MASSIVE CEREBRAL INFARCTION PRODUCING VENTRICULOGRAPHIC CHANGES SUGGESTING A BRAIN TUMOR

ARTHUR B. KING, M.D.
Guthrie Clinic, Sayre, Pennsylvania
(Received for publication March 26, 1951)

Thrombosis of intracranial arteries is not ordinarily associated with a rise in intracranial pressure. Edema and swelling may occur with cerebral softenings 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. 137453. E.C., a white female aged 59 years, was admitted because of inability to use the left arm and left 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
CEREBRAL INFARCTION SUGGESTING BRAIN TUMOR

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).

Fig. 1 (left). Right carotid arteriogram, showing thrombosis at junction of middle cerebral and carotid arteries. Note reflux filling of vertebral artery.

Fig. 2. (right). Ventriculogram, A-P projection, showing compression and deviation of right lateral and third ventricles.

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.
Postoperative Course. The patient's condition remained precarious and although she survived for approximately 48 hours, her course was downhill and she died without regaining consciousness. The right pupil during this time had again become equal to that on the left and once more responded to light.

Postmortem Examination. The internal carotid artery was thrombosed as it emerged from the cavernous sinus. The entire length of the right anterior cerebral and middle cerebral systems was completely occluded. A prominent cerebellar pressure cone was present. Multiple sections of brain showed softening which extended over almost the entire right hemisphere except for the portions of occipital lobe, posterior parietal and temporal lobes supplied by the posterior cerebral artery. The necrosis extended to the right basal ganglia and down into the mid brain. It was softening in this region that undoubtedly produced the final outcome.

Fig. 3 (left). P-A projection showing obliteration of temporal horn and posterior portion of lateral ventricle. The third ventricle is markedly shifted and compressed.

Fig. 4 (right). This view shows normal left ventricle and benign osteoma of skull.

DISCUSSION

This case is presented as an example of how an extensive thrombotic softening with swelling of the necrotic tissues may mimic a tumor. When the history, physical findings and x-ray studies appear to point toward a slowly developing intracranial neoplasm rather than a cerebrovascular accident, the problem of differential diagnosis is even more difficult. A hemianopsia was not present when the patient was first seen, but it must have developed later as the softening progressed. The changes in the eyegrounds seen in some cases of thrombosis of the internal carotid artery when the ophthalmic artery is also occluded, as noted by King and Langworthy, were not present in this patient. At necropsy the thrombosis in the carotid had not extended that far inferiorly to involve the orifice of the ophthalmic artery.

From the clinical history one would suspect that the thrombosis was a slowly developing affair, at first producing mild symptoms until finally the last remnants of collateral circulation disappeared. There was no possibility of large parts of the hemisphere being supplied with blood since all the collaterals had been thrombosed. The edema resulting from the necrosis undoubtedly produced the signs of intracranial hypertension and exerted pressure on the hypothalamus and mid brain, causing additional softening in these areas. Although shift of the pineal was noted
in x-ray, lumbar puncture at the time showed a spinal fluid pressure of only 82 mm. of fluid. At operation 24 hours later, however, the ventricular fluid was under great pressure, so probably there was inadequate communication of the intracranial fluid with the lumbar spinal fluid. Despite the low pressure on lumbar puncture, there was adequate evidence of elevated intracranial pressure a few hours later. This was: (a) rapid deterioration of consciousness, (b) shift of the pineal to the left, (c) enlarged and fixed pupil on the right, which later became smaller and reacted to light after removal of brain tissue, (d) bulging of the right hemisphere on opening the dura, despite ventricular drainage, (e) respirations were restored almost immediately after ventricular puncture and were spontaneous for 48 hours after intracranial decompression.

Rarely has shift of the pineal been noted in cerebral vascular accidents, and often it is due to intracerebral hemorrhage rather than edema alone. Such a pronounced shift of the ventricular system as was noted in this case is unique in my experience. A perusal of the literature available does not reveal an exactly similar case although I am certain that the condition has probably been observed before. While the arteriograms indicated a thrombosis of the internal carotid artery, the ventriculographic findings made a neoplasm seem more likely. At operation the intracranial pressure produced a moderate herniation of cerebral tissue through the bony defect upon incising the dura, even after ventricular drainage had been established. The extensive necrosis and the complete absence of bleeding whenever an incision was made into the brain, confirmed the arteriographic findings.

Postmortem studies were considerably hindered since the brain could not be properly injected because of the extensive thrombosis and even after considerable hardening so much of it was necrotic that the extent of the softening into the mid brain and basal ganglia could not be precisely delineated. It could be established, however, that the posterior cerebral was adequately functioning, as were the cerebral arteries on the left side. Microscopic sections disclosed only necrosis. There was no evidence of a neoplasm. Sections from the main cerebral arteries of the right hemisphere confirmed the occlusion. The thrombi were of recent origin and showed no sign of organization.

SUMMARY

The patient had sustained a thrombosis of the right internal carotid artery together with complete occlusion of the anterior and middle cerebral systems on the same side. The resulting arteriographic and ventriculographic changes are presented, and the difficulties in differential diagnosis between a vascular accident and a neoplasm are discussed.

REFERENCE