ENCEPHALOMALACIA SIMULATING THE CLINICAL AND RADIOLOGICAL ASPECTS OF BRAIN TUMOR

A REPORT OF 6 CASES

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And when the time comes to make public one's score, it is done somewhat apologetically, but with the expectation that others may profit by it and with the assurance they will come to improve upon it.—Harvey Cushing

The differential diagnosis between cerebral vascular disease and a space-occupying lesion is a challenge, even to the experienced neurologist. The occurrence of symptoms suggesting an acute cerebrovascular disorder in patients harboring brain tumors is perhaps the greatest source of error. Apoplectiform symptoms, such as occur in cases of glioblastoma multiforme, with its tendency towards intravascular thrombosis and hemorrhage, or metastatic carcinoma, or infection of the brain of embolic origin, may be the first evidence of an expanding lesion and may be indistinguishable from those of cerebral vascular disease.

On the other hand, progressive symptomatology and neurological deficit may occur with generalized cerebral arteriosclerosis, or with occlusion of major intra- or extra-craniial vessels, such as middle cerebral or internal carotid artery. The symptoms of these vascular lesions may be indistinguishable from those of an infiltrating brain tumor. Thrombosis of the internal carotid artery may simulate a mass lesion by its slowly evolving course, with headache, in at least half of the cases, preceding or associated with progressive disability involving one half of the body.

Even more difficult is the diagnosis in those patients whose history and clinical findings may be compatible with either cerebral vascular disease or intracranial neoplasm. Usually supplementary tests, such as cerebral angiography and/or air studies, will clarify the diagnosis. Occasionally, however, massive cerebral infarction will produce radiographic changes compatible with an expanding lesion and craniotomy is performed. The vascular etiology of the process becomes apparent only after thorough exploration and biopsy are negative for brain tumor, as well as from the further evolution of the symptoms.

The relative absence of such reports in the literature has prompted the discussion of 6 patients with encephalomalacia simulating an expanding lesion in whom craniotomy was performed.
ENCEPHALOMALACIA SIMULATING BRAIN TUMOR

CASE REPORTS

Case 1. E.K. (#119945), a 31-year-old female, was admitted to the Methodist Hospital on Sept. 11, 1950, with the complaint of left hemiparesis of 5 weeks' duration. About 6 weeks previously the patient had become drowsy and lethargic, sleeping 18-20 hours a day. She had some domestic difficulties, and when seen by previous examiners, her symptoms were attributed to emotional disturbances. Later, she had an episode of smelling natural gas, accompanied by a spell of moderately severe pain in the right frontal region; this was followed by generalized headache, all symptoms disappearing in approximately 18 hours. The patient was up during the night of August 9 to go to the bathroom, but on awakening the next morning, she was unable to move her left arm and had weakness of her left leg. The left hemiparesis persisted during the month prior to admission.

Examination. Blood pressure was 146/82, pulse rate 82, respiratory rate 20, and temperature 99°F. The heart was slightly enlarged to the left with the pulmonic 2nd sound greater than the aortic 2nd sound. The patient was fairly cooperative, but over-responded to any kind of stimuli. The left arm was completely paralyzed, and the left leg was markedly weak. Myotatic reflexes were increased on the left. Hoffmann and Babinski signs were present. Abdominal reflexes were absent on the left. All modalities of sensation were reduced over the left half of the body. There were poor upward and lateral gaze of the left eye, with diplopia, dullness to pin prick on the left side of the face, left supranuclear facial weakness, deviation of the tongue to the left, and the optic discs showed papilledema of 2D.

Laboratory Data. Hemogram, urinalysis, blood chemistry and serology showed no abnormalities. Spinal fluid pressure was 200 mm. of water; cell count was 10 polymorphonuclear leucocytes; protein was 96 mg. per cent. Roentgenograms of skull and chest were within normal limits. Electroencephalogram (Sept. 13, 1950) revealed a diffusely slow disorganized tracing with a discharging focus in the region of the lower end of the motor area on the right side. The findings were interpreted as compatible with the diagnosis of cerebral thrombosis, but the generalized involvement and the organization of the electric activity was considered more suggestive of a space-occupying lesion, especially glioblastoma.

Comment. A lesion in or near the internal capsule was considered with the differential diagnosis between (1) thrombosis of the middle cerebral artery, (2) encephalitic process, and (3) cerebral neoplasm. It was felt that the first possibility was most likely, but that tumor could not be excluded.

Course. A pneumoencephalogram was performed on Sept. 15, 1950. The anteroposterior projection (Fig. 1) showed dilatation of the lateral and third ventricles with displacement towards the left side; the lateral projections were not localizing. There was some difference in the appearance of the inferior horns on the two sides, but the anteroposterior projection indicated that the lesion was probably not in the temporal lobe, but above this level, appearing to be intermediate in position. A diagnosis of tumor, probably glioblastoma multiforme, was entertained.

Operation, Sept. 15, 1950. A right parietal osteoplastic flap was turned. On opening the tense dura mater near the Sylvian fissure, a 5 cm. fairly well demarcated degenerative area with hemorrhagic and necrotic changes was encountered; this area was removed by suction.

Pathological Report. The specimen consisted of several pieces of mottled pink to red tissue resembling brain in appearance with foci of hemorrhage and necrosis.