THALAMIC ANGIOMA AND ANEURYSM OF THE ANTERIOR CHOROIDAL ARTERY WITH INTRAVENTRICULAR HEMATOMA

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Vascular anomalies in the basal ganglia and thalamus have been the cause of convulsive disorders, multiple neurological deficits and sudden death. Prior to the use of contrast studies, the vascular nature of these catastrophes could only be suspected, but their etiology and often their localization remained undetermined until the postmortem examination. With the advent of angiography and air studies, they can be well localized, their feeding vessel demonstrated, and their relationship to the ventricular wall and surrounding structures determined.

Since Gordon's investigations on ventricular hemorrhage as a "symptom group" and Steel's suggestion of a typical syndrome for ventricular hemorrhage (pin-point pupils, bilateral rigidity, bilateral Babinski sign, unconsciousness and death in 12 hours), considerable interest has been shown in the pathologic physiology of intraventricular hemorrhage as well as the validity of a typical clinical syndrome. Thompson et al. attempted to correlate the pathologic findings with the clinical course of their patients and suggested four syndromes which would indicate intraventricular hemorrhage.

Paterson and McKissock pointed out that patients with central angiomas (involving thalamus, basal ganglia, internal capsule and midbrain) tend to present a progressive hemiparesis and that they rarely presented complaints referable to epilepsy (1 out of 9 cases) or periodic migrainous headaches (0 out of 9 cases). It was further shown by Crawford and Russell that in their Group II of vascular anomalies (deep hamartomas) a large and fatal intraventricular hemorrhage is the frequent outcome because of their proximity to the ventricular system.

The case presently reported does not fit exactly into any of these categories, but presented a picture compatible with subarachnoid hemorrhage and intracerebral hematoma rather than intraventricular hemorrhage.

CASE REPORT

The patient, a 34-year-old, right-handed male, was in good health until June 11, 1956. He was suddenly awakened at midnight by a severe right-sided headache which radiated behind his right eye. Almost immediately he became restless, confused, and disoriented, and began to flail his right arm around wildly. This was followed approximately 15 minutes later by a grand-mal type of generalized convulsive seizure. Postictally it was observed that he could not move his right extremities, nor could he be aroused. The patient was taken within the hour to a local hospital, where he was given sedation and then was transferred to the Veterans Administration Hospital, Houston, Texas, where he arrived 5 hours after the onset of his illness.

Examination. He was a well-developed, well-nourished, stuporous white male who responded to painful stimuli by withdrawal of his right extremities. Temperature was 98.6°F., pulse rate 76/min., respiratory rate 20/min., and blood pressure was 140/64 in the right arm. There was marked nuchal rigidity and he had a left central type of facial paralysis. The pupils were small and reacted minimally to light. The optic fundi showed only slight venous engorgement. A left hemiplegia was present and the myotatic reflexes were slightly hyper-
active in the left lower extremity. There was a sustained left ankle clonus, and superficial abdominal and cremasteric reflexes on the left were absent. Sensory examination, although not satisfactory, suggested a left hemihypesthesia. Babinski, Chaddock, Gordon and Oppenheim’s signs were present on the left. Kernig’s and Brudzinski’s signs were present.

Course. A lumbar puncture was performed on June 11, 1956 (9 hours after onset of illness). The opening pressure was 200 mm. of fluid, and the fluid was grossly bloody. The cellular content of the fluid was: red blood cells 1,760,000; white blood cells 2,310 with 83 per cent polymorphonuclear leucocytes and 17 per cent lymphocytes.

Because of respiratory difficulties a tracheostomy was performed on June 12, 1956. By June 18, 1956, the patient was able to carry on some conversation but remained generally confused. A lumbar puncture that day showed an opening pressure of 300 mm. of fluid, which was grossly bloody. On June 20, 1956 a left homonymous hemianopsia was revealed. Lumbar puncture on this date showed an opening pressure of 240 mm. The fluid contained: red blood cells 12,280; white blood cells 6,900 with 86 per cent polymorphonuclear leucocytes and 14 per cent lymphocytes; the supernatant fluid was xanthochromic. On June 25, 1956, electroencephalogram was interpreted as being grossly abnormal, and diffusely slow with almost no normal frequencies and with questionable lateralization to the right.

Bilateral percutaneous carotid arteriography on June 26, 1956 revealed a small deep aneurysm on the right side, although the vessel supplying it could not be determined. The patient continued without further improvement with a complete left hemiplegia. On July 10, 1956 ventriculography was carried out. Xanthochromic fluid was obtained from the left ventricle; however, the fluid from the right ventricle was blood-tinged. The ventriculogram revealed a mass bulging from the right ventricular wall near the foramen of Monro. In addition the lateral ventricles were dilated. A right carotid arteriogram was then performed so that the relationship of the aneurysm and ventricular system could be ascertained. The aneurysm was deep in the right thalamus, supplied by a branch of the anterior choroidal artery (Figs. 1 and 2).

Operation. On July 20, 1956, a right frontoparietotemporal craniotomy was carried out under hypotensive anesthesia. A cortical incision through the posterior aspect of the superior and middle frontal convolutions anterior to the motor strip was made, allowing entrance into the dilated right ventricle. A clot, measuring 1 X 1.5 X 3 cm., was seen to be attached to the floor of the right ventricle. Since it extended across to block the posterior aspect of the foramen of Monro, the clot was partially removed. At the point of attachment of the clot to the floor of the right ventricle was an area of infarcted and disrupted tissue which involved much of the body of the caudate nucleus and thalamus and extended into the internal capsule. The aneurysm was not identified, but several vessels in the area of the original hemorrhage and infarct were clipped. During surgery, a small portion of the roof of the third ventricle was inadvertently opened. A rubber catheter drain was left in the right lateral ventricle.

Postoperative Course. During the 1st postoperative day, the blood pressure fluctuated between 154/60 and 184/80, the pulse rate from 96 to 104/min., and the respiratory rate from 18 to 26/min. On the 2nd postoperative day the patient vomited a small amount of “coffee-ground” material at 8:00 A.M. and was found to have a paralytic ileus. His pupils were dilated and fixed at 3:00 P.M. Cheyne-Stokes respirations started. The ventricular catheter was opened and 30 cc. of xanthochromic fluid were removed. External ventricular drainage was instituted, resulting in transient respiratory improvement. A lumbar puncture at this time revealed an opening pressure of 103 mm. and no evidence of block. At 3:20 P.M. on the 3rd postoperative day, respirations stopped, and the patient was placed in an Emerson lung, where he remained until death on the 4th postoperative day.

Autopsy Findings (N-221-56). Examination was limited to the head.

Grossly the brain showed hemorrhagic destruction of the right lateral thalamus, and areas of softening and cystic change in the globus pallidus and internal capsule. The body of the right lateral ventricle was slightly depressed because of herniation of the cingulate gyrus from right to left. The right temporal horn was dilated, and the left temporal horn was narrowed. There was a slight shift of the third ventricle to the left. The ependyma of the