Cerebellar Histoplasmoma

Case Report

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Histoplasmosis is a common disease which most frequently affects the pulmonary system. On the basis of positive reactions to skin tests, it has been estimated that from 25 to 30 million people in the United States have had some form of histoplasma infection.

Cooper and Goldstein were able to find only 18 reported cases of involvement of the central nervous system with Histoplasma capsulatum in English medical publications between 1952 and 1960. Even rarer are cases of H. capsulatum granuloma manifested as expanding intracranial neoplasms, and for this reason, the following case is reported.

Case Report

A 42-year-old Negro driller in a salt mine was admitted to Ochsner Foundation Hospital on February 2, 1966. For 3 weeks he had had frontal and suboccipital headaches, more severe on the right side. About 2 weeks before admission, he complained of “dizziness” and “loss of balance.” He would stagger when walking, falling more frequently to the right. He also noted some unsteadiness and clumsiness of his right hand. The patient had traveled within Louisiana but never outside of the state.

Examination. On February 2, 1966, the patient was examined and found to be alert and well-oriented. He held his head in a tilted position with the occiput toward the right. Other abnormalities noted were an apical systolic murmur and hepatomegaly. The oral temperature was 98.6°F.

Neurological abnormalities included prominent nystagmus on vertical and lateral gaze. Moderate hypotonia of the right arm and leg was noted, with slight clumsiness of the right hand. Terminal tremor on finger-to-nose testing with the right hand and heel-to-knee testing with the right foot was present.

The tendon reflexes were generally active, with right ankle clonus. Severe ataxia of gait was noted, with a tendency to fall toward the right. The ataxia was thought to be out of proportion to the degree of incoordination demonstrated in the right arm and leg.

The white blood cell count was 5,250/cu ml, with a differential of 60 segmented cells, 3 basophils, 3 eosinophils, 30 lymphocytes, and 4 monocytes; hematocrit, 49; sedimentation rate, 28 mm/hr; alkaline phosphatase, 86 King-Armstrong units; serum glutamic oxalacetic transaminase, 46 Karmen units; total bilirubin, 0.7 mg/100 ml; bromsulphalein retention, 46%. No abnormalities were detected in roentgenograms of the skull and chest.

Because of the hepatomegaly and the abnormal results of certain laboratory tests, needle biopsy of the liver was performed. On microscopic examination, a heavy infiltration of chronic inflammatory cells was seen in the portal areas, with some extension into the hepatic lobules. The hepatic cells showed a moderate degree of regenerative activity. These observations were thought to be compatible with a diagnosis of posthepatitic cirrhosis.

On February 4, 1966, an electroencephalogram demonstrated biocicipitoparietal delta activity which was sometimes maximal on the left and sometimes spread to the adjacent temporal area on the left. Independent delta activity was noted, however, in the left temporal area, and the possibility of an additional lesion in this region was raised.

Pneumoencephalography was performed on February 14, 1966. The opening pressure was 170 mm of water. No ventricular filling was demonstrated in the roentgenograms. The cisterna magna was obliterated, with both cerebellar tonsils at the level of the atlas.

The cerebrospinal fluid contained 16 white blood cells/cu ml with 6% neutrophils and 94% lymphocytes, protein 43 mg/100 ml, and a first-zone colloidal gold curve. The

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cerebrospinal-fluid Kolmer reaction was negative.

**Operation.** With the patient seated, bilateral suboccipital craniectomy was performed on February 14, 1966. Palpation of the dura disclosed a hard mass in the left cerebellar hemisphere. When the dura over this mass was opened, an area of necrotic-appearing cerebellar tissue was noted. Removal of this necrotic cortex with a sucker exposed a yellowish, hard, nodular mass, 3.5 cm in diameter, which was delivered intact without bleeding. It was thought to be an ordinary brain abscess.

The cyst wall measured 5 mm at its median thickness. Multiple sections contained a central area of necrosis boardered by inflammatory cells. Included within this inflammatory infiltration were innumerable histiocytes which contained myriad microorganisms morphologically typical of *Histoplasma capsulatum*. These were well demonstrated by Gomori's methenamine-silver-nitrate stain (Fig. 1).

**Postoperative Course.** Postoperatively, definite improvement of the ataxia, nystagmus, and dysmetria occurred over a period of weeks. Subsequent examination revealed carious lesions of the upper and lower right molars with an associated indurated area of gingiva and tongue. Biopsy of this area demonstrated *H. capsulatum*.

On February 19, 1966, Amphotericin B therapy was instituted. A total dosage of 2910 mg was administered intravenously and 46 mg intrathecally during a period of 12 weeks in 26 injections.

No other lesions were demonstrated in a brain scan made on March 11, 1966. When the patient left the hospital on May 17, he had no complaints. The scalp over the suboccipital craniotomy pulsated freely. There was only a hint of nystagmus and no residual of ataxia.
Discussion

Shapiro and associates\(^4\) classified histoplasmosis of the central nervous system into three categories, the first two being a meningeal inflammation, most severe in the meninges at the base of the brain, and miliary granulomas seen in association with small veins, and the third being areas of focal destruction of the cerebral parenchyma. They used the term histoplasmosa to describe this lesion and suggested that such a lesion might attain sufficient size to mimic an intracranial neoplasm.

White and Fritzlen\(^5\) reported a case of \(H.\) capsulatum granuloma, which was manifested as a right cerebral expanding lesion. The patient died 2 hours after removal of a \(4\times3\times3\) cm mass from the right frontal lobe. Multiple subcortical nodules were found in the brain at necropsy. Cooper and Goldstein\(^1\) reported a second case in which symptoms suggested cerebellar dysfunction. A single granuloma involving the fourth ventricle and midline cerebellum was found at necropsy. Greer and associates\(^2\) reported a case with manifestations of a cerebral neoplasm. A granuloma 2 cm in diameter was removed from the frontal cortex. Postoperatively, the patient was treated with Amphotericin B and was reported to be well and working two years later. Tveten\(^5\) reported a case with presenting complaints of fever, headache, and diplopia, but no localizing neurological signs. A solitary cerebellar granuloma was found at autopsy.

The foregoing cases, as well as the presently reported case, demonstrate that a histoplasma may simulate an intracranial neoplasm. Apparently, these lesions may be multiple or solitary. In our case the neurologic observations and electroencephalographic tracings suggested the possibility of other areas of cortical damage.

References

5. Tveten, L. Cerebral mycosis. A clinico-pathological report of four cases. \(Acta neurol. scand.,\) 1965, \(41:19-38.\)