J Korean Med Sci 1998; 13: 445-8
ISSN 1011-8934

Temporal Lobe Epilepsy Caused by Intrahippocampal Calcified Cysticercus
: A Case Report

The major differential diagnoses of epilepsy associated with small solitary lesion are tuberculous or cysticercus granuloma which are enhanced in CT and/or MRI study. We report of a 47-year-old man with intractable temporal lobe epilepsy as the presenting feature of a solitary intrahippocampal calcified mass without enhancement, which turned out to be a *Taenia solium* cysticercus. There was no apparent evidence of systemic cysticercosis. Imaging studies revealed a small solitary intrahippocampal calcification without perilesional enhancement, and atrophy of the hippocampal head portion. Cysticercosis should be considered as an etiology in the differential diagnosis in lesional medial temporal lobe epilepsy even without perilesional enhancement.

Key Words : *Temporal lobe; Epilepsy; Hippocampus; Taenia solium; Cysticercosis*

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Received : December 19, 1997
Accepted : March 22, 1998

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*This work was in part supported by a clinical research grant from Seoul National University Hospital.

INTRODUCTION

Tuberculous or cysticercus granuloma are the major differential diagnoses in epilepsy associated with a small solitary enhancing lesion in computerized tomography (CT) and/or magnetic resonance imaging (MRI) (1-5). Cerebral cysticercosis is one of the most common causes of epileptic disorders in several parts of Asia and Latin America (4, 6). Here we report a case of a 47-year- old man with intractable temporal lobe epilepsy as the presenting feature of a solitary intrahippocampal calcified cysticercus without enhancement.

CASE REPORT

This 47-year-old right-handed man presented with a 20-year history of intractable temporal lobe epilepsy, which did not respond to major antiepileptic drugs. He usually felt epigastric rising sense as an aura, then stared blankly, and did oroalimentary automatism. Postictal confusion persisted for several hours. Frequency ranged from several times a month to several times a day. He denied a history of febrile convulsion.

Physical examination

Detailed physical examination failed to reveal any neurological deficit. Subcutaneous nodules were not noted. Serum and cerebrospinal fluid enzyme-linked immunoassay for antibody for cysticercosis had been done 3 times over 10 years, all of which were negative. Though he complained of memory disturbance, minimental test revealed full mentality. A cranial non-enhanced CT scan showed a calcification, 6 to 8 mm in diameter, in the region of the left medial temporal lobe (Fig. 1). MRI (1.0 T) revealed a focal low signal intensity lesion in the left medial temporal region, which did not enhance with gadolinium, and minimal atrophy of the left hippocampus. The interictal electroencephalography (EEG) and prolonged video-EEG monitoring were compatible with the left temporal lobe epilepsy. Interictal positron emission tomography scan with 18-fluorodeoxyglucose revealed hypometabolism in the region of the left temporal lobe. On neurocognitive test, there was a relative weakness in storing verbal and visual associative characteristics and retrieving over brief periods of time. However, there was no significant difference between verbal and visual memory. Wada test showed a left dominance for language and

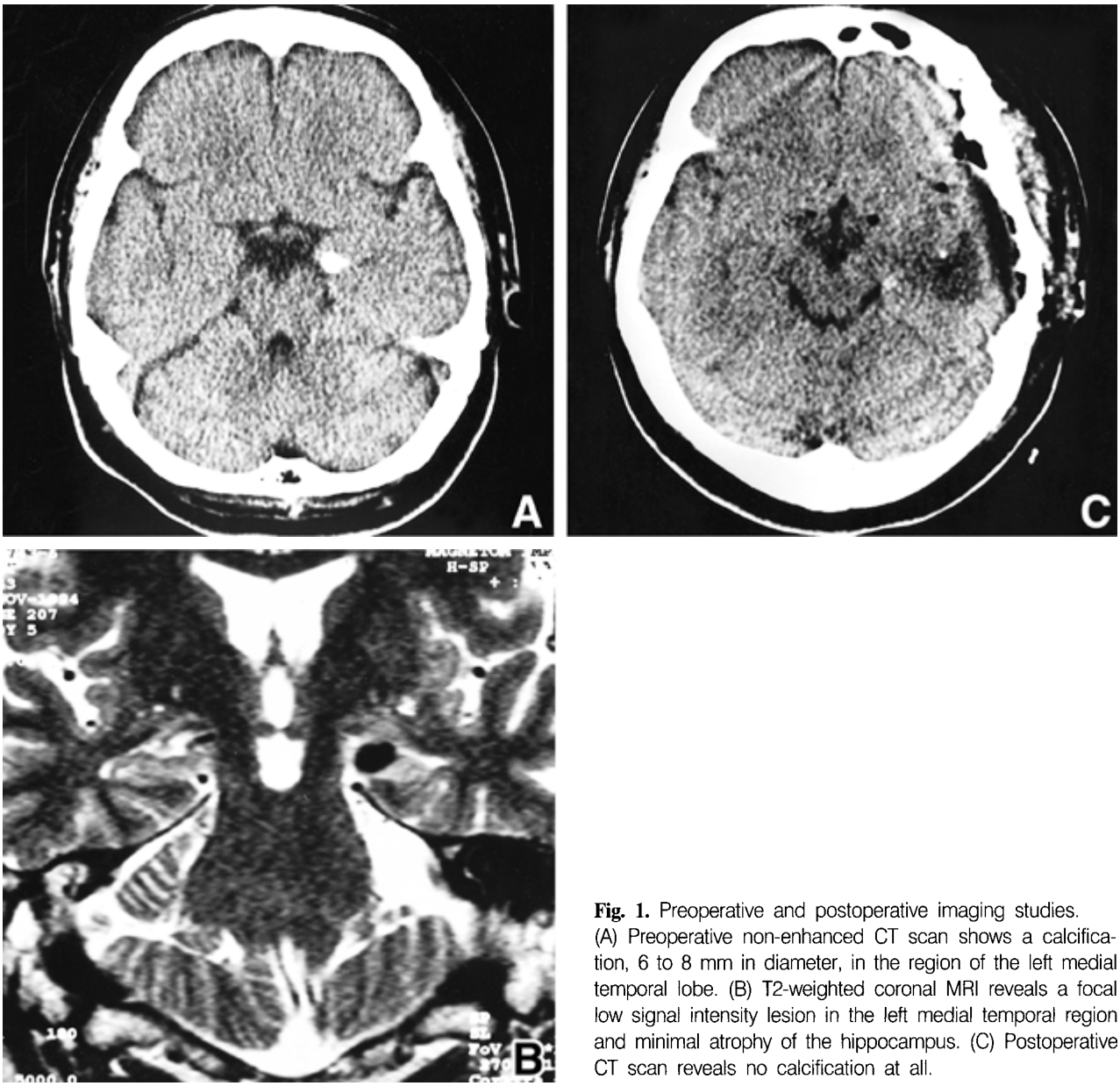

Fig. 1. Preoperative and postoperative imaging studies. (A) Preoperative non-enhanced CT scan shows a calcification, 6 to 8 mm in diameter, in the region of the left medial temporal lobe. (B) T2-weighted coronal MRI reveals a focal low signal intensity lesion in the left medial temporal region and minimal atrophy of the hippocampus. (C) Postoperative CT scan reveals no calcification at all.

bilateral memory function.

Operation

The patient underwent standard left temporal lobectomy. Briefly, the lateral neocortex was removed 4.5 cm from the temporal tip. The amygdala and hippocamus were removed. On the superomedial edge of the hippocampus, a calcified mass was noted, and was resected en toto.

Postoperative course

He experienced several episodes of generalized tonic clonic seizures on the first postoperative day. Thereafter he has been seizure-free for two years. Mild dysphasia was noted postoperatively, but disappeared in 7 days. He complained of further impairment of memory, which did not hinder normal daily activity. A CT scan taken on the second postoperative day revealed no calcification at all.

Pathology

Histologic examination revealed neuronal loss and gliosis in the fascia dentata (Fig. 2). Abundant corpora amylacea was noted in the hippocampus. The calcified mass found superomedial to the hippocampus was measured

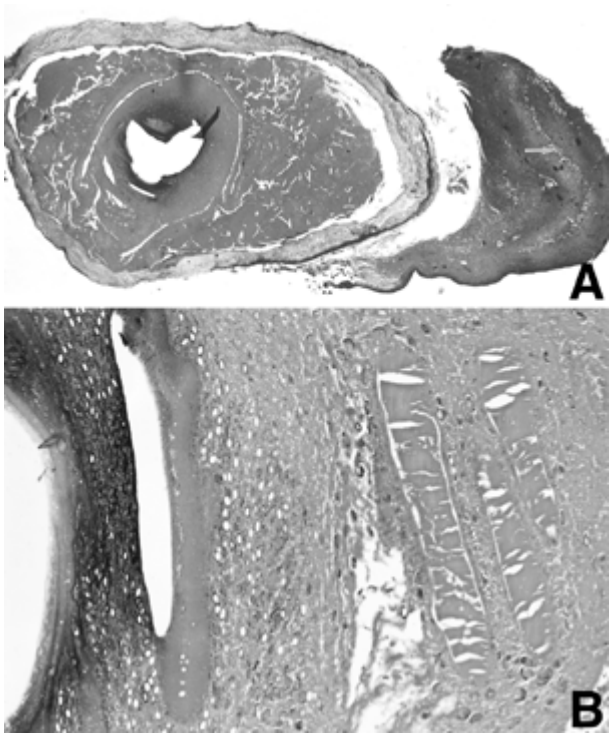

Fig. 2. Pathological examination. H & E. (A) Histologic examination reveals cystic lesion containing a degenerated cysticercus worm embedded in hippocampus showing fascia dentata (arrow) with neuronal loss and gliosis (×20). (B) The scolex portion of the worm is shown, with corrugated appearance and numerous calcospherules (×250).

at 1.5×1 cm grossly. Microscopically, degenerated cysticercus and scolex surrounded by hippocampal tissue were observed.

DISCUSSION

The major differential diagnoses of single intrahippocampal calcified mass without enhancement and associated with hippocampal atrophy could be calcified glioma or late-stage neurocysticercosis. Temporal lobe epilepsy due to calcified glioma of amygdalo-hippocampal region has been described (7). In this case, CT and MRI showed dense calcification without perilesional enhancement (7). However, any patients with late-onset epilepsy living in an endemic area of taeniasis/cysticercosis, should be considered as having neurocysticercosis, regardless of the seizure type and the EEG findings (8).

Taeniasis and cysticercosis caused by *Taenia solium*, the pork tapeworm, is widely prevalent in human and swine hosts in many developing countries of Latin America, Africa, and Asia (9). Human taeniasis is acquired by ingesting larvae (cysticerci) in raw or inadequately cooked meat of the natural intermedite host, the pig. Humans and pigs acquire cysticercosis by ingesting the eggs of the cestode passed in the feces of a human tapeworm carrier. Infection in pigs is facilitated by their coprophagic habits. Humans are exposed to eggs by autoinfection, by direct contact with another tapeworm carrier, or indirectly by ingestion of food or water contaminated with human feces (10).

Neurocysticercosis was diagnosed in from 5.1% to 50 % late-onset epilepsy in developing countries (6, 11). In 750 normal persons who had a routine physical examination, the antibody positive rate was 2.1% in Korea (12). This means that physicians treating late-onset epilepsy shoud include cysticercosis to their differential diagnosis in Korea.

For a non-invasive serological test for cysticercosis, enzyme-linked immunosorbent assay has been used due to its high diagnostic accuracy and simplicity (1, 13). However, anti-cysticercus antibody is negative when only few worms were infected, or when the worms were completely calcified (14, 15). Partial or generalized epilepsy is still caused and continued by the calcified worm(s) (12).

In one outcome study for patients with epilepsy due to neurocysticercosis, there were fewer seizures after medical treatment than after surgical extirpation of the cystic lesion, perhaps because surgical excision carries its own risk of tissue damage (16).

Our case was in the calcific stage, which meant a dead parasite, and had no evidence of systemic cysticercosis preoperatively. The patient's epilepsy was intractable in spite of several antiepileptic drugs. In this case, surgery should be performed not only for diagnostic purposes to rule out tumorous conditions, but also for control of intractable temporal lobe epilepsy.

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