INTERNAL CAROTID ARTERY ANEURYSM PRESENTING WITH SEVERE EPISTAXIS

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Abstract
A case of aneurysm of the cavernous part of right Internal Carotid Artery is reported. This is a rare condition. The patient presented with recurrent episodes of epistaxis from right nostril for 10 years. Previously his right Common Carotid Artery and subsequently the Internal Carotid Artery had been ligated in the neck without benefit. A right frontal craniotomy had failed to reveal the aneurysm. Eventually his to the origin of right posterior communicating artery right Internal Carotid Artery was clipped proximal resulting in cure (JPMA: 37: 53, 1987).

Aneurysm of the petrous and cavernous parts of the Internal Carotid Artery is a rare condition, accounting for less than 1% of all intracranial aneurysms. Rupture into the paranasal sinuses leading to severe epistaxis, a potentially serious and occasionally fatal condition, is even less frequent. Delens was first to report on this condition in 1870 in a young man who died of rupture of an Internal Carotid Artery aneurysm following a penetrating injury to the eye. Beadles reported a case of epistaxis from a non-traumatic aneurysm of Internal Carotid Artery. The patient was a 61 year old woman who died from haemorrhage at St. Thomas’s Hospital, London, in 1889. Since then, 70 cases have been reported in world literature. Ninety percent of these cases had a history of trauma, either penetrating or non-penetrating. The history in most cases was short and the events dramatic.

We present a patient with epistaxis from an Internal Carotid Artery aneurysm without a history of trauma and where the illness extended over a period of 10 years.

CASE REPORT
The patient was a 53 year old Middle Eastern male. For 10 years he had suffered from epistaxis from his right nostril. Each incident was preceded by right frontal headache which was relieved following haemorrhage. In the last three years, epistaxis had become more frequent and on occasions haemorrhage had been severe enough to require blood transfusion. There was no history of trauma or infection of the paranasal sinuses.

In 1980, a carotid angiogram had demonstrated an aneurysm of the cavernous part of right Internal Carotid Artery. The right Common Carotid Artery was ligated and a year later the right Internal Carotid Artery was ligated in the neck without benefit. An exploration through a right frontal craniotomy failed to reveal the aneurysm and no further treatment was offered.

In May 1983, a left carotid angiogram showed adequate cross-filling to the right side but no filling of the aneurysmal sac. The patient continued to haemorrhage from his right nostril, once or twice a week. On admission to this department in November 1983, he was anaemic (Haemoglobin 8.2 GIL). His B.P. was 110/70 mm Hg. The fundi showed no abnormality. His visual acuity was normal as were his visual fields. His ocular movements were normal. There was no bruit and no other neurological abnormalities. In the six days between admission and surgery, he had four further episodes of severe epistaxis.
requiring transfusion of six units of whole blood. Tomography demonstrated erosion of the sphenoid sinus on the right side due to an expanding aneurysm. Digital Vascular Imaging (DVI) showed filling of intracranial part of his right Internal Carotid Artery from the left Internal Carotid and the right posterior communicating arteries. CT scan appearances were highly suggestive of a vascular lesion in the anterior part of the right cavernous sinus, extending into middle fossa and eroding the ethmoid-sphenoid air cell complex.

On the 9th of November 1983, the right sided craniotomy was reopened. No aneurysm sac was visible. The right posterior communicating artery was abnormally large and its origin very close to the point where the Internal Carotid Artery penetrated the dura. A Scoville clip was applied across the Internal Carotid Artery proximal to the posterior communicating artery.

After this procedure, the patient experienced no further episodes of epistaxis.

DISCUSSION

Haemorrhage from aneurysm of the petrous and cavernous parts of Internal Carotid Artery as a cause of epistaxis is a rare event; perhaps so rare that the standard text-books of Otorhinolaryngology hardly ever mention Internal Carotid Artery aneurysm as a cause of epistaxis.

The relative infrequency of haemorrhage from these aneurysms may be due to partial thrombosis within the sac of the aneurysm.

Although epistaxis may follow injury to the anterior ethmoidal artery, such haemorrhage is seldom a threat to life. The Internal Carotid Artery is the most likely source of massive haemorrhage into the nasophraynx. The carotid-cavernous fistula behaves in a more benign fashion.

Haemorrhage originating from the cavernous part of the Internal Carotid Artery may come through a traumatic defect in the wall of sphenoid sinus or as in our case, from gradual erosion of the wall of the sphenoid sinus.

Of the 70 cases reported by McMormick and Beals in 1964 and Mahmoud in 1979, only 10% patients had no history of trauma, as in our case. Ninety percent cases were involved in trauma ranging from penetrating bullet wound to closed head injury with fracture of skull involving the sphenoid bone. As the literature shows, this condition frequently proves fatal due to recurrent massive epistaxis. An urgent ‘trapping’ procedure is required to isolate the aneurysm. Our patient was fortunate that despite recurrent haemorrhage, increasingly severe in the last months, he survived some 10 years until finally receiving effective treatment.

REFERENCES