Neurocysticercosis: A Rare Cause of Occipital Lobe Epilepsy

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Thirteen-year-old boy admitted to our clinic because of intractable occipital lobe epilepsy and underwent surgical resection of epileptogenic focus at the left occipital lobe. Histopathological examinations revealed a parasitic infestation, neurocysticercosis. After surgery the patient was seizure-free at short-term follow-up and referred to the outpatient clinic of Infectious Diseases for further medical treatment of taenia solium. A case of intractable occipital lobe epilepsy due to cysticercosus was discussed with possible pitfalls in diagnosis.

Key words: Epilepsy, neurocysticercosis, occipital lobe

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Occipital lobe epilepsy per se is a rare entity, and modern imaging techniques are revealing more discrete lesions responsible for the epilepsy. Neurocysticercosis, caused by the larval form of taenia solium, is a parasitic infection of the central nervous system, and one of the most common causes of symptomatic epilepsy in underdeveloped and developing countries. However; scarce data exist in the current literature with respect to cysticercosis as an etiological factor for occipital lobe epilepsy.

The larval form of taenia solium enters into the body by ingestion of the eggs of the parasite in the focally contaminated food or water. The larvae can penetrate the intestinal mucosa and enter the circulatory system, by which they reach to and localize within the skeletal muscles, eyes, or brain as cystic form (1). Although the first mode of treatment in neurocysticercosis is anthelmintic therapy with preventive therapy for epileptic seizures and perifocal edema, surgery should also be considered in selected patients who have a discrete lesion and intractable epilepsy.

CASE REPORT

A 13-year-old boy was hospitalized due to the intractable occipital lobe epilepsy, which has been treated for 1 year with several antiepileptic drugs (AEDs). The medical history revealed that...
the AED regimen with different combinations could not prevent seizures and the frequency of seizures increased with time. In every episode, he always had an aura that was defined by the patient as seeing snowy points. The aura followed by having a blush of colors and limitation of visual field. His seizures became generalized once, and contractions especially on the right side of the body and loss of consciousness occurred.

Neurological examinations including formal ophthalmologic tests revealed nothing abnormal. He had no particular medical history and family history of seizure was negative.

The patient was extensively evaluated depending on epilepsy protocol including scalp electroencephalography (EEG) and imaging studies. Scalp EEG findings showing interictal and ictal spikes and waves from the left occipital lobe, were consistent with the semiology. Computed tomography (CT) revealed a small calcified nodule surrounded by contrast-enhanced minimal hypodensity on the left occipital lobe (Figure 1). The lesion was found to be hypointense and hyperintense on T1- and T2-weighted magnetic resonans imaging (MRI), respectively (Figure 2a and b). On FLAIR images, perilesional edema was well appreciated (Figure 2c). After gadolinium administration, rim enhancing lesion with peripheral edema was observed (Figure 2d-f). Preliminary diagnosis was thought to be a glial/neuroglial tumor or a chronic abcess.

He underwent surgery, in which the lesion was resected through an occipital craniotomy incision. Postoperative period was uneventful and there was no neurologic deficit. The patient has been seizure-free in the short-term follow-up (2 months after surgery) and is still on AED.

Histopathological examinations revealed encysted scolex of a tapeworm. A granulomatous reaction against it with growing abscess, chronic inflamatory infiltration, and reactive gliofibrosis were noted (Figure 3). The final diagnosis was neurocysticercosis caused by taenia solium and the patient was transferred to the department of Infectious Diseasses for further antihelminthic treatment.
DISCUSSION

Cystocercosis is an infestation of body tissues by taenia solium, the larval form of the pig tapeworm. The larvae enter into the body by the ingestion of taenia solium eggs in fecally contaminated food or water. After ingestion, the eggs hatch in the duodenum and release larvae that can penetrate intestinal mucosa and enter the circulatory system, by which they localize as cysts within the skeletal muscles, eyes, or brain (1). When a larva is detected in the nervous system, this clinical manifestation is named as neurocysticercosis, which is the most common helminthic infection of the central nervous system (2).

In the brain, immature cysts appear within one to four weeks after ingestion of eggs (stage I) and become mature in two months (stage II). There is no or minimal surrounding inflammation at this stage and the host is mostly asymptomatic. Stage III is characterized by the degeneration of the cysts (granuloma). Cysts don’t prevent the development of host’s immune response at this stage, so that dense inflammation, which leads to clinical signs and symptoms, comes out. At stage IV, the cyst calcifies or resolves without scarring and the inflammation disappears. Epileptic seizures can be present at any stage. However, seizures are manifested mainly in stage III, as the death of the cysticercus releases larval antigens that stimulate an inflammatory host response (3). Generally in stages I and II seizures are not induced (2).

Considering our case, one may found that the patient has been in stage III, which generally causes seizures, and at the time of the diagnosis, the cysticercosis was in stage IV, which includes calcification that was observed on imaging studies.

Histopathological examination of the current case revealed that the cyst had a thin translucent wall filled with clear fluid and a four sucker scolectes with a double row of birefringent hooklets arising from the parenchymatous area. The diagnosis of taenia solium as a cause of occipital lobe epilepsy was surprising to us since it is very rare to see a patient who has been suffering from occipital lobe epilepsy caused by a discrete lesion like cysticercosis.

Our case has led us to be more careful about such lesions and helminthic infections, which should be considered in the differential diagnosis that may include glioneuronal tumors such as ganglioglioma or dysembryoplastic neuroepithelial tumors, and small abscess due to tuberculosis, which is relatively common in Turkey since there is no specific MRI appearance that could be suggestive of neurocysticercosis. We have to underline that if we were able to make the correct diagnosis depending on the preoperative images, we would not take the patient to the operating room given that the primary mode of the treatment is medical rather than surgery (4).

CONCLUSION

Neurocysticercosis is an uncommon cause of epilepsy, particularly the occipital lobe epilepsy, even in developing countries and should be considered in differential diagnosis due to that correct diagnosis may prevent unnecessary surgery, since antihelminthic drug therapy is the first treatment of choice.

REFERENCES