Traumatic Dissecting Aneurysm of Middle Cerebral Artery and Carotid-Cavernous Fistula with Massive Intracerebral Hemorrhage*

Case Report

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CAROTID-Cavernous fistula poses no problem for diagnosis because of the impressive pulsating exophthalmos. In spite of a common belief that the lesion is usually easily and successfully treated by ligation of one or another artery leading to the fistula, the rate of cure is actually less than 50%. Reasons for such poor results are not clear, since the complications of treatment are not frequently reported, but cerebral ischemia and hemorrhage appear to predominate. The former is easily understood, but the pathogenesis of cerebral hemorrhage is not clear. Postmortem analysis of the following case showed an interesting series of lesions which could have accounted for the terminal massive intracerebral hemorrhage.

Case Report

The patient was a 32-year-old man who had suffered a head injury in a mine accident in 1948 at the age of 20. Examination at that time revealed ecchymoses around both orbits, old clotted blood in the right external auditory canal and in the right nostril, bilateral sixth nerve palsies, slight left facial weakness, impaired hearing on the right, and Babinski's signs that were suggestive bilaterally. The spinal fluid pressure was 170 mm, the fluid was xanthochromic and contained 150 mg% protein. X-rays showed a fracture of the skull, possibly extending into the right orbit. While the patient was still in the hospital, he developed proptosis of the right eye associated with a bruit. Shortly thereafter the right internal carotid artery was ligated, following which a bruit developed over the left side, but this gradually subsided.

In June, 1956, the patient, now 28, was examined again when he complained of a pounding sensation in his right ear synchronous with the pulse. Proptosis of his right eye had remained unchanged in the last 5 years, and he still had double vision at times, impaired hearing on the right, and anosmia. Examination in November 1956 revealed pronounced proptosis of the right eye with obvious enlargement of the orbital veins in both the upper and lower lids and with an extremely large and tortuous vein carrying visibly pulsating blood near the medial canthus. The pulsations disappeared following manual occlusion of the right carotid artery above the ligation, but the whizzing bruit over the right globe, and the right frontal and temporal region could not be abolished. There was marked limitation of motion of the right eye, which was able to move only about 30° from the midpoint in any plane.

In February, 1957, a right carotid arteriogram showed complete occlusion of the right internal carotid artery; the contrast medium in the right external carotid artery passed through the orbital-ophthalmic system to enter the fistula in the right cavernous sinus. The right external carotid artery was ligated, but a retrograde injection of medium down the common carotid filled the right vertebral artery and demonstrated a massively enlarged posterior communicating artery feeding into the fistula in the right cavernous sinus.

Operation. In October, 1960, a right retrograde brachial cerebral arteriogram confirmed filling of the cavernous sinus from the markedly enlarged right posterior communicating artery (Fig. 1). On October 27, under general anesthesia with controlled hypotension, a right frontal craniotomy was performed with ligation of the intracranial portion of the right internal carotid and ophthalmic arteries. The patient did not regain consciousness. The pupils were dilated and fixed, there were bilateral Babinski's signs, and decerebrate posturing with progressive deterioration. The patient died the following morning at the age of 32, 12 years after the initial accident.
Autopsy. The major pathological findings were confined to the central nervous system. The cavernous sinus and petrous bones were removed. Both petrous bones appeared to be dark red because of numerous tortuous blood vessels inside. The dura over the clivus was densely fibrous and there was a horizontal groove running across the clivus about 1 inch below the dorsum sellae. The groove was about 2 mm deep, lined by overgrown bony prominences with rough surfaces. This lesion was considered to be the site of the old basal skull fracture. The right ophthalmic artery was 4–5 mm in diameter engorged and occluded by a silver clip. The right internal carotid artery was occluded by silver clips at the level immediately distal to the carotid-ophthalmic junction. The proximal internal carotid artery in the neck was completely thrombosed and fibrous up to the siphon immediately proximal to the carotid-cavernous fistulous opening. The right cavernous sinus was bulging, fluctuant and blue, and the dura was tightly adherent to the underlying cranial nerves. The sinus was honeycombed with numerous small blood-filled chambers separated by white fibrous septa. All these small chambers communicated with a large chamber situated medial to the carotid artery and the cranial nerves. The carotid artery opened into this large chamber through a fistula approximately 1 cm in diameter on the medial wall of the internal carotid artery about 2 cm below the anterior clinoid process. The cavernous sinus communicated widely with irregular channels around the sella leading towards the tortuous vessels in the petrous bones.

The brain was markedly edematous, with marked transtentorial herniation on the right side and bilateral tonsillar herniation. There was a moderate amount of blood on the orbital surface of the right frontal lobe. The major blood vessels at the base of the brain showed no atherosclerotic changes. Both posterior communicating arteries were larger than the right internal carotid artery. The right anterior choroidal artery was actually larger than the normal sized right middle cerebral artery. About 2 cm from its origin, however, the right middle cerebral artery tapered down to a fine, firm, solid white strand for about 4-5 cm (Fig. 2); distal to this point it gradually resumed its normal size. Numerous perforating branches derived from the most proximal 2 cm.

There was a fresh hematoma of 200–300 cc in the right cerebral hemisphere, destroying the internal capsule, putamen, globus pallidus, and displacing the insula laterally. The cavity created by the hematoma was 5–6 cm in diameter coronally and 12–13 cm long anteroposteriorly. It had ruptured into the right lateral ventricle, and both lateral ventricles were filled with blood clots and displaced to the left side (Fig. 2). The tegmentum of the midbrain was soft, with small punctate hemorrhages. The right cerebral peduncle was