Childhood Seizure-A Case of Neurocysticercosis Involving Left Parietal Lobe

Çocukluk Epilepsisi-Sol Pariyetal Lobu Tutan Bir Sistiserkozis Olgusu

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ÖZET

On iki yaşında erkek çocuk üç kez jeneralize olan parsiyel nöbetler nedeniyle getirildi. Tetkiklerden sonra sol pariyetal lobda nörosistiserkozis olarak tanı kondu. Albendazol ve fenitoin ile tedavi verildi ve sekiz yıl izlendi. Bilgisayarlı tomografisinde düzelme gözlendi ve nöbeti olmadı.

Anahtar Kelimeler: Nöbetler, nörosistiserkozis, fenitoin.

ABSTRACT

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A 12-year-old boy presented with three episodes of partial seizures with secondary generalization. On investigation, he was diagnosed as a case of neurocysticercosis of the left parietal lobe. He was treated with albendazole and phenytoin and was followed for the next eight years. Computerized tomography scan showed signs of resolution with no further seizure activity.

Key Words: Seizures, neurocysticercosis, phenytoin.

INTRODUCTION

Human cysticercosis, an infection by the larval stage (cysticercus) of the pork tapeworm *Taenia solium*, is the most common parasitic central nervous infection worldwide. Though it affects adults commonly, pediatric infection is also increasingly being recognized in the developing world. The computerized tomography (CT) scan shows vesicular cyst (viable cyst with no host immune reaction), colloidal cyst (dead or dying cyst), granuloma, or calcification.

CASE

A 12-year-old boy presented with partial seizure with secondary generalization. The convulsion started in the left lower limb and spread to the whole body. He remained unconscious for 8-10 minutes. There was no history suggestive of limb paralysis, fever, vomiting, headache, or blurring of vision. There was no family history of convulsion. His systemic examination was normal, and there was no focal neurologic deficit. The convulsion was immediately controlled with intravenous diazepam. His report showed total white blood count (WBC) count of 8.4 x 10^9 /L with neutrophil 63%, lymphocyte 28%, eosinophil count 5%, basophil 2%, and monocyte 2%. His Mantoux test was low-positive, but three morning sputum samples for acid-fast bacilli were negative. Chest X-ray [posteroanterior (PA)] view and serum IgG and IgM for tuberculosis were normal. Interictal EEG showed cerebral dysrhythmia with focus in the left parieto-temporal region. Cerebrospinal fluid (CSF) study was not done. His CT brain scan with contrast study revealed a solitary ring-enhancing lesion of approximately 1 cm diameter in the left parietal lobe area with significant perilesional edema and no midline shift (Figure 1). His serum IgG for cysticercus (ELISA) was high.



Figure 1. Brain CT with contrast: A left parietal lobe single ringenhancing lesion with significant perilesional edema.

He was diagnosed as neurocysticercosis of the left parietal lobe using the criteria of Del Brutto et al. (1) (Table 1).

Diagnosis is confirmed by either one absolute criterion or a combination of two major criteria, one minor criterion, and one epidemiologic criterion. We confirmed the diagnosis with major criteria 2a & 2c (evidenced by resolution of the lesion by calcification), minor criteria 3a & 3b (focal seizure with secondary generalization), and epidemiologic criterion 4a (as India is an endemic area).

He was discharged with albendazole 15 mg/kg/day (twice daily divided doses) for 28 days and oral prednisolone tablet 1 mg/kg/day for 30 days (started 2 days before the initiation of albendazole therapy and continued until the albendazole was used) (2-5). Phenytoin 100 mg thrice daily was also added (4,6). A repeat brain CT 30 days later showed reduction of the perilesional edema, but ring size was almost similar. Albendazole was stopped after 28 days and oral prednisolone was stopped 30 days after the introduction of the therapy with a tapering dose. Oral phenytoin was continued at the same dose. Unfortunately, he experienced two further episodes of simple partial seizure without secondary generalization or loss of consciousness in the next six months after the introduction of the phenytoin therapy. The rest of the clinical, neurological (higher functions, motor, sensory and cerebellar systems), cardiovascular, respiratory, and gastrointestinal examinations were normal during the followup. Almost 12 months after the initial seizure, a repeat brain CT showed signs of mild resolution of the lesion and the surrounding edema, but an interictal EEG showed a similar focal pattern (left parieto-temporal) of cerebral dysrhythmia. Phenytoin was continued according to the previous dose. Four years after the initial seizure, a repeat brain CT with contrast showed the disappearance of the lesion with a calcified spot in the left frontoparietal region and absence of edema (Figure 2). No seizure attack had been noted since the last two episodes of simple partial seizure without generalization observed within the first half year of the initiation of the phenytoin therapy (300 mg daily). His phenytoin dose was gradually tapered to 100 mg 2 tablets per day and is continuing thusly, with no new seizure attack.

DISCUSSION

In patients from the endemic areas (our hospital is in eastern India) like Southeast Asia, and Latin and Central America, a solitary ring-enhancing lesion presenting with seizure, normal physical examination, no evidence of any systemic disease, and the constellation of thin-walled rounded CT lesion of 0.5 to 20 mm diameter, with no midline shift in the CT scan of the brain, is almost always caused by neurocysticercosis (7). The closest differential diagnosis in endemic areas is tuberculoma. Evidences sup-



Figure 2. Brain CT with contrast: A left frontoparietal area calcification with no perilesional edema following albendazole therapy.

porting tuberculoma include a CT ring size of more 2 cm, associated midline shift, irregular and often thick-walled outline, absence of scolex, and lack of early spontaneous disappearance (8). On clinical grounds, neurocysticercosis can be confused with encephalitis, stroke or meningitis, etc. On imaging studies, the solitary ring-enhancing lesion of cysticercus can be difficult to distinguish from tuberculosis, mycosis, metastasis, early glioma, toxoplasmosis, abscess, histoplasmosis, and arteriovenous malformations (4,9).

As the host immune response caused gradual death of the cyst, a marked inflammatory response or pericyst edema occurred, producing a nodular enhancement with the contrast media. Convulsion occurred for the first time due to the parenchymal irritation. Subsequent episodes post-albendazole therapy occurred because of parenchymal irritation or inflammation following the death of the cyst due to albendazole therapy or gliosis-associated endstage calcified lesion (3). Oral steroid as prednisolone (1 mg/kg/day) was started 2 days prior to the albendazole therapy to reduce the perilesional edema and the inflammatory reactions post-albendazole therapy, as this inflammatory response could have precipitated further seizure attacks (4). The parietal lobe is the commonest area to be affected in the pediatric age group, but the cause is not clear (10). In this case of neurocysticercosis, CSF study was not done deliberately, as eosinophilia in the CSF is not a reliable finding, and if absent, does not preclude the diagnosis.

Serum enzyme-linked immunoelectrotransfer blot (EITB) has 100% specificity and 98% sensitivity in the diagnosis, while CSF EITB has lower sensitivity and specifi-

Table 1. Diagnostic criteria for human cysticercosis (1).

1. Absolute criteria

- a. Demonstration of cysticerci by histologic or microscopic examination of biopsy material
- b. Visualization of the parasite in the eye by funduscopy
- c. Neuroradiologic demonstration of cystic lesions containing a characteristic scolex
- 2. Major criteria
- a. Neuroradiologic lesions suggestive of neurocysticercosis
- b. Demonstration of antibodies to cysticerci in serum by enzyme-linked immunoelectrotransfer blot (EITB)
- c. Resolution of intracranial cystic lesions spontaneously or after therapy with albendazole or praziquantel alone
- 3. Minor criteria
- a. Lesions compatible with neurocysticercosis detected by neuroimaging studies
- b. Clinical manifestations suggestive of neurocysticercosis
- c. Demonstration of antibodies to cysticerci or cysticercal antigen in cerebrospinal fluid by ELISA
- d. Evidence of cysticercosis outside the central nervous system (e.g., cigar-shaped soft tissue calcifications)
- 4. Epidemiologic criteria
- a. Residence in a cysticercosis-endemic area
- b. Frequent travel to a cysticercosis-endemic area
- c. Household contact with an individual infected with Taenia solium

city. If EITB is not available, older serum ELISA is 63% specific and 65% sensitive, whereas CSF ELISA is 95% specific and 87% sensitive for cysticercus IgM and IgG (3). ELI-SA for neurocysticercosis is positive in almost 50% cases of single CT ring lesion (5).

Though albendazole is the widely recommended therapy, it did not provide a satisfactory result in the shortterm in this case in terms of radiological improvement (3-5,11). In India, spontaneous resolution was observed in one-third of cases of solitary cysticercus brain lesion (8). The role of albendazole is doubted since the natural history of neurocysticercosis in adults is to disappear spontaneously or to calcify. The natural history of neurocysticercosis in the pediatric age group appears in no way different from that in adults.

REFERENCES

- Del Brutto OH, Rajshekhar V, White AC Jr, Tsang VC, Nash TE, Takayanagui OM, et al. Proposed diagnostic criteria for neurocysticercosis. Neurology 2001;57:177-83.
- Garcia HH, Evans CA , Nash TE Takayanagui OM, White AC Jr, Botero D, et al. Current consensus guidelines for treatment of neurocysticercosis. Clin Microbiol Rev 2002;15:747-56.
- Goldsmith RS. Infectious diseases-protozoal and helminthic infections. In: McPhee SJ, Papadakis MA, Turney LM Jr. Current Medical Diagnosis Treatment. 46th ed. (International Edition). New York: McGraw Hill (Medical), 2007:1541-3.
- Blanton R. Cysticercosis. In: Kliegman RM, Jenson HB, Behrman RE, Stanton BF (eds). Nelson Textbook of Pediatrics. Vol 1. 18th ed (South & South East Asia). Philadelphia: Saunders Elsevier, 2007:1514-6.
- Kalra V. Central Nervous System. Ghai Essential Pediatrics. 7th ed. New Delhi: CBS Publishers and Distributers Pvt Ltd, 2009:546-7.

- Roos KL, Tyler KL. Meningitis, encephalitis, brain abscess and empyema. In: Kasper DL, Fauci AS, Longo DL, Braunwald E, Hauser SL, Jameson JL, Loscalzo J, et al. (eds). Harrison's Principles of Internal Medicine. Vol 2. 17th ed. New York: McGraw Hill (Medical Publishing Division), 2008:2638.
- White AC Jr, Weller PF. Cestodes. In: Kasper DL, Fauci AS, Longo DL, Braunwald E, Hauser SL, Jameson JL, Loscalzo J, et al. (eds). Harrison's Principles of Internal Medicine. Vol 1. 17th ed. New York: McGraw Hill (Medical Publishing Division), 2008:1337-8.
- Kalra V. Neurocysticercosis. In: Parthasarathy A, Menon PSN, et al. (eds). IAP Textbook of Pediatrics. Vol 1. 4th ed. New Delhi: Jaypee Brothers Medical Publishers (P) Ltd, 2009:469-70.
- Garcia HH, Gonzalez AE, Evans CA, Gilman RH; Cysticercosis Working Group in Peru. Taenia solium cysticercosis. Lancet 2003;361:547-56.
- Basu S, Ramchandran U, Thapliyal A. Clinical profile and outcome of pediatric neuro-cysticercosis: a study from Western Nepal. J Pediatr Neurol 2007;5:45-52.
- 11. Singhi P, Ray M, Singhi S, et al. Clinical spectrum of 500 children with neurocysticercosis and response to albendazole therapy. J Child Neurol 2000;15:207-13.

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