, it should be ligated. The procedure of intracranial entrapment discussed by Echols and Jackson may be considered if a significant blood supply is obtained by cross-flow through the circle of Willis.

SUMMARY

1. The case presented together with the 8 reported previously in the literature establish a definite triad of unilateral blindness, orbital fractures and massive epistaxis after head injury.

2. The epistaxis is caused by rupture of a traumatic aneurysm of the internal carotid artery into the sphenoidal sinus.

3. An approach to the surgical management of these cases is suggested.

REFERENCES


