Cerebellar cysticercosis

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Cerebellar cysticercosis has been rarely reported [1) 2) 3) 4) 5) 6) 7)].

Rarity of cysticercosis in the cerebellum may be the result of less abundant blood flow, compared to the cerebrum.

Differential diagnoses

May simulate an astrocytoma, epidermoid, cystic schwannoma, cystic meningioma or arachnoid cyst. Thus, it may lead to misdiagnosis. Therefore, Zhu et al. suggest that the form of this disease should be considered when one attempts to characterize cerebellar neoplasms as detected by brain scan, especially in those geographical areas with high prevalence of neurocysticercosis [8)].

Treatment

Albendazole is useful in the treatment of cerebellar cysticerosis, even though the disease can be approached with surgery [9)].

Catapano et al. treated a case of left cerebellar cystic mass with anti-cysticercus chemotherapy of high dosages of dexamethasone (40 mg/d) and praziquantel (1200 mg every eight hours orally). The patient showed minimal improvement for three days and ended with a posterior fossa craniotomy, in which the left cerebellar lesion was removed to deviate herniation.

In the case of Zhu et al., the patient was treated with albendazole at the dosage of 150 mg/kg for three courses, and he experienced minimal drug-induced side effects during the therapy. The patient was free from neurological symptoms and signs. A series of cranial MRI also showed that the cystic lesion decreased dramatically. Of note, calcification of the cyst has not yet occurred during the follow-up examinations [10)].

Cerebellar cysticercosis caused by larval Taenia crassiceps

Ntoukas et al.published a cerebellar cysticercosis caused by larval Taenia crassiceps tapeworm in immunocompetent woman in Germany [11)].

At the time of admission, the patient showed cerebellar ataxia but no further neurologic deficits. She did not have fever or other symptoms. She had no known chronic preconditions or recent hospital stays and had never taken immunosuppressant drugs. She had no family history of neurologic symptoms or malignant diseases. Cranial computed tomography was performed and demonstrated a tumorous lesion (≈30 × 30 mm) in the right cerebellar hemisphere compressing the fourth ventricle. Magnetic resonance imaging revealed a multicystic mass with little perifocal edema.

The patient's leukocyte count was elevated (27.4 × 109 cells), and a differential count indicated 84% neutrophils, 8% lymphocytes, and 4% eosinophils. Aspartate aminotransferase (129 U/L), alanine aminotransferase (335 U/L), and gamma glutamyl transferase (196 U/L) levels were elevated, and total plasma protein concentration was slightly lowered (4.7 g/dL). Kidney function test results, C-reactive protein levels, and gamma globulin levels were within normal values.

Craniotomy revealed subdural and intracerebellar jelly-like tumorous tissue. The tumor, which consisted of multiple spherical masses with diameters of 2–4 mm, was resected. No infiltration of meningeal structures or the skull was evident.

Because an intracranial parasitosis or tumor was suspected, serum, tissue, and fluid from the cystic lesion were examined. Gross and histologic aspects of the excised tissue revealed typical structures for cestode larvae.

Serum and tissue samples were sent to a reference laboratory for further examination. Serologic test results for echinococcosis, which used crude and recombinant antigen ELISAs, and indirect hemagglutination test results were negative.

Commercial Western blots for cysticercosis and echinococcosis (LDBIO Diagnostics, Lyon, France) showed weak atypical bands of ≈47 kDa and 30 kDa, respectively.

For the tissue samples, cestode-specific PCRs selective for the parasite's mitochondrial 12S rRNA gene and mitochondrial cytochrome c oxidase subunit I gene were positive. After sequencing and conducting a BLAST search (www.ncbi.nlm.nih.gov/blast/Blast.cgi) of the 380-bp and 450-bp amplicons, we found that the sequences showed 99% and 100% homology with the T. crassiceps tapeworm, respectively.

A crude T. crassiceps ELISA similar to an in-house Echinococcus multilocularis assay was set up by using laboratory-kept T. crassiceps tapeworm larvae from another human patient. Serum samples from 10 healthy blood donors served as negative controls, and a standardized threshold index of 1.0 was calculated. Because no serum from patients with proven T. crassiceps tapeworm infections was available to use as a positive control, we used serum from patients with histologically confirmed cystic echinococcosis (5 patients), alveolar echinococcosis (7 patients), and peripheral cysticercosis (2 patients). All serum samples were positive, showing indices of 1.2–9.1, 1.4–6.6, and 2.2–3.3, respectively. The patient's serum, however, had an index below the threshold (0.76). When 5-µm cryosections from T. crassiceps tapeworm larvae were used for immunofluorescence tests, the patient's serum exhibited a weak tegumental signal.

After surgery, the patient was given praziquantel (600 mg twice daily) and albendazole (400 mg twice daily) for 3 months. The postoperative course was uneventful, the patient recovered rapidly, and there were no clinical or radiographic signs of recurrence after a follow-up period of 18 months. Extended imaging investigations showed no further sites of infection.

When the patient was asked about potential risk factors, she indicated that she had been living with her dog near a forest in a local rural area for many years. Consumption of wild berries or mushrooms possibly contaminated by fox feces could not be excluded. The dog, which had not regularly undergone deworming, had access to the garden and the nearby forest.

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