Dense Calcification in a Large Cavernous Angioma
Case Report

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Cavernous angiomas are important vascular malformations in the central nervous system in that, as space-occupying lesions, they may produce a wide range of clinical symptoms, or, as vascular anomalies, they may spontaneously and catastrophically rupture. They are potentially curably by surgical removal, with less surgical risk from interruption of cerebrovascular supply than most other vascular malformations, since there are no large feeding arteries or draining veins. The variation in size, location, and radiological findings make the diagnosis of cavernous angiomas confusing.

Case Report

A 30-year-old unemployed farm laborer was transferred to the Palo Alto Veterans Administration Hospital in July, 1967, for evaluation of left leg tremor. In 1947, at the age of 10 years, the patient had a severe, acute, febrile illness with headache, stiff neck, and coma for about 7 days. Recovery had been slow but complete, and a diagnosis of meningitis had been made.

In 1961, 14 years later, the patient developed progressive contracture of the left leg until he was walking on his left tiptoes. In January, 1963, focal seizures in this leg led to hospitalization at Fresno, California. Significant findings at that time were left calf atrophy, a coarse resting tremor and spasticity of the left leg, a visual field defect, and retinal scarring consistent with old chorioreinitis, a right frontotemporal electroencephalographic focus, and calcification in the right temporal region (Fig. 1 left). A pneumoencephalogram was unsuccessful. A right carotid arteriogram was interpreted as normal. A ventriculogram was performed, with visualization of the left but not the right ventricle. He was then discharged to receive physical therapy as an outpatient and declined further investigation.

Over the ensuing 4 years the tremor became an explosive and disabling phenomenon. In June, 1967, he was admitted to the Fresno Veterans Administration Hospital and was subsequently transferred to Palo Alto for neurosurgical investigation.

Examination. The patient was a husky, slow speaking, but alert and intelligent man in no apparent distress except for inability to halt a violent tremor of the resting left leg. Bilateral posterior parietal burr holes were palpable. Left visual acuity was impaired to 20/40, and the left visual field was markedly constricted with preservation of only central and some inferior vision. There was moderate scarring in the left retina with pigmented deposits medially and about the disc margins, and fresh hemorrhages at the inferolateral disc margin. There was marked left leg and thigh atrophy with left Achilles shortening. There was generalized hyperreflexia and hypertonia, more marked on the left, without clonus or extensor plantar sign. The left leg tremor stopped during purposive movement, when attention was diverted, and during sleep. There was a mild left central facial paresis.

A brain scan with 7 mc of Technetium99m revealed a very large area of increased uptake (11 × 11 × 9 cm) in the right frontotemporoparietal region (Fig. 2); rapid sequence scintiphotos showed well-defined major arteries on the left but a very poorly-defined right middle cerebral artery on the right. Also, there was markedly diminished flow seen both early and late on the right side, with no paradoxical late filling (Fig. 3). Skull x-rays revealed an extensive area of calcification in the right temporoparietal region.
region (Fig. 1 right). Electroencephalography (EEG) showed slow waves over the entire right hemisphere, with focal abnormality in the right frontotemporal region; there appeared to be no consistent relationship of the involuntary movements of the left leg to the focal EEG abnormality; when the patient was asleep the left leg tremor was absent, but the focal EEG abnormality was unchanged.

Bilateral carotid arteriograms revealed a shift of the anterior cerebral artery system 3 mm to the left, and a large avascular deep frontal mass (Fig. 4 left). A pneumoencephalogram was performed with an opening pressure of 250 mm; spinal fluid protein was 76 mg%. The third ventricle was displaced 13 mm to the left. There was some filling of the right occipital horn and partial interventricular foraminal obstruction with no filling of the right temporal horn. The left lateral ventricle was dilated. The right lateral ventricle was extremely narrowed, and the right cisterna ambiens was compressed (Fig. 4 right).

Operation. On July 26, 1967, a right frontoparietal craniotomy was performed. A calcified, rubbery, purplish-brown, knobby, subcortical right frontotemporoparietal mass was found extending into the right lateral ventricle and basal ganglia. Although the mass appeared to be adherent to the surrounding neural tissue, it could be rather easily separated from it by blunt dissection. An estimated 75% subtotal removal was accomplished with the cutting electrocautery loop and curettage; hemorrhage was difficult to control, and at one point responded only

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Fig. 1. Skull x-rays showing increase in punctate calcifications of right deep frontal region between November 1962 and July 1967. Left: Fresno County General Hospital, November, 1962. Right: Palo Alto Veterans Administration Hospital, July, 1967.

Fig. 2. Brain scan, Tc$^{99m}$, right lateral view, showing a very large area of increased uptake in the frontotemporal region.