CASE REPORT

A Case of Neurocysticercosis in Immigrant Worker Presented with Seizures

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ABSTRACT

Neurocysticercosis is an infection of the central nervous system (CNS) by larval stage of *Taenia solium* (pork tapeworms) called cysticerci. Humans may acquire the infection through ingestion of *T. solium* embryonated eggs after consuming contaminated undercooked food or water. Being a multiracial country where pig farming is limited and consumption of raw or undercooked food is an uncommon practice, the prevalence of neurocysticercosis in Malaysia is presumably low. However, through immigration and international travel, cases of neurocysticercosis may go underreported. Due to unfamiliarity with the disease, cases of neurocysticercosis also may be missed in Malaysia. Therefore, thorough history taking and physical examinations with high index of suspicion are required by the clinicians to warrant proper investigations in order to meet the diagnosis of neurocysticercosis. Patients diagnosed with neurocysticercosis should be treated cautiously with anthelmintic, to avoid unwanted overwhelming immunological response that can lead to unfavourable outcomes. This is a case report of a foreign worker who presented with first onset of focal seizure associated with intermittent headache.

Keywords: *Taenia solium*, Neurocysticercosis, Pork Tapeworms

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INTRODUCTION

Taenia solium larvae (cysticerci) infection in organs or tissues is known as cysticercosis. The infection is a result of ingestion of *T. solium* embryonated eggs via contaminated food or water especially in endemic regions where humans live in close proximity with roaming pigs, in addition to strong association with consumption of undercooked pork. Many different sites of cysticercosis infections have been recognised, but neurocysticercosis or infection of the cysticerci in the central nervous system (CNS) is the most important as it can cause seizures, altered mental states, increased intracranial pressure and even death. In high prevalence areas such as in developing countries located in South America, Asia and Africa, any person with impaired CNS function should be investigated for the possibility of *T. solium* infection. Today, cases of human infestation by this parasite are becoming more frequent even in developed countries as a result of immigration and travel. In Asia, the overall prevalence of *T. solium* cysticercosis is around 3.9%, while the prevalence in endemic neighbouring countries such as Vietnam, Philippines and Papua, it can be as high as 7.2%, 24.6% and 45.8% respectively (1). Nevertheless, cysticercosis cases are still rarely reported in Malaysia, but presumably underreported especially among immigrants from endemic regions. At present, there is no published data regarding its prevalence in this country.

CASE REPORT

A 38-year-old Nepali man, who migrated to Malaysia less than two years ago to work as a security guard, presented with two episodes of seizures within 24 hours. The seizures involved twitching of the left eye and left side of the face that lasted for about three to five minutes each time with spontaneous abortion and associated with post ictal drowsiness and headache. No other signs and symptoms of meningitis such as fever, neck stiffness or photophobia was reported. Past information includes no history of trauma, recent travel, prior seizure episodes nor family history of epilepsy. Patient had no past medical history of hypertension, diabetes or tuberculosis, and denied any history of recreational drug usage or alcohol abuse. Originating from a Burmese Hindu tribe, the patient usually practiced a restricted diet. However, he did have some history of consuming pork whilst he was back in Nepal, but was unsure whether the pork was raw or properly cooked.

On examination, the patient was alert, orientated, afebrile and haemodynamically stable. There was no

palpable lymph nodes or superficial mass noted. All the systemic examinations including the eye and cranial nerves were unremarkable. Laboratory investigations for routine haematological, biochemical and viral screening tests revealed a normal parameter (except for fasting lipid profile) with normal renal and liver function tests, normal glucose levels and negative results for hepatitis B surface antigen, hepatitis C virus antibody, HIV antigen and antibody. The patient was initially diagnosed as having a cerebrovascular accident (CVA) based on a preliminary plain CT brain which showed left frontal hypodense lesions, in addition to high serum total cholesterol (6.02 mmol/L) and LDL (4.07 mmol/L), and thus was treated with aspirin and atorvastatin with oral sodium valproate 200 mg BD for seizure control. The formal plain CT brain report showed multifocal cortical calcifications and cerebral hypodensities suggestive of cortical tubers, to consider tuberous sclerosis. However, upon review by neuromedical and infectious disease (ID) physicians, a diagnosis of neurocysticercosis was proposed. Oral dexamethasone was started, and anticonvulsant was continued while aspirin and atorvastatin were ceased. A further contrast-enhanced CT brain (CECT) was done, which supported the diagnosis of neurocysticercosis, due to the presence of multiple intracranial calcifications with perilesional oedema and rim enhancement. Enzyme Linked Immunoabsorbant Assay (ELISA) test for serum IgG antibodies to *T. solium* cysticercosis (DRG, Russia) was positive (> 0.3 OD units). The patient subsequently had an MRI brain which showed multiple cystic lesions mainly seen at the grey-white matter junction and subarachnoid space of both cerebral hemispheres, associated with perilesional oedema, suggestive of neurocysticercosis (as shown in Figure 1). Anthelmintic was only initiated after the extra-parenchymal lesions had been identified from the MRI. He was started on oral albendazole 800 mg OD for 8 days, with oral phenytoin 300 mg ON and a plan to taper down oral dexamethasone over a period of 4 weeks. The patient was to be reviewed in an Infectious Disease Clinic two weeks later, but regrettably defaulted the follow-up.

DISCUSSION

The infection of *T. solium* in humans can cause either taeniasis or cysticercosis although sometimes both can occur concurrently. Humans acquired taeniasis through the ingestion of *T. solium* larval cysts (cysticerci) from undercooked of infected pork, in which humans become the definite host for the intestinal adult *T. solium*. The adult *T. solium* may survive for 10 to 20 years in the human intestine (2). Humans with taeniasis (carriers) of intestinal adult *T. solium* are able to contaminate the environment when they defecate as their faeces may contain thousands of *T. solium* embryonated eggs in gravid proglottids. They may pass on the eggs to another human host via faecal-oral route if there is no proper personal hygiene and environmental sanitation.

T. solium cystercercosis on the other hand, is acquired by humans through ingestion of embryonated eggs of *T. solium* via contaminated food or water, or in rare occasions, autoinfection via reverse peristalsis. In both possible scenarios, the eggs will hatch in the small intestines to release oncospheres (embryos). The oncospheres then invade the intestinal wall, and are carried by blood and lymphatics, and eventually develop into cysticerci in different sites of the human tissues over 60–70 days causing cysticercosis. In neurocysticercosis, humans become the intermediate hosts for the cysticerci located in the CNS particularly the brain. Patients with neurocysticercosis may present with symptoms of seizures, altered mental states, increased intracranial pressure and even death.

It is uncertain to know when and where the patient had accidentally ingested the eggs of *T. solium* as human carriers can carry the adult worms in their intestines for a long period of time. This also means that *T. solium* neurocystercercosis can occur without the history of pork consumption as ingesting *T. solium* cysticerci from contaminated undercooked pork can only perpetuate the infection causing intestinal taeniasis. As the patient lived in a crowded flat house with other foreigner housemates, factors such as improper sanitation, poor

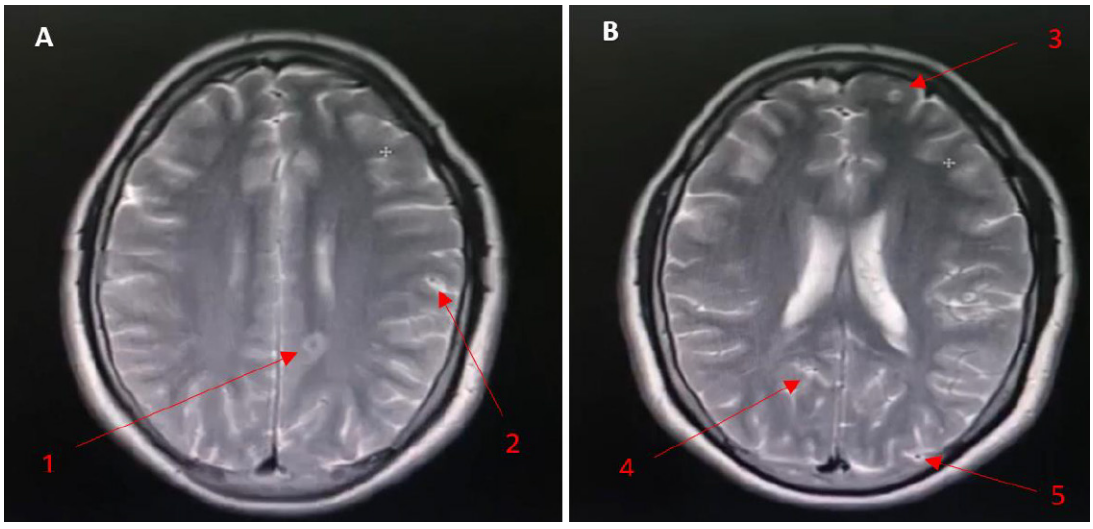

Figure 1: T2-weighted MRI, multiple cystic lesions with eccentrically target lesion mainly seen at grey-white matter junction (A1, B4) and subarachnoid space (A2, B3, B5) of cerebral hemispheres. There is associated perilesional oedema

personal hygiene and habit of eating raw vegetables or undercooked food may predisposed him to get the infection. Screening the patient and his housemates through microscopic examination for eggs or gravid proglottids of Taenia in faecal samples is one of the effective methods to find the source of infection.

Reliable diagnosis of neurocysticercosis requires a combination of clinical findings and multiple investigation tools such as neuroimaging, serology and even biopsy (usually in autopsy). Serology test such as ELISA cysticercosis IgG antibody test can be used for screening purposes. However, even with positive results, there are significant cross reactivity with other helminthic infection such as echinococcosis (2). Another serological test option is the immunoblot. It is the preferred test by Centers for Disease Control and Prevention (CDC) due to its well characterised sensitivity and specificity, as well as no reported cross reaction with other parasites (3). In both serological methods, the results are dependent on the quantity and the activity of the cysts, thus they are only useful for supporting the diagnosis of neurocysticercosis. Negative results should not exclude neurocysticercosis infection. Neuroimaging investigations such CT scan and MRI are still the most effective means of diagnosis. CT scan has the advantage of identifying dead calcified cysts, while the MRI is superior in terms of evaluating inflammatory changes in the brain, non-calcified cysts and ventricular involvement due to its higher contrast resolution (4). The only pathognomonic neuroimaging findings of neurocysticercosis is the rounded cystic lesions with an eccentric bright dot representing the scolex (4). Without that unique feature, radiological findings of cystic cerebral lesions may suggest other differential diagnosis, including abscess, tubercle, subacute CVA or metastasis. These can be differentiated between one another with good history taking, thorough examination and correct investigation tools.

Treating neurocysticercosis with anthelmintic is not a medical emergency in almost all of the cases. Initial therapy should emphasise on controlling the seizures, intracranial hypertension, cerebral oedema and treatment of hydrocephalus if present. Several studies suggest that albendazole may be superior to praziquantel in terms of choice of anthelmintic therapy. CDC suggests oral albendazole 15 mg/kg/day in two separate doses for two weeks (3). Albendazole has high efficacy for treatment of neurocysticercosis as its metabolites can penetrate the CNS better to achieve higher brain levels than in plasma (1). Anthelmintic should be used with great caution in this case, due to the involvement of neurocysticercosis in the subarachnoid space of both cerebral hemispheres (extra-parenchymal). The anthelminthic therapy was given for a total of 8 days in conjunction with corticosteroids to prevent overwhelming immunological response such as reactive cerebral oedema caused by the death of cyst (5).

CONCLUSION

Neurocysticercosis could pose a diagnostic challenge for clinicians in Malaysia as the disease is rarely seen in local population. However, the disease should be part of differential diagnosis of acute seizure especially among immigrant workers. Thorough history taking, detailed physical examination and prompt usage of multiple investigation modalities including serology, neuroimaging and even biopsy should be able to aid clinicians to achieve a conclusive diagnosis. Treatment with albendazole or praziquantel should be used cautiously and administration of anticonvulsant and corticosteroid are crucial in controlling seizure and prevent overwhelming immunological response respectively.

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