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 catastrophies 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 abdomi-
nal and cremasteric reflexes on the left were absent. Sensory examination, although not satisfactory, suggested a left hemihypalgesia. Babinski, Chaddock, Gordon and Oppen-
heim’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 ill-
ness). 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. Lum-
bar 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 ad-
dition 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 su-
perior and middle frontal convolutions anterior to the motor strip was made, allowing en-
trance into the dilated right ventricle. A clot, measuring 1×1.5×3 cm., was seen to be at-
tached 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 be-
tween 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
ventricular system was blood-stained. A hematoma, which was partially adherent to the ependyma, arose from the floor of the left lateral ventricle in the thalamic area. Small hemorrhages were present in the pons, posterior hypothalamus, and the rostral midbrain.

Serial sections were prepared from paraffin blocks of the thalamus containing the aneurysm and stained with hematoxylin and eosin, trichrome, and van Gieson's technique for collagen. The aneurysm, which was 0.3 cm. in diameter, had fibrous walls varying from 200 to 1000 microns in thickness. The lumen was filled with a recent thrombus containing numerous strands of fibrin and leucocytes, especially at the periphery. At the thickest portion of the aneurysmal wall, two small lumina were located. The smaller curved dorsally through the wall of the aneurysm and entered a small group of vessels which coursed medially around the dorsal edge of the aneurysm toward a cluster of about 30 sinusoidal vessels, occupying an area about 1.5 mm. in diameter (Fig. 3). The larger channel coursed laterally and joined a group of dilated vessels whose subsequent course could not be identified (Fig. 4). There was no evidence of rupture of the aneurysm. Five mm. ventral to the aneurysm, a third cluster of thin-walled sinusoidal vessels was identified. Throughout this entire area numerous hemosiderin-containing macrophages were seen, which were especially numerous in the center of the angiomatous masses. Perivascular infiltration, consisting chiefly of lymphocytes, was located in the internal capsule. Foamy gitter cells and remnants of ischemic neurons were scattered throughout the softened area in the globus pallidus.
Fig. 2. In this view the anterior choroidal artery can be identified as the parent vessel of the thalamic aneurysm. In none of the views could the angiomas be identified.

DISCUSSION

This case is of interest particularly because of the rarity of an intrathalamic aneurysm and the association of several angiomatous clusters in the same area. Until postmortem examination, the intraventricular hemorrhage was thought to have been caused by rupture of this aneurysm. However, after careful serial sections, the aneurysm was seen to be intact, and the intrathalamic and intraventricular hemorrhage was apparently from the adjacent angiomatous malformations.

It should be emphasized that deep angiomas and aneurysms of the anterior choroidal artery might be considered as occasional sources of subarachnoid, intracerebral or intraventricular hemorrhage in addition to the rupture of atheromatous vessels, aneurysms and angiomatous malformations in the more usual sites, and miliary aneurysms of the choroid plexus.

Bleeding from the small thalamic angioma, rather than the usually more massive and disastrous hemorrhage from an aneurysm, could account for the discrete hematoma that was removed at surgery. It has been pointed out by Carton and Alvord that the bleeding leading to the formation of localized clots (those smaller than the volume of one ventricle) is probably the result of bleeding in small amounts and under low pressure.

At ventriculography the ventricular system was seen to be dilated. Paterson and McKissock noted that deep lesions may result in a progressive internal hy-
drocephalus caused by "clot formation within the ventricles" or by an "obstructive arachnoiditis around the exit foramina." Because of the encroachment and partial obstruction of the foramen of Monro this clot was resected at surgery.

This case further demonstrates that slight bleeding with diffusion into the ventricular fluid and clot formation can be tolerated as evidenced by the period of improvement following the initial episode of bleeding.

The advantage of doing a combined contrast study under certain circumstances is well demonstrated in this case. The ventriculogram outlined the dilated ventricular cavity and the attached clot, while the angiogram showed the relationship of this aneurysm to these structures as well as its parent vessel, the anterior choroidal artery. In this way, a fairly precise localization of this aneurysm in the substance of the right thalamus was made. However, the small angiomas were not recognized until microscopic examination of the brain. As has been pointed out by Crawford and Russell, some of these cryptic hamartomas are so small that they are not detected and fail to fill during angiography. It is emphasized, as suggested by Crawford and Russell, that serial angiography would be more apt to demonstrate these smaller angiomas.

**SUMMARY**

1. One case of thalamic angioma and an aneurysm of the anterior choroidal artery associated with intraventricular hemorrhage has been presented.

2. Small deep angiomas are often difficult to demonstrate and may be missed on routine study. The value of combined contrast studies and serial angiography in indicated cases is emphasized.
3. Although an aneurysm was demonstrated by angiography, microscopic study showed no rupture of this malformation. The intraventricular bleeding and clot formation were most likely from the thalamic angioma.

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REFERENCES