Intracavernous Compression of the Third Nerve by an Extracavernous Carotid Aneurysm*

Case Report

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The sudden onset of a third nerve palsy, especially when accompanied by retro-orbital headache, suggests the diagnosis of internal carotid aneurysm at or near the junction of the posterior communicating artery. Rarely, an isolated oculomotor palsy may be caused by an aneurysm of the rostral basilar, superior cerebellar, or proximal posterior cerebral artery. Intact intracavernous aneurysms may compress the third nerve, but usually involve in addition, at least the ophthalmic division of the fifth nerve, if not all of the cranial nerves to the eye. When only one nerve is involved by an intracavernous aneurysm, it is almost always the abducens. Carotid aneurysms originating at the level of the ophthalmic artery are expected to compress the optic nerve, producing loss of visual acuity, defects in the visual fields, and optic atrophy.

These relatively reliable clinical correlations are challenged by this report of a patient whose isolated partial third nerve palsy and retro-orbital pain were produced by an extracavernous carotid aneurysm situated beneath the ipsilateral optic nerve.

Case Report

The patient, a 39-year-old woman, experienced the abrupt onset of left-sided ptosis and retro-orbital headache 7 weeks prior to admission. The ptosis and headache persisted, but she did not develop diplopia, loss of sensation in the face, or symptoms of subarachnoid hemorrhage. Vitamin B_{12} injections failed to improve her condition, and she was referred for neurological evaluation.

Examination. There was moderate ptosis and pupillary dilatation on the left, but the left pupil would react slightly, directly and consensually to light. The extra-ocular movements were entirely normal. Visual acuity was normal as were the visual fields. Corneal reflexes were quite active and symmetrical, and no facial hypalgesia could be elicited. Except for mild hypertension the remainder of the physical examination was unremarkable. Routine laboratory studies, including blood glucose determinations, were also normal. X-rays of the skull with views of the optic foramina showed no abnormalities. Bilateral carotid angiograms demonstrated an aneurysm of the left internal carotid artery arising near the origin of the ophthalmic artery and pointing medially beneath the left optic nerve (Fig. 1). There was no evidence of an aneurysm at the posterior communicating-carotid junction, the usual position for an aneurysm compressing the oculomotor nerve. Retrograde brachial and vertebral angiograms were normal.

Operation. Preoperatively, a Crutchfield clamp was placed on the left internal carotid artery, but the vessel was not occluded. Through a left frontotemporal craniotomy, the aneurysm was visualized beneath the left optic nerve, its dome appeared to be embedded in the dura or bone beneath and medial to the left optic foramen. The carotid artery lay lateral to the optic nerve in its normal anatomical course. The optic nerve could be retracted medially to visualize the upper medial aspect of the carotid artery, and laterally, to expose the aneurysm better. The ophthalmic artery was not seen, but a nerve hook could be passed under the optic nerve without touching the aneurysm. The neck of the aneurysm could not be approached without excessive traction on or possibly division of the left optic nerve; therefore, it was decided to treat the patient by cervical carotid ligation. Prior to closure of the craniotomy, the carotid artery was exposed back to its bifurcation, and no other aneurysms were found. At the same time, the left oculomotor nerve was traced from its entrance into the

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cavernous sinus back to the interpeduncular cistern, and no pathologic conditions were evident.

Postoperative Course. After waiting for postoperative edema to subside, gradual occlusion of the left internal carotid artery was accomplished. The patient had almost immediate reversal of her partial third nerve palsy, and was discharged from the hospital without neurological deficit.

Discussion

The initial clinical impression of internal carotid aneurysm was confirmed, but the unusual position of the lesion was a surprise. When carotid angiography demonstrated a medially pointing carotid aneurysm beneath the left optic nerve, the mechanism by which the third nerve palsy might be explained was obscure. The possibility that the carotid aneurysm was an incidental finding and that some other aneurysm or pathologic process accounted for the oculomotor palsy, could not be dismissed. Thus, angiography of the vertebral-basilar circulation was of paramount importance, certainly before considering cervical carotid occlusion as a therapeutic measure. Moreover, the not unusual occurrence of aneurysms that do not fill at the time of angiography led to some unrest.

After exploration of the entire intradural course of the carotid artery and the oculomotor nerve to the interpeduncular cistern, and after visualization of the vertebral-basilar circulation angiographically, we still were unable to explain the relationship between the aneurysm and the oculomotor palsy. Subsequent repeated neurological examinations by several observers verified that the optic, abducens, or trigeminal nerves were not involved. The fact that the third nerve palsy promptly cleared after occlusion of the cervical carotid artery lent credibility to a causal relationship between the palsy and the unusually placed aneurysm.

Dissections of the cavernous sinus in cadavers exposed a possible mechanism for the oculomotor palsy. The carotid aneurysm pointed medially in its subclinoid but extra-cavernous position, pressing against bone, and laterally displaced the distal intracavernous part of the carotid artery. As shown in Fig. 2, slight lateral displacement of the most distal portion of the intracavernous carotid artery will impinge upon the intracavernous course of the third nerve, and will affect none of the other cranial nerves. The fact that ptosis and pupillary dilatation occurred without involvement of the extraocular muscles defies anatomical analysis, since both the superior and inferior divisions of the third nerve in the cavernous sinus must have been compressed. In view of the extensive diagnostic studies, the operative findings, and the response to cervical carotid ligation, it is concluded that the aneurysm described

Fig. 1. Left: Anteroposterior view of the left carotid angiogram shows the medially pointing carotid aneurysm at the level of the cavernous sinus. Right: Lateral view of the angiogram reveals only the double density (arrow) at the subclinoid site of the aneurysm.

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