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Neurocysticercosis in a Malaysian Muslim

Norlinah IBRAHIM MRCP, Raymond AZMAN ALI FRCP MD, Hamidon BASRI MMED, *Pramod PHADKE MD

Neurology Unit, Department of Medicine and *Department of Radiology, Universiti Kebangsaan Malaysia, Kuala Lumpur.

Abstract

To date there have been only two cases of neurocysticercosis reported in Malaysia, the first was diagnosed at autopsy in 1934, and the second was an immigrant who probably acquired the infection when she was in India. This is the report of a local born Malay woman with neurocysticercosis who presented with headache, and confusion. CT brain scan showed pathognomonic cysts with invaginated scolex. Serological testing for cysticercosis using enzyme immunoassay was strongly positive. The clinical symptoms and signs and brain lesion resolved with cysticidal therapy. Other than Mecca for pilgrimage, the patient has not traveled overseas. As taeniasis is not known among the local population, the infection was probably acquired from the immigrant workers who were food handlers. The case showed the importance of screening for cysticercosis among food handlers from cysticercosis endemic areas.

INTRODUCTION

Neurocysticercosis is one of the commonest parasitic infections of the central nervous system worldwide and is an important cause of acquired epilepsy especially in endemic areas such as Latin America, Sub-Saharan Africa, India and parts of South East Asia and China.[1] It is also an emerging disease in the United States due to an increase in the number of immigrants from endemic areas. As the life cycle of the parasite involves pig, the disease is almost non-existent in Muslim countries.[2] Neurocysticercosis is rarely reported in Malaysia. To date there have been only two case reports of neurocysticercosis in Malaysia. The first was a Malaysian Chinese at autopsy in 1934[4] and the other was an immigrant from India.[3] This is the report of a Malay Muslim with neurocysticercosis, who probably acquired the disease locally.

CASE REPORT

A 59-year-old local born Muslim Malay lady presented with a one-month history of progressive frontal headache, fever and intermittent confusion. The headache was nocturnal and interfered with her sleep. She denied any photophobia or vomiting. There was no contact with patients with similar symptoms. She was diagnosed with bronchial asthma 5 years previously and was on regular bronchodilators. She also had a large goitre, which was noted about 18 years prior to presentation for which the patient did not seek any medical treatment. There was no history of use of traditional medicine. She was married and lived with her husband who worked as a farmer in an oil palm plantation in Jerantut, Pahang. She has previously not travelled overseas, other than for pilgrimage to Mecca. She denied any consumption of pork or porcine products. On examination, she was confused with a Mini Mental State Examination (MMSE) score of 23/30. The rest of neurological examination was normal with no meningism. A large multinodular goitre was noted which was clinically euthyroid. Examination of the other systems was unremarkable.

Further investigations showed normal full blood count, renal profile, serum calcium and magnesium. The erythrocyte sedimentation rate was raised at 61mm/hr. The blood film analysis was negative for Plasmodium. Blood, urine and sputum cultures did not yield any growth. The Widal-Weil Felix and HIV screening were also negative. The serum TSH was < 0.06mU/l and the free T4 was 17.6 pmol/l. The antinuclear antibody, rheumatoid factor and serum complements C3 and C4 were also negative.

Address correspondence to: Dr Norlinah Ibrahim, Department of Medicine, Universiti Kebangsaan Malaysia, Jalan Yaacob Latif, Bandar Tun Razak, 56000 Cheras, Kuala Lumpur, Malaysia

The CT brain scan showed multiple ring enhancing well circumscribed lesions, 5-10mm in the periventricular white matter and centrum semiovale with no perilesional oedema. There were central mural nodules seen within some of these lesions (Figure 1). Brain MR imaging showed multiple non-enhancing round lesions of low signal intensity on T1 weighted images and high signal intensity on FLAIR sequences and T2 weighted images. The lesions were in the periventricular area, basal ganglia, brain stem, centrum semiovale and corpus callosum. CT scan of the neck revealed a large benign-looking multinodular goitre with retrosternal extension.

Lumbar puncture revealed an opening pressure of 11cm H_2O. No acid-fast bacilli, cryptococci or other pathogens were isolated either on direct microscopy or culture. The cerebrospinal fluid glucose and protein were normal. The tests for cryptococcal antigen and polymerase chain reaction for tuberculous DNA in cerebrospinal fluid and cryptococcal antigen in serum were all negative. A skeletal survey did not show any rectilinear calcifications in the subcutaneous tissues. Stool testing was negative for ova, however coproantigen testing for *Taenia solium* was not done. Serological testing for cysticercosis using enzyme immunoassay (EIA) was strongly positive.

A diagnosis of neurocysticercosis was made and the patient was treated with albendazole 200mg three times daily for one month. She was discharged well and a repeat CT brain scan two months following presentation showed almost complete resolution of the lesions. The patient remained well at 5 months follow up.

DISCUSSION

The clinical manifestations of neurocysticercosis are diverse, ranging from asymptomatic, mild, severe to life threatening infection with residual neurological sequelae, depending on the number, size, localization of cysts, and hosts' immune response. Epilepsy is the commonest presenting illness and has been reported in 70 – 80% of patients.[5] Obstructive hydrocephalus is seen in patients with intraventricular cysts, ependymitis or arachnoiditis.[6] Other rare manifestations include spinal[6] and retinal cysticercosis.[7] A 'non-

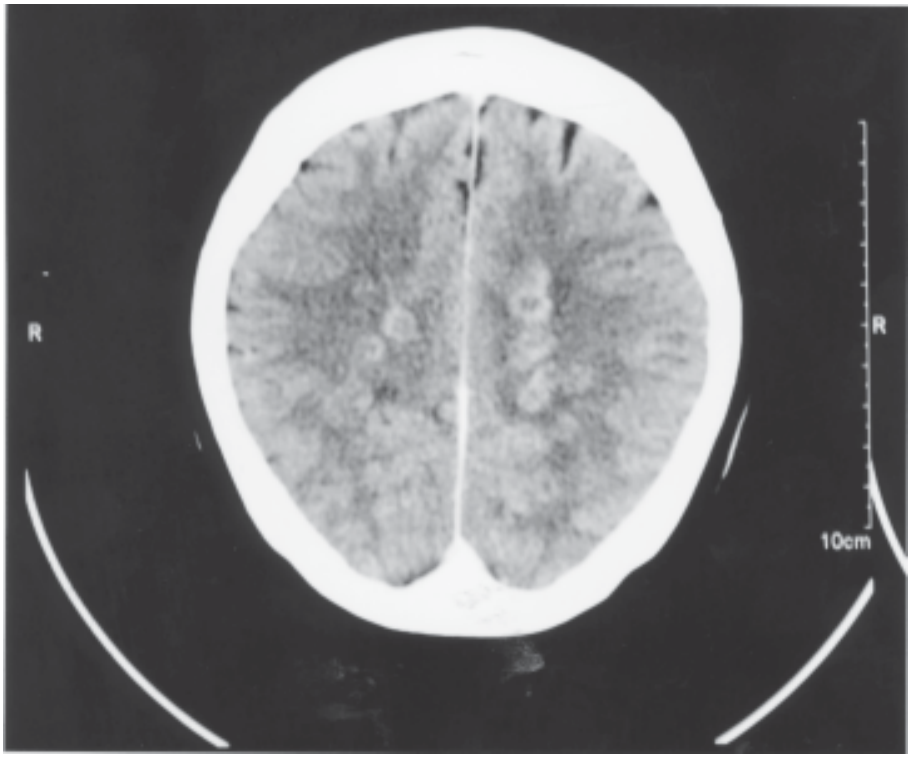

Figure 1a. Axial section of contrast-enhanced CT brain scan showing multiple well circumscribed ring enhancing rounded lesions with central mural nodule and minimal surrounding oedema.

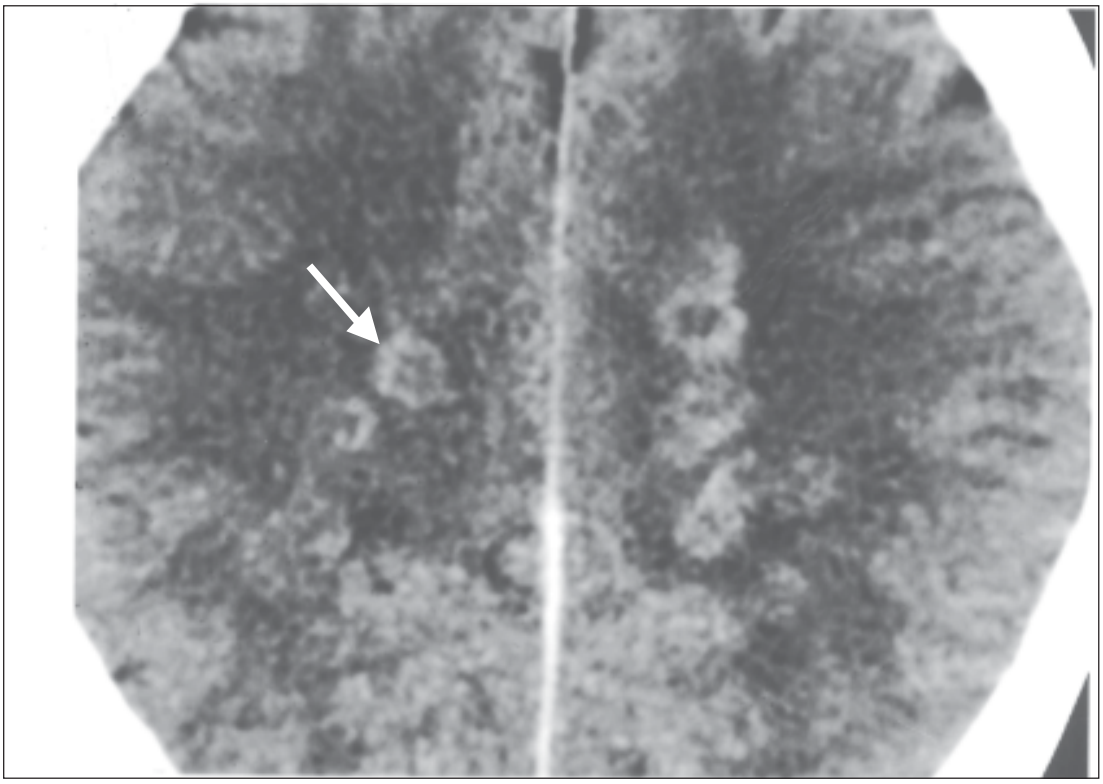

Figure 1b. Enlarged view with arrow showing a central mural nodule.

encephalitic' variant has recently been described with heavy intraparenchymal cysts, undetectable inflammatory reaction in neuroimaging, and relatively benign clinical course.[8]

Our patient presented with headache, fever and confusion over one month, which were features of chronic meningoencephalitis. The cerebrospinal fluid examination showed that infective causes from cryptococcosis and tuberculosis were unlikely. The presence of central scolex on CT brain scan fulfilled the absolute criteria for the recently proposed diagnostic criteria of neurocysticercosis.[6] The imaging findings of multiple supratentorial, parenchymal lesions smaller than 20mm, not associated with displacement of midline structures, were typical of cysticercal cysts. With the positive serology, she had two major criteria for the diagnosis of neurocysticercosis.[6] The presence of clinical findings consistent with neurocysticercosis, and the good response to albendazole were minor criteria for the diagnosis of neurocysticercosis.[6]

Taeniasis, an intestinal tapeworm infection from *Taenia solium,* is acquired by eating undercooked pork contaminated with cysticerci or "measly" pork.[2] Neurocysticercosis is an infection of the central nervous system from the larval forms of the pork tapeworm. Neurocysticercosis is acquired by ingesting taenia eggs shed in the feces of a human carrier of the tapeworms. This may occur in the human carrier with taeniasis by auto ingestion via faecal-oral route. Our patient, being a devout Muslim strongly denied any ingestion of pork or porcine products. Furthermore, examination of her stool for tapeworm ova was negative. She was unlikely to be a human carrier with taeniasis. Auto ingestion as a source of neurocysticercosis was unlikely.

Her infection was probably acquired via faecal-oral transmission from a chronic human carrier with taeniasis. With systemic examination of carcasses in the abattoirs, and the local practice of thorough cooking of pork before human consumption, there has been no report of taeniasis in the local population among both Muslims and non-Muslims. On the other hand, over the last two decades, there has been influx of 1.5 million immigrant workers in Malaysia. Some of the immigrants were from countries endemic for cysticercosis, such as India and Nepal, and parts of Indonesia.[9] A previous serological survey showed that 0.05% of the immigrants was positive

for *Taenia solium*.[3] Some of the immigrant workers were employed as food handlers. These workers can be the source of *Taenia solium* eggs giving rise to neurocysticercosis of the local population, including the Muslim population even though they do not consume pork. In support of this hypothesis, there have been reported cases of neurocysticercosis among the Orthodox Jews living in New York City who adhered to Jewish dietary law, which forbid the eating of pork. The infection was attributable to the transmission of the eggs by household workers who were recent immigrants from Latin American countries where *Taenia solium* is endemic.[10] To our knowledge, the present case of neurocysticercosis may be the first reported among Muslims.

As mentioned earlier, to-date there has been only two case reports of neurocysticercosis in Malaysia. The first was a Malaysian Chinese at autopsy in 1934[4] and the other was an immigrant, who was thought to acquire the illness when she was in India.[3] This is thus a recent case of neurocysticercosis, where the infection is believed to be acquired locally. The case illustrates the importance of screening for cysticercosis among food handlers who are from endemic areas.

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