Giant Aneurysm of Middle Cerebral Artery

A Case Report

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The purpose of this communication is to report a so-called “giant” aneurysm of the middle cerebral artery. Previous reports of this condition as cited by Walton20 are those of Kraus,11 Stehbens,19 Schmidt,18 and Richardson and Hyland.16 Such lesions tend to occur in all locations on the circle of Willis and have been reported in the basilar-vertebral systems,5,15,22 on the middle cerebral vessels,19 on the internal carotid, and on the anterior cerebral vessels.4 The size of an aneurysm qualifying it to be called “giant” has varied in the range of 7×5×5 cm. (7×7×6.2 cm., 19 7×5×5 cm., 4 6×5×5 cm., and 5.5×4×2 cm.2). The aneurysm in the case reported herein measured 8.5×5.5×5 cm., and involved the middle cerebral artery in a 47-year-old male.

Case Report

#21763-63. E.N., a 47-year-old, left-handed male, was admitted in April 1963 to the New York University Neurosurgical Service at Bellevue Hospital with the chief complaint of progressive loss of vision and mental depression for 5–6 months. The patient had worn glasses since childhood. Eighteen months before admission he began to have intermittent episodes of dizziness lasting 3–4 minutes associated with nausea and blurred vision. Because of the above complaints, 9 months prior to the present admission he was hospitalized elsewhere and is reported to have demonstrated left-sided weakness. Roentgenograms of the skull, and cerebrospinal fluid were normal. Electroencephalogram showed abnormal bilateral slow activity, more pronounced in the right frontal region. The patient left the hospital without further studies.

For the last 6 months he had progressive decrease in vision, headaches, and progressive mental depression with fatigue and inability to concentrate, causing him to lose his job as an accountant.

Examination. He was fully awake, alert, cooperative and oriented to time, place and person, but showed short periods of depression and deficit of memory. There was no aphasia. The left pupil was larger than the right, both reacting to light and in accommodation. The fundi showed bilateral papilledema of 2–5 D. and hemorrhages in the left fundus. The corrected visual acuity was decreased in both eyes: 20/200 O.D., 20/400 O.S. No visual-field defect could be demonstrated. The extracocular movements were intact with moderate proptosis of the right eye. There was a left facial weakness of the supranuclear type, and a mild but definite left-sided motor weakness.

Course. The results of blood studies including hemogram, sugar, electrolytes, and serology were normal. Urine was normal as was the electrocardiogram. Roentgenograms of the chest and skull were negative; the pineal gland was not visualized. Because of signs of increased intracranial pressure, it was decided not to do a spinal puncture.

Bilateral serial carotid angiograms performed on April 25, 1963 demonstrated the presence of a large globular aneurysm of the right middle cerebral artery with a marked shift of the right internal carotid and right anterior cerebral arteries to the left side as seen in the anterior-posterior projection (Figs. 1 and 2). The whole main trunk of the right middle cerebral artery was dilated in a tortuous fashion (Fig. 3). The left carotid angiogram demonstrated 90 per cent stenosis of the internal carotid artery at its origin in the neck, and the same shift of the midline structures intracranially caused by the lesion in the right hemisphere. These studies, in addition, showed marked slowing of the intracranial circulation. There was no evidence of spasm of the intracranial vessels.

In the immediate post-angiographic period, the patient was talking coherently and following commands; his mild left hemiparesis remained unchanged. About 4 hours later he suddenly deteriorated and became stuporous, responding only to painful stimuli. At this time there was a marked right hemiparesis including the face with a loss of power of about 80–90 per cent, and bilateral Babinski’s sign was present. His papilledema became worse with more hemorrhages. The preoperative diagnosis was ruptured intracranial aneurysm of the middle cerebral artery with adjacent hemotoma producing ipsilateral pyramidal-tract signs by contralateral compression of the peduncle.

Operation. On April 25, 1963, under general endotracheal anesthesia with the use of intravenous urea and hypothermia, a right frontal (Frazier) flap was turned. The dura mater was found to be tense, and the brain was under moderate pressure but not bulging. A small subarachnoid cyst, approximately 3×3 cm., containing clear, xanthochromic fluid was evacuated anterior to the right temporal lobe. In spite of the use of urea and hypothermia, it was felt that the decompression was insufficient to approach the aneurysm, and hence a partial frontal lobectomy was carried out. At a point 4 cm. posterior to the frontal pole and at a depth of 1.5 cm. a solid, well delineated, nonpulsatile, encapsulated mass was found. On further exposure, the estimated size of the mass was 7×6×3 cm., lying over the sphenoid ridge. At this stage, a faint transmitted pulsation could be felt and it was possible to move the mass gently only in an anterior-posterior direction.

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Needling the mass did not produce any blood; therefore an incision was made through the capsule revealing firm yellowish-grey gelatinous material. A specimen was taken for biopsy. There was no bleeding from the site of the biopsy. The opinion was that we were dealing with an organized, encapsulated and thrombosed aneurysm of the middle cerebral artery which had not bled, and the procedure was terminated.

Postoperative Course. The patient remained unresponsive and moved his left side only in response to painful stimuli. A tracheostomy was performed. Because inadequate explanation had been found for the left cerebral dysfunction and lessening of the level of consciousness, carotid endarterectomy was performed under local anesthesia with an internal by-pass. An atheromatous plaque occluding approximately 50–60 per cent of the lumen of the internal carotid artery was removed. Back flow of blood was moderate and angioplasty was performed.

The course was that of progressive deterioration. Despite supportive measures, the patient expired on the 7th postoperative day.

Autopsy. #50441. The brain weighed 1410 gm. The dura mater over the right frontal lobe was covered by a 2 mm. layer of a recent blood clot. The posterior inferior portion of the right frontal lobe removed at operation left a shallow defect which measured 6.5 cm. across. In the above defect was an ovoid, smooth-surfaced structure which occupied the greatly expanded right Sylvian fissure and was partly covered by the markedly atrophied temporal lobe (Fig. 4). This structure had displaced the remaining portions of the homolateral orbital gyri for 1.0 cm. to the left and herniation of the midportion of the right cingulate gyrus was present. On dissection of the arteries at the base of the brain, the above-described structure proved to be an aneurysm which measured 8.5X5.5X5.0 cm. (Fig. 5), and involved the right middle cerebral artery almost in its entirety.

On sectioning, the wall of the aneurysm was composed of dense, extensively calcified, fibrous tissue and measured in areas up to 3 mm. in thickness. Its lumen was occupied largely by layered, pinkish-grey to almost