Cerebral Cysticercosis of the Fourth Ventricle: A Problem in Primary Care Diagnosis

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Cerebral cysticercosis, human infection with larvae of the pork tapeworm, can no longer be regarded as a medical curiosity. In areas where cysticercosis is endemic (Latin America, eastern Europe, and Asia), it is the most frequently seen nonneoplastic, space-occupying lesion of the central nervous system,[1] and the most frequent cause of epilepsy in young adults.[2] With increasing immigration from Latin America, cerebral cysticercosis is now diagnosed in many parts of the United States. As physicians of first contact, family physicians are likely to see this illness in its early form. The following case illustrates some characteristic presenting signs and symptoms of cerebral cysticercosis with ventricular obstruction.

Case Report

A 32-year-old Spanish-speaking man who had been acting abnormally, sleeping to excess, and complaining of headache for 36 hours was brought to Mercy Medical Center by his wife. There was no history of trauma, substance abuse, or recent infection. Awake and alert on admission, he was comatose and responsive only to noxious stimuli within one hour. Examination revealed nonreactive pupils, flat optic discs, dysconjugate gaze, and symmetrical deep tendon reflexes with bilateral Babinski signs. The white blood cell count was normal. Lumbar puncture yielded clear cerebrospinal fluid (CSF) with a glucose level of 6 mg/dL (blood glucose, 177 mg/dL), 1,000 red cells per milliliter, and 140 white cells per milliliter (97 percent mononuclear). CSF Gram stain and cultures were negative. A computerized tomographic (CT) scan of the head showed possible ventricular enlargement with loss of cortical sulci; a chest film revealed apical pleural thickening, consistent with old tuberculosis.

The patient was raised in Mexico and had moved to the United States ten years before admission. Two years before admission, he had returned to Mexico, visiting siblings whose tuberculin tests were positive and who were taking isoniazid.

After his CT scan, the patient regained consciousness. A neurologic consultant diagnosed acute tuberculous meningoencephalitis, and a triple drug antituberculosis regimen was started. A neurosurgeon attributed the episode of unresponsiveness to acute hydrocephalus and placed a ventricular drain in the left frontal horn under local anesthesia. Ventriculography performed through the drain showed possible spontaneous rupture of an enlarged CSF channel through the tectal plate into the quadrigeminal cistern. A fourth-ventricle isodense cyst, believed to be a cysticercus, was seen. On the seventh hospital day, following removal of the drain several days earlier, the patient underwent suboccipital craniectomy and exploration of the fourth ventricle. A gelatinous, cystic

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mass was removed from the fourth ventricle; results of the pathologic examination were consistent with cysticercosis. Following surgery, the patient required placement of a ventriculoperitoneal shunt to control persistent hydrocephalus.

Discussion

Epidemiology

Although the incidence of cysticercosis in the United States is unknown, in Mexico it accounts for 11 percent of all general hospital admissions and 35 percent of craniotomies.[1] At one large medical center in Los Angeles, cysticercosis is the leading cause in adults of nonalcoholic seizures and hydrocephalus.[3]

Pathogenesis

Cysticercosis results from infestation by the larva of the tapeworm, Taenia solium. In the worm's normal life cycle, the pig is the larval intermediate host. Man becomes the intermediate host when he ingests water or food contaminated with ova. These ingested eggs do not develop into adult worms. Instead, they migrate into the blood stream and are preferentially deposited in the central nervous system and in striated muscle. Morbidity is caused almost exclusively by central nervous system involvement.[4] Obstruction of cerebral spinal fluid flow may cause acute hydrocephalus, as in this patient. Cysts may also cause intense inflammation and subsequent chronic meningitis.

Clinical Presentation

Cysticercosis should be considered whenever patients from endemic areas experience seizures, acute or progressive focal neurologic deficits, chronic meningitis, or other symptoms consistent with increased intracranial pressure.[5] Family physicians are particularly likely to see subtle, early manifestations. There are no pathognomonic features. Parenchymal cysticercosis is usually associated with epilepsy.[6] Ventricular involvement can result in protean neurologic manifestations, including severe headaches, nausea, and vomiting. Movement of a fourth ventricle cyst may cause positional headache, abrupt vertigo, gait imbalance, and flaccid paralysis.[2]

Diagnostic Procedures

Computerized tomography has the highest yield. In the proper clinical setting, a CT scan showing hydrocephalus, intracranial calcification, or cysts should lead to a presumptive diagnosis of cysticercosis. Cerebral spinal fluid abnormalities, while nonspecific, include a lymphocytic pleocytosis and decreased glucose.

Treatment

Management is directed toward relief of symptoms and treatment of the underlying pathologic condition. Corticosteroids are often used to reduce inflammation and brain edema. Anticonvulsant drugs may be needed. Praziquantel, an anticestodal agent, has shown promise as a safe and effective treatment.[6]

Ventricular shunting is often required to alleviate hydrocephalus. If a single cyst or conglomeration of cysts causes obstruction, as in this case, its removal should provide dramatic relief.[7]

Prognosis

The prognosis of cysticercosis is controversial. Patients often have long remissions between exacerbations, and many die of other causes. The poorest prognosis is found in widely disseminated disease or where there is obstruction of cerebral spinal fluid flow. The long-term results of permanent ventricular shunting await further evaluation.

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