Case Report

Racemose Cysticercosis in the Cerebellar Hemisphere

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Neurocysticercosis is the most common parasitic disease of the central nervous system in humans, caused by infection of the larval stage of the pork tapeworm, Taenia solium. However, cerebellar involvement is rarely reported. We report a case of racemose cysticercosis in the cerebellar hemisphere. A 44-year-old man presented with headache and dizziness. Magnetic resonance imaging showed hydrocephalus and an ill-defined, multiloculated cystic mass with hypointensity on T2-weighted images, hyperintensity on T1-weighted images, and rim enhancement after gadolinium injection. The patient underwent endoscopic third ventriculostomy and the cyst resection was done through a craniotomy. In surgical field, cysts were conglomerated in a dense collagen capsule that were severely adherent to surrounding cerebellar tissue, and transparent cysts contained white, milky fluid. Histological findings confirmed the diagnosis of cysticercosis. He received antiparasitic therapy with praziquantel after surgery. Racemose cysticercosis is rare in the cerebellar hemisphere but neurocysticercosis should be taken into consideration as a differential diagnosis of multiple cystic lesions in the cerebellum.

KEY WORDS: Cysticercosis · Cerebellum · Cyst.

INTRODUCTION

Cysticercosis is the most common parasitic disease of the central nervous system (CNS) in humans, caused by infection of the larval stage of the tapeworm, Taenia solium. Human cysticercosis results from fecal-oral contamination with Taenia solium eggs from a human tapeworm carrier. Neurocysticercosis is common in less developed countries but is increasingly reported in more developed countries because of immigration of people from endemic areas. Clinical manifestations are nonspecific and vary according to locations, number and size of cysts, and host immune response to the cysticerci. The parasites spread through the bloodstream and may locate almost anywhere in the body. In CNS, it frequently involves the cerebral hemispheres, ventricles and basal cisterns, subarachnoid space and spine whereas the most frequent location is in the cerebral hemispheres, mainly at the junction of gray and white matter. Moreover, racemose cysticercosis is common in the cisternal or subarachnoid space. However, cerebellar cysticercosis has been rarely reported. We present a case of racemose cysticercosis in the cerebellar hemisphere.

CASE REPORT

A 44-year-old man presented with headache and dizziness for two weeks. On neurological examination, he was drowsy and showed ataxic gait. He had a previous history of frequent ingestion of pork in his youth. Precontrast computed tomographic (CT) scan showed a large ill-defined cerebellar mass with multiple cysts (Fig. 1A) and contrast-enhanced CT scan revealed heterogeneous enhancement (Fig. 1B). Magnetic

![Fig. 1. Precontrast CT scan showing a large ill-defined cerebellar mass with multiple cysts (A) and contrast-enhanced CT scan revealed heterogenous enhancement (B).](image-url)
resonance imaging (MRI) showed hydrocephalus and an ill-defined, multicystic cerebellar mass with hyposignal on T1-weighted images, hypersignal on T2-weighted images, and rim enhancement after gadolinium injection (Fig. 2). He underwent endoscopic third ventriculostomy and removal of cerebellar mass through a suboccipital craniotomy. The mass consisted of multiloculated cysts, which were severely adherent to surrounding cerebellar gliotic tissue and transparent cysts contained white, milky fluid (Fig. 3). Pathological findings were consistent with cysticercus (Fig. 4). Serum and CSF enzyme-linked immunosorbent assay (ELISA) for cysticercosis were positive. He received antiparasitic treatment with praziquantel after surgery as an add-on therapy. One month after operation, his symptoms improved and he returned to his work. Brain MRI showed no residual mass and surgical defect in left cerebellar hemisphere (Fig. 5). In addition, serum and CSF ELISA for cysticercosis were converted to be negative.

DISCUSSION

Extraneural cysticercosis causes no major symptoms but neurocysticercosis and ophthalmic cysticercosis are associated substantial morbidity. Neurocysticercosis causes symptoms in most patients years after CNS infection by the parasite. Clinical manifestations result from inflammatory response to cyst degeneration, mass effect, obstruction of CSF pathway or residual scarring but are related to the numbers, size and location of lesions and the severity of host’s immune response. Cerebral hemispheres are the most commonly affected, mainly at the gray-white matter junction and cysts can also be found in the ventricles, basal cisterns and subarachnoid spaces. In the posterior fossa, neurocysticercosis usually involve the fourth ventricle, cerebellopontine angle cistern, cisterna magna and rarely, the cerebellum. Rarity of cysticercosis in the cerebellum may be the result of less abundant blood flow, compared to the cerebrum.

Imaging findings vary with development stage of neuro-

![Fig. 2](image.png). MRI showing an ill-defined, multicystic cerebellar mass with hyposignal on T1-weighted images (A), hypersignal on T2-weighted images (B), and rim enhancement after gadolinium injection (C).

![Fig. 3](image.png). Intraoperative views showing a cystic mass in the cerebellar hemisphere (A) and a white cystic content, grasped with forceps (B).

![Fig. 4](image.png). Photograph of the specimen showing dense collagen capsules with multiple cystic cavities and yellow cysts (A) and photomicrograph of cyst wall of cysticercus cellulosae showing a wavy, eosinophilic lamina with hair-like protrusions (hematoxylin and eosin, original magnification, x40) (B).

![Fig. 5](image.png). Postoperative-8-month T1-weighted axial postcontrast MRI scan showing no residual cyst with cerebellar surgical defect.
cysticercosis or host response and lesions may be at different stages in same patient. On imaging studies, viable cysts appear isodense with the cerebrospinal fluid and there are no contrast enhancement with little or no evidence of perilesional inflammation (vesicular stage). As the parasites degenerate, the cysts show pericystic contrast enhancement with perilesional edema (colloid stage). In later stage, they have nodular or ring enhancement after the administration of contrast, like in our case (granular-nodular stage). Finally, the cysts are not detectable or become calcified lesions (calcific stage). The differential diagnoses of neurocysticercosis on neuroimaging include abscess, tuberculosis, metastasis and other parasitic diseases. However, differential diagnosis between cysticercosis and other diseases may be difficult because clinical manifestations are not specific and some neuroimaging findings are not pathognomonic. Therefore, immunological tests and epidermological data as well as neuroimaging studies are helpful to diagnose neurocysticercosis.

Epileptic seizures are the most frequent presentation of parenchymal neurocysticercosis but large cyst may produce mass effect. Extraparenchymal neurocysticercosis in the ventricles and basal cisterns can be present with hydrocephalus or intracranial hypertension by mechanical obstruction of CSF pathway, either by the cysts or by an inflammation. In our case, a large multicystic cerebellar mass caused hydrocephalus and intracranial hypertension by mechanical obstruction of fourth ventricle.

Racemose neurocysticercosis is characterized by abnormal, large growth of cystic membrane without scolex. Racemose form usually occurs in the ventricles or basal cisterns and is rare in the brain parenchyma because there is not enough room in the brain parenchyma for growing of large cysts. However, our case was cerebellar racemose variety with large, multilobulated grapes-like cysts without scolex.

**CONCLUSION**

Even though cerebellar involvement of cysticercosis is very rare, neurocysticercosis should be taken into consideration as a differential diagnosis of multiple cystic mass lesions in the cerebellum, especially in the endemic area.

**References**