Tuberculoma of the brain stem: control of relapses by steroid therapy

Case report

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A tuberculoma is reported, with an acute onset and a recurring course of remissions. The successful use of steroid therapy is discussed.

KEY WORDS: brain stem tumor • tuberculoma • relapses and remissions • steroid therapy

REPORTS of tuberculoma of the brain are now rare except from the developing countries including India.1,3,7,9 Occurrence in the midbrain is particularly rare. We are reporting a case of midbrain tuberculoma with unusual clinical features.

Case Report

This 20-year-old man was admitted to the All India Institute of Medical Science on August 1, 1969, with chief complaints of bilateral ptosis and unsteady gait of 15 days duration; he had had frontal headaches for 1 month. At the age of 6 months he had developed a persistent right hemiparesis following a febrile episode. There was no history of loss of consciousness, seizures, or fever.

First Examination. There was no general physical abnormality, and mental function was normal but slow. Visual acuity was normal and there was no papilledema or optic atrophy. Pupils were moderately dilated but equal; they reacted poorly to direct and indirect light on both sides. There was bilateral ptosis with restriction of eye movements in all directions, more on the right side than the left. Bilateral horizontal nystagmus was present. The right half of the face was small and wasted; there was minimal upper motor neuron facial palsy. The other cranial nerves were normal. There was wasting and atrophy of the right half of the body. Muscle tone was increased on the right side and normal on the left. Deep tendon reflexes were exaggerated on the right side. The right plantar reflex was extensor, the left, flexor. All sensory modalities were intact. Finger-to-nose and heel-to-shin tests were abnormal on both sides. The gait was wide-based, and the patient had a tendency to fall to either side.

Routine blood and urine examinations were normal. Fasting and postprandial blood sugar levels were normal. Serological tests for syphilis were negative. Chest and skull films were normal. Cerebrospinal fluid (CSF) pressure was normal; the fluid contained no cells, 60 mg% of protein, and
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50 mg% of sugar. Electroencephalogram showed poorly organized alpha activity from both sides with paroxysmal slow and high voltage delta activity from both the hemispheres, more so anteriorly and from the left side. A right carotid angiogram was normal. Pneumoencephalogram showed dilatation of both lateral ventricles, more marked on the left side; the position of the aqueduct and fourth ventricle did not suggest a mass lesion.

Due to the sudden onset of symptoms, a possibility of acute demyelinating process was entertained, and the patient was put on prednisone 40 mg daily. He improved considerably in the next 2 weeks; the ptosis and cerebellar signs disappeared completely. He was discharged with the advice to continue the steroids at home.

Second Examination. The patient discontinued the steroid therapy, however, and was readmitted after 3 weeks with bilateral ptosis and ataxia. Examination revealed essentially the same findings as on the first admission, and he was again started on steroids. Once again he made almost complete recovery and was discharged on steroid therapy. Steroids were voluntarily tapered off over the next 6 weeks.

Third Examination. Two weeks after stopping the steroids the patient was admitted for the third time, with dull headaches, drowsiness, and unsteadiness of gait. There was no papilledema, but return of bilateral ptosis, dilated pupils, third, fourth, and sixth cranial nerve palsies, bilateral cerebellar signs, and bilateral Babinski signs. The muscle stretch reflexes were exaggerated, with ankle clonus bilaterally. An air and Myodil ventriculogram was done. There was dilatation of both lateral ventricles, more marked in the left side. The third and fourth ventricles, as well as the aqueduct, were normal in position and size (Fig. 1). The ventricular fluid was clear; there were no cells and the protein content was 20 mg%. Following this procedure the patient developed a high fever, became deeply comatose, and died the next day.

Postmortem Examination. At the partial autopsy only the brain was examined. The midbrain showed a fairly well defined nodular lesion measuring $2.5 \times 2 \text{ cm}$, replacing a large part of the midbrain substance and pushing the aqueduct backward toward the right (Fig. 2). Microscopic examination of the mass showed it to be a tuberculoma consisting of a large central area of caseous necrosis surrounded by inflammatory granulation tissue containing many granulomas. The granulomas were composed mainly of epitheloid cells in the midst of which were scattered giant cells. The pathological diagnosis was tuberculoma
of the midbrain, internal hydrocephalus, cerebral edema, atrophy of the left temporoparietal region, and right thalamic hemorrhage (traumatic).

Discussion

In India the incidence of tuberculomas varies from 8% to 30% of the brain tumors reported. The most common site of the tuberculoma appears to be the cerebellum, and least common, the brain stem. Brain stem tuberculoma constituted 8% of the series reported by Asenjo, et al., while none occurred in the 114 tuberculomas reported by Dastur and Desai. Except for the solitary report of Roedenbeck, which appears to have been based entirely on clinical impression, no specific case of midbrain tuberculoma has been reported.

The clinical course in our case was most unusual because of the acute onset, the absence of any symptoms or signs of raised intracranial tension, and the patient's dramatic recovery with steroid therapy and relapse with its withdrawal. Remissions and relapses have occasionally been described in brain stem gliomas and vascular tumors. The mechanisms of these relapses and remissions have been attributed to necrosis and edema in the tumor itself. Hemorrhage into and around the tumor, and necrosis and edema in the surrounding brain tissue due to the involvement of small blood vessels have also been described.

The effect of steroids on the course of edema was the reason for the almost complete remissions in our patient. Whether the relapses were due to enlargement of the tuberculoma, to necrosis within it, or to recurrence of brain edema is difficult to decide.

References


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