MASSIVE CEREBRAL INFARCTION PRODUCING VENTRICULOGRAPHIC
CHANGES SUGGESTING A BRAIN TUMOR

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Thrombosis of intracranial arteries is not ordinarily associated with a rise in intracranial pressure. Edema and swelling may occur with cerebral softening for a period of time, but usually they do not produce symptoms of intracranial pressure. The following case presented a difficult clinical problem. While parts of the history suggested a vascular occlusion, other aspects aroused a strong suspicion of intracranial neoplasm. As the patient’s condition became more precarious, it was felt that the latter diagnosis should be excluded. Procedures with the least risk were carried out first, but later it appeared mandatory that a more radical course be undertaken.

CASE REPORT

No. 187453. E.C., a white female aged 59 years, was admitted because of inability to use the left arm and leg. During a previous hospital stay in February, 1950, it had been disclosed that she had mild diabetes mellitus, mild arteriosclerosis, a blood pressure of 160/100, and a history of transient diplopia.

The present illness began about May 1950, when the patient experienced numbness and paresthesias in the tips of the fingers of the left hand. Shortly thereafter fine movements of the hand became impaired and she had difficulty in sewing. This became more pronounced with time and later she often dropped her fork while using it. Weakness of the left leg appeared during the following month and at times she had difficulty in walking and getting out of a chair. The night before admission, she attempted to get up from a chair but fell to the floor, almost completely paralyzed. After about 10 minutes voluntary motion reappeared in the right arm and leg and with help she managed to stagger to bed, using the left leg slightly. During the night she was somewhat disoriented and attempted to get out of bed and again fell to the floor because of weakness. The next day there was complete left-sided paralysis, with facial involvement. The sensorium was intact. She was alert and able to make known her wants. Her daughter reported that the patient had lost 40 pounds during the past year and had not been following her diet properly.

Examination. She was a thin but well developed white woman, lying quietly in bed, who showed obvious loss of weight. B.P. 150/100; pulse 84; respirations 20, regular and even. There was abundant evidence of generalized arteriosclerosis. The optic discs were flat, well outlined and of good color, the vessels showing moderate arteriosclerosis. There were no visual field defects on gross tests. There was left facial paralysis of central type and the tongue deviated slightly to the right. Except for ability to flex the fingers slightly, the left arm and shoulder were paralyzed. The patient was able to raise and lower the left leg while lying in bed but movements were weak and could not be performed against gravity. There was no increase in tone. The reflexes on the left were hyperactive. Clonus in the left ankle, a Hoffmann reflex in the left hand, and Babinski response in the left foot were easily elicited. No gross sensory changes were present. Cerebellar tests could not be adequately carried out but there was nothing to suggest a cerebellar disturbance.

X-rays of the skull disclosed an osteoma in the left parietal region; this was regarded as benign and asymptomatic. The pineal was displaced to the left about 3 mm.

Laboratory Data. Urinalysis showed only a faint trace of sugar and no albumen. Fasting
blood sugar was 181 mg. per cent; blood urea was 24 mg. Hb., RBC and WBC were not remarkable; Wassermann was negative. Lumbar puncture was done; CSF pressure was 82 mm. of fluid; protein was 33 mg. per cent; Wassermann was negative; there were no cells.

The first impression was that the patient probably had a vascular accident. However, in view of the history of slow progression of symptoms, it was felt that she might have a neoplasm into which a hemorrhage had occurred.

Course. Since the patient was in fair condition, it was thought best to do an arteriogram. The percutaneous method was used. We were unable to force any dye beyond the junction of the carotid and middle cerebral arteries (Fig. 1).

Following this, the patient became very much worse. She recovered consciousness slowly from the small dose of pentothal given. The hemiplegia became more profound within a few hours. She lapsed into stupor but could be roused with difficulty. On the next day there was further deterioration and the right pupil became enlarged and fixed to light.

In view of the pronounced pineal shift, the diagnosis of neoplasm appeared as probable as that of thrombosis, so it was decided to perform ventriculography. Bilateral occipital trephines were done, during which time breathing stopped. The left ventricle was entered without difficulty and fluid gushed through the needle under considerable pressure, following which spontaneous respirations were resumed. There was a marked ventricular shift to the left suggesting a mass in the frontoparietal region of the right cerebral hemisphere. A massive thrombosis producing edema was considered but a large intracranial neoplasm seemed more likely (Figs. 2, 3 and 4).

Operation. A right-sided flap was turned down under local anesthesia. The brain was under moderate tension but almost entirely necrotic. The blood vessels in the Sylvian fissure were thrombosed along their entire course. A large part of the white matter of the frontal, parietal and anterior temporal regions was removed without causing any bleeding whatsoever. The anterior cerebral artery, too, was thrombosed along its complete length. There was no bleeding from the cortex until the region of the junction of the lateral and temporal horn was reached, where the brain is supplied by the posterior cerebral artery. The area of necrosis extended down into the basal ganglia and into the corpus callosum.