Neurocysticercosis, a disease caused by a tapeworm larva in the central nervous system, is the leading cause of acquired epilepsy in undeveloped regions of the world. In Taiwan, after improvements in sanitation, tapeworm infection became very rare except in mountain areas inhabited by aborigines. However, in recent years, immigration from other Asian countries has increased rapidly, and parasite infection of the central nervous system may again become an important cause of adult-onset epilepsy in clinical practice. Here, we describe a 27-year-old female Thai immigrant who presented with adult-onset epilepsy partialis continua of the right upper extremity. Electroencephalography showed epileptiform discharge in the left central region, while brain magnetic resonance imaging showed a small enhanced lesion in the left premotor cortex. She underwent operation and pathology of the mass revealed a degenerated cysticercus. In this article, we provide detailed neuroimaging findings, pathologic report, and literature review of this parasitic infection of the central nervous system. This case calls for physicians to be aware of cysticercosis as an etiology of adult-onset epilepsy in immigrants from endemic countries.

Key Words: epilepsy partialis continua, epilepsy, neurocysticercosis, Taenia solium, tapeworm
the infection in the central nervous system are reviewed. A detailed differential diagnosis of the neuroimaging findings in this case is described.

**Case Report**

A 27-year-old Thai woman attended our neurologic clinic with the chief complaint of intermittent twitching of the right elbow for 12 hours, which occurred about 20 days after a 1-minute episode of clonic movement in her right wrist, with clear consciousness. The second episode consisted of stereotyped, semirhythmic, clonic flexion movement of her right wrist that lasted for 1–2 minutes, which then involved her right elbow, and finally her right shoulder. This was followed by frequent clonic flexion movements of her right elbow, initially occurring every 10–20 minutes, then becoming more frequent and finally persistent. She could not control the movement. Of note, the clonic movement did not disappear during sleep. Prior to these episodes, she had no medical disease, headache, fever, head trauma, or previous seizure. Her personal history showed that she had immigrated to Taiwan with her family to work 4 years previously and returned to her homeland every year. Her last visit to Thailand was 1.5 years ago; while there, her drinking water was obtained from a well in a field.

On physical examination, she did not have fever, neck stiffness, or lymphadenopathy. The neurologic examination showed clear consciousness, mild weakness in the right upper limb graded 4 on the Medical Research Council grading system, and increased tendon reflex and hypesthesia in the right upper limb. Epilepsia partialis continua was diagnosed clinically, the lesion site probably being in the left motor cortex, near the central vertex area.

Awake electroencephalography (EEG) was performed. The background activity consisted of symmetrical alpha rhythm in the posterior head area, with an adequate alerting response to eye opening. There were frequent regional semirhythmic slow waves at 1–3 Hz, 25–50 μV in the left central region, with occasional lateralization or extension to the right side. There was an episode of clinical seizure characterized by twitching of the right upper limb, but the EEG pattern did not change during the attack (Figure 1A). In addition, hyperventilation induced an episode of generalized sharp waves with phase reversal at C3 (Figure 1B). These EEG findings suggested a focal lesion in the left central region.

Brain magnetic resonance imaging (MRI) showed a well-defined rim-enhanced nodule with a mural nodule in the left posterior frontal lobe, with marked perifocal edema (Figure 2). There was no signal change in the lesion on diffusion-weighted image or apparent diffusion coefficient. Tumor or granuloma was considered. Blood tests, including white blood cell count with a differential count (eosinophils, 4.2%), C-reactive protein,
tumor markers, and anti-HIV antibody titer, were all within normal limits.

Her seizure was completely controlled with valproic acid (1000 mg/day) on the second day of hospitalization. The lesion in the left premotor area was excised by craniotomy, and pathology showed a partially degenerated cysticercus (Figure 3). The stools were checked twice for parasite ova and were negative. She received praziquantel for 2 weeks after the operation. Intermittent paresthesia on the right side of the body was well controlled by increasing the dosage of valproic acid to 1500 mg/day.

**Discussion**

A 27-year-old Thai woman had a late-onset seizure with continuous clonic convulsion in her right elbow. EEG study showed an epileptogenic focus in the left central area, which was compatible with the clinical impression of epilepsia partialis continua. No epileptiform activity was recorded during attack; this discrepancy between motor convulsion and electrophysiologic findings may be due to multiple closely allied small groups of neurons firing asynchronously or the discharge focus being deeply seated in the folded cortex.8 MRI revealed a single enhanced mass with perifocal edema in the left premotor area. For a single brain lesion causing focal seizure, the differential diagnoses include tumor, granuloma, and pyogenic abscess;8 the characteristic brain MRI findings are summarized in the Table.9–13 Unexpectedly, the pathologic findings of the mass lesion showed a partially degenerated cysticercus (Figure 3). The cysticercus in this case was probably *T. solium*, because there is no report that *T. saginata* produces cysticercosis in the human brain.14
Human cysticercosis of *T. solium* is caused by accidental ingestion of its eggs. Its lifecycle is shown in Figure 4. The cysticercus (larva) hatches from eggs in the human stomach and enters various tissues through the blood stream. If it enters the central nervous system, it may lodge in the parenchyma, ventricle, or cistern, resulting in different clinical manifestations. In parenchymal neurocysticercosis, there are four histopathologic stages on neuroimaging, these being the viable stage, colloid stage, nodular-granular stage, and calcified stage. Staging depends on the viability of the cysticercus and the host inflammatory response. In the viable stage, the larva elicits little or no inflammatory response, so it is always asymptomatic. Computed tomography (CT) or MRI shows

**Figure 3.** (A) Pathologic findings of a partially degenerated cysticercus in the brain (hematoxylin & eosin, 10×). (B) The partially degenerated cysticercus was surrounded by inflammatory infiltrates of lymphocytes, plasma cells and numerous eosinophils (hematoxylin & eosin, 40×).

**Figure 4.** The lifecycle of *Taenia solium*.

**Table.** Magnetic resonance imaging (MRI) sequence findings in the differential diagnosis of a single enhanced lesion in the brain

<table>
<thead>
<tr>
<th>MRI sequence</th>
<th>Abscess</th>
<th>Necrotic tumor</th>
<th>CNS lymphoma</th>
<th>Tuberculoma</th>
<th>Cysticercosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>T1WI</td>
<td>Hypointense</td>
<td>Hypointense</td>
<td>Iso- or hypointense</td>
<td>Iso- or hyperintense</td>
<td>Iso- or hyperintense, hole-with-dot</td>
</tr>
<tr>
<td>T2WI</td>
<td>Hyperintense</td>
<td>Hyperintense</td>
<td>Iso- or hypointense</td>
<td>Iso- or hyperintense</td>
<td>Iso- or hyperintense, hole-with-dot</td>
</tr>
<tr>
<td>Ring enhancement on T1WI with contrast</td>
<td>(+)</td>
<td>(+) Thick &amp; irregular ring</td>
<td>(+) With little or no perifocal edema</td>
<td>(+)</td>
<td>(+)</td>
</tr>
<tr>
<td>DWI</td>
<td>Hyperintense</td>
<td>Hypointense</td>
<td>Hyperintense</td>
<td>Hyperintense</td>
<td>Isointense</td>
</tr>
<tr>
<td>ADC</td>
<td>Low value</td>
<td>High value</td>
<td>Low value</td>
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</table>

*CNS lymphoma in an immunocompromised patient. CNS = central nervous system; T1WI = T1-weighted image; T2WI = T2-weighted image; DWI = diffusion-weighted image; ADC = apparent diffusion coefficient.*
a vesicle containing a small eccentric nodule, which is the scolex (head). The “hole-with-dot” imaging is pathognomonic to cysticercosis. There is little or no perilesional edema or enhancement due to mechanisms for successful escape from the host’s immunologic system. After several years, the larva dies and an inflammatory response occurs. The fluid in the vesicle becomes opaque, so this stage is called the colloidal stage. On imaging, the wall of the cyst is thickened, with perifocal edema and contrast enhancement. Later, the cyst develops into a granuloma, which is the nodular-granular stage. On MRI, it is signal-devoid on T1- and T2-weighted images and surrounded by a hyperintense rim and perifocal edema. Finally, the granuloma becomes a calcified nodule. The calcified stage is better seen on CT; on MRI, the calcified nodule is not readily visible, but perifocal edema and contrast enhancement can be seen. In our patient, the neurocysticercosis was a parenchymal form and in the colloidal stage (Figure 3).

The symptoms of parenchymal neurocysticercosis are mainly due to the inflammatory response induced by the degenerated or calcified cyst. Seizure is the most common manifestation; others include encephalitis, meningitis, intracranial hypertension, or focal neurologic signs. If the cysts are in the ventricle or basal cistern, they may result in hydrocephalus by blocking the circulation of cerebrospinal fluid, in focal signs due to direct compression, or in stroke due to endarteritis. It was unclear how and where the patient ingested the eggs of *T. solium*. The lifespan of a cysticercus in the human brain is estimated to be several years, and they usually do not cause symptoms until they are dying. *Taenia* infection is rare in Taiwan but common in Thailand, with the prevalence ranging from 0% to 18.2% in a study assessed by worm purging. In rural areas in Thailand, open-air defecation occurs and contaminates the water supply, soil and vegetation. This patient did not eat uncooked vegetables, but her drinking water was from a well that might have been contaminated by *Taenia* eggs. It might be one of the possible sources of her parasite infection.

In summary, the patient was a female Thai immigrant with neurocysticercosis who presented with epilepsia partialis continua. This case report highlights the importance of *Taenia* infection as an etiology of late-onset focal seizure in immigrants from endemic countries.

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**References**