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CASE REPORT

NEUROCYSTICERCOSIS, A RARE BUT NOT UNCOMMON CAUSE OF EPILEPSY.

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Abstract

Neurocysticercosis is considered to be endemic in many underdeveloped countries. In Malaysia, neurocysticercosis cases are rarely reported. We report a local born Malay Muslim man who had been diagnosed with epilepsy for the past 7 years, presented with a breakthrough seizure and altered behavior. Computed tomography (CT) scan of the brain showed multiple hyperdense lesions suggestive calcified rings with possible dead parasite within it. Toxoplasma IgG was positive while IgM was negative. Neurocysticercosis is treated with cysticidal drugs such as albendazole or Praziquantel. Albendazole is the drug of choice as its anti-parasitic effect is superior compared to Praziquantel.

Key words: Neurocysticercosis, seizure, Muslim, CT scan, albendazole.

Introduction

Neurocysticercosis, in the larvae or cyst phase of *Taenia solium* (*T. solium*) is the most common parasitic infection of the central nervous system (CNS), compared to *T. saginata* which is innocuous, predominantly affect the gastrointestinal tract. It is the primary cause of acquired epilepsy in most underdeveloped as well as developed countries and is attributed to high migration rates and international travel.[1, 2] The major clinical manifestations of taeniasis are seizures, headache, and focal neurological deficits resulting in complications such as epilepsy, hydrocephalus, cognitive dysfunction and dementia.[2, 3] Diagnosis of neurocysticercosis is mainly by history supported by neuroimaging and serological assessment.[1] Cysticidal drugs such as praziquantel and albendazole are drugs of choice.[2] We report a local born Malay Muslim man who had been diagnosed with epilepsy for the past 7 years, presented with a breakthrough seizure and altered behavior. The diagnosis of neurocysticercosis was made following a CT scan of the brain.

Case report

A 50-year-old Malay Muslim man presented with two episodes of generalised tonic clonic seizures. He was diagnosed with epilepsy for the past 7 years which had been poorly controlled (1-2 episodes per month) due to poor compliance. He was noted to have altered behavior 2 weeks prior to admission, talking incoherently and somnolent. There was no aura, post-ictal drowsiness, loss of consciousness or Todd's paralysis. Further history revealed that he had recurrent seizures since 15 years old and had a motor vehicle accident following a fitting episode in 2012 when he was diagnosed and treated as neurocysticercosis based on CT scan brain. There was no other diagnostic tests or results available to confirm the diagnosis. Five-years prior to the diagnosis, he was noted to have poor memory and was a slow learner. However, he was still able to perform his daily chores, ambulate and ride his motorcycle. Neither had he any high risk behavior or any previous blood transfusion nor consumed forbidden food especially pork. In 2012, he was given phenytoin and albendazole. As his seizure was not well controlled, sodium valproate, and lamotrigine was then added.

At the Emergency Department, his Glasgow Coma Scale (GCS) was 5/15, confused, disorientated and restless. Other systemic examination were unremarkable.

Laboratory investigations reveals leukocytosis, $20.1x10^9$/L (4.0 – 11.0 x 109/L) with neutrophilia, 88.9% (40-75%) and lymphopenia, 5.3% (20-45%). Renal and liver profiles, and blood glucose levels were normal. Serum calcium and magnesium were normal (2.4 mmol/L and 0.9 mmol/L). The Human Immunodeficiency Virus (HIV), Hepatitis B and C screening were negative. Blood culture and sensitivity was also negative. Lumbar puncture was not performed. Serum for Toxoplasma IgG was positive. CT brain revealed multiple hyperdense lesions involving both cerebral hemispheres and brain stem but there was no associated perilesional oedema or mass effect and no midline shift (Figure 1). Oral albendazole 400 mg twice daily was given once. Over the ensuing days of hospitalization, his seizure was controlled with intravenous phenytoin 100 mg three times a day, intravenous diazepam as needed, lamotrigine 50 mg daily, sodium valproate 600 mg twice daily, and folate 5 mg daily.

Discussion

Neurocysticercosis (NCC) is a common parasitic infestation caused by a tapeworm, *T. solium* also known as pork tapeworm, involving either parenchyma or extra-parenchymal structures of the brain [1, 2, 3] which may causes acute epilepsy in humans. [4] Ingestion of contaminated food or drinks with the egg or ova of the parasite is the main mode of transmission that results in taeniasis.

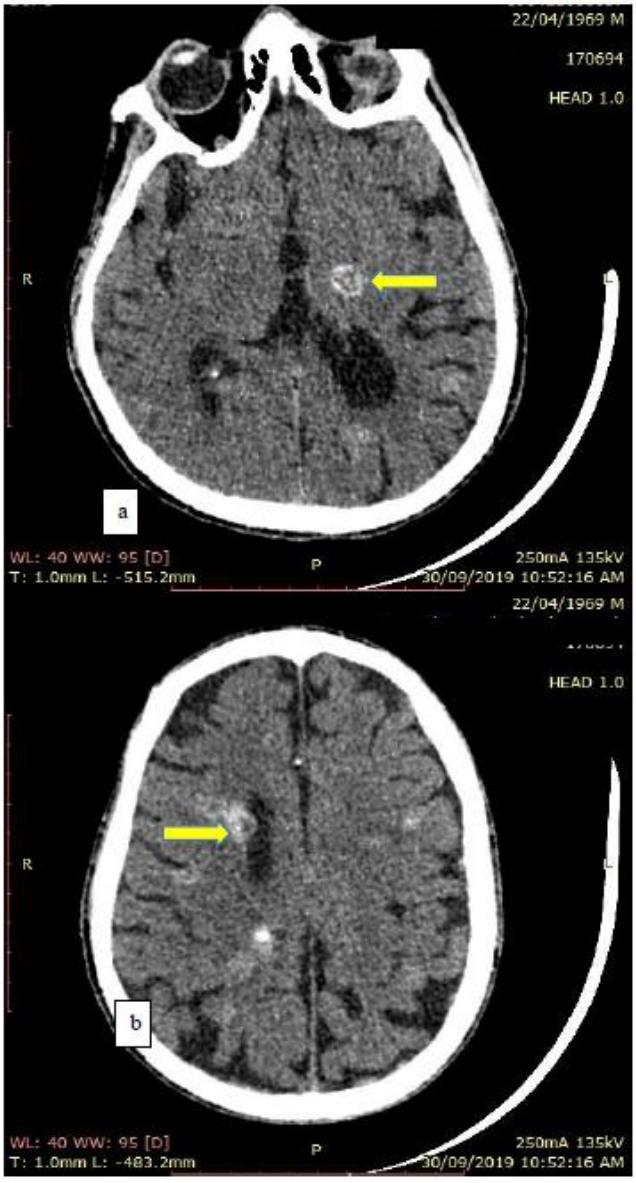

Figure 1a, b. CT scan brain showing granular nodular stage of NCC - multiple scattered small hyperdense lesions in both cerebral hemispheres (involving cortical and subcortical regions), periventricular, bilateral cerebellum and brain stem. There is minimal perilesional hypodensity seen at some of the lesions with no significant mass effect, intracranial bleed, midline shift or hydrocephalus. The basal cisterns are patent. Scolex is seen as an eccentric focus of enhancement.

It is endemic in South East Asia, India, China, Latin America, Eastern Europe, and also Africa. [5, 6] However it is quite rare in Malaysia but there have been a few case reports. [5, 7, 8] The global prevalence is still unknown. Although pork is strictly forbidden in Islam, accidental faecal-oral transmission through either cross-contamination of food or in the fields where defecation takes place may explain the possibility for this patient contracted the disease.

The clinical manifestation is heterogenous depending on the site of cysticerci in the central nervous system, i.e. parenchymal or extra parenchymal. Seizures is the most common manifestation of NCC.[9] Diagnosis may be difficult due to the varied presentations and NCC may remain asymptomatic for long periods as the cyst remain undetectable due to complex immune response.[10] This patient had multiple fitting episodes since childhood and subsequently developed behavioral changes in adulthood which was thought to be epileptic psychosis or organic brain syndrome. The CT brain findings of calcifications in both cerebral hemispheres is not pathognomonic of NCC as similar changes can be seen after recovery from other infections such as tuberculoma, and toxoplasmosis, although certain neuroimaging features may strongly indicate NCC such as the presence of scolex and other evolutionary stages. [11, 12]

However, neurocysticercosis was considered the most likely diagnosis in this patient based on the clinical presentation and CT scan findings. There is controversy whether the seizures are triggered by calcified NCC.[13] It has been suggested that calcified cysticerci may be active clinically and pathologically, and may trigger recurrent seizures due to host immune system response during the process of remodeling.[14] The toxoplasma IgG positivity was most likely due a cross reaction. The serological test result for cysticercosis in this patient could not be traced as this had been done many years ago.

The prognosis of NCC depends on the parasite load, multiplication of cysticerci, and favorable parenchymal involvement.[15] The diagnosis of parenchymal NCC in this patient was based on recurrent seizures, highly suggestive CT scan findings of a combination of parenchymal cysticerci in different stages of evolution: vesicular with or without scolex, degenerative (colloidal or nodular), and calcified fulfilling the new NCC diagnostic criteria, and for parenchymal NCC, with a sensitivity of 89.8% and specificity of 80.7%. [16]

Immunological testing using the enzyme‐linked immunosorbent assay (ELISA) and enzyme‐linked immunoelectrotransfer blot (EITB) assay are usually used, either in sera or in CSF to detect antibody or antigen.[17, 18] The sensitivity is higher in sera of patient with parenchymal sites EITB compared to extra parenchymal which is higher in CSF. [19] Serology test is an unreliable diagnostic tool as its sensitivity and specificity are relatively poor and may yield false positive results in 50% of NCC patient with solitary cyst or calcification alone. [20]

Albendazole or praziquantel are the cysticidal drugs of choice with the former more preferable and effective in symptomatic patient with viable cyst and offers a better outcome.[21] Steroids as an adjunct therapy also helps to control the acute inflammation due to immune response triggered following degradation of viable cysts and as a consequence of cysticidal treatment.[22]

Conclusion

Neurocysticercosis (NCC) is uncommon in Malaysia but it is still the commonest parasitic infection of human central nervous system and the commonest cause of acute and recurrent seizures. Early diagnosis is crucial but can be difficult because of the varied clinical manifestations. CT scan findings and immunological tests are the diagnostic tools to confirm NCC. The prognosis parenchymal type is good as long as the symptoms are controlled well and patient is compliant to medication.

Conflict of Interest

The authors declared no conflict of interest.

References

1. Garcia HH, Del Brutto OH; Cysticercosis Working Group in Peru. Neurocysticercosis: updated concepts about an old disease. Lancet Neurol. 2005 Oct;4(10):653-61. doi: 10.1016/S1474-4422(05)70194-0. PMID: 16168934.
2. Carpio A. Neurocysticercosis: an update. Lancet Infect Dis. 2002 Dec;2(12):751-62. doi: 10.1016/s1473-3099(02)00454-1. PMID: 12467692.
3. Del Brutto OH. Neurocysticercosis. Neurohospitalist. 2014;4(4):205-212. doi:10.1177/1941874414533351
4. Rajshekhar V. Neurocysticercosis: Diagnostic problems & current therapeutic strategies. Indian J Med Res. 2016;144(3):319-326. doi:10.4103/0971-5916.198686
5. Sahu PS, Lim YAL, Mahmud R, Somanath SD, Tan CT, Ramachandran CP. Needs of exploring the burden of recent onset seizures due to neurocysticercosis and challenges in

southeast Asia focusing on scenario in Malaysia. Asian Pac J Trop Med. 2017;10(4):332-340. doi: 10.1016/j.apjtm.2017.03.024. PMID: 28552103.

6. Garcia HH, Del Brutto OH, Nash TE, White AC Jr, Tsang VC, Gilman RH. New concepts in the diagnosis and management of neurocysticercosis (Taenia solium). Am J Trop Med Hyg. 2005 Jan;72(1):3-9. PMID: 15728858.

7. Chew NK, Tan CT, Goh KJ, Kamarulzaman A, Paul G, Ramli N, Anuar K. The first case of neurocysticercosis diagnosed ante-mortem in Malaysia. Neurol J Southeast Asia 2001;7:135-138.

8. Ibrahim N, Ali RA, Basri H, Phadke P. Neurocysticercosis in a Malaysian Muslim. Neurol J Southeast Asia 2003; 8:45-48.

9. Carabin H, Ndimubanzi PC, Budke CM, Nguyen H, Qian Y, Cowan LD et al. Clinical manifestations associated with neurocysticercosis: a systematic review. PLoS Negl Trop Dis. 2011;5(5):e1152. doi:10.1371/journal.pntd.0001152

10. Kimura-Hayama ET, Higuera JA, Corona-Cedillo R, Chávez-Macías L, Perochena A, Quiroz-Rojas LY, et al. Neurocysticercosis: radiologic-pathologic correlation. Radiographics. 2010 Oct;30(6):1705-19. doi: 10.1148/rg.306105522. PMID: 21071384.

11. Del Brutto OH, Rajshekhar V, White AC Jr, Tsang VC, Nash TE, Takayanagui OM, et al. Proposed diagnostic criteria for neurocysticercosis. Neurology. 2001 Jul 24;57(2):177-83. doi: 10.1212/wnl.57.2.177. PMID: 11480424; PMCID: PMC2912527.

12. Del Brutto OH. Neurocysticercosis: a review. ScientificWorldJournal. 2012;2012:159821. doi: 10.1100/2012/159821.

13. Nash TE, Del Brutto OH, Butman JA, Corona T, Delgado-Escueta A, Duron RM, et al. Calcific neurocysticercosis and epileptogenesis. Neurology. 2004 Jun 8;62(11):1934-8. doi: 10.1212/01.wnl.0000129481.12067.06..

14. Nash TE, Pretell EJ, Lescano AG, Bustos JA, Gilman RH, Gonzalez AE, et al. Cysticercosis Working Group in Peru. Perilesional brain oedema and seizure activity in patients with calcified neurocysticercosis: a prospective cohort and nested case-control study. Lancet Neurol. 2008 Dec; 7(12):1099-105.

15. Carpio A, Fleury A, Hauser WA. Neurocysticercosis: five new things. Neurol Clin Pract 2013;3:118–125

16. Carpio A, Fleury A, Romo ML, Abraham R, Fandiño J, Durán JC, et al. New diagnostic criteria for neurocysticercosis: Reliability and validity. Ann Neurol. 2016 Sep;80(3):434-42. doi: 10.1002/ana.24732.

17. Tsang VC, Brand JA, Boyer AE. An enzyme‐linked immunoelectrotransfer blot assay and glycoprotein antigens for diagnosing human cysticercosis (Taenia solium). J Infect Dis 1989;159:50‐59.

18. Brandt JR, Geerts S, De Deken R, Kumar V, Ceulemans F, Brijs L, et al. A monoclonal antibody-based ELISA for the detection of circulating excretory-secretory antigens in Taenia saginata cysticercosis. Int J Parasitol. 1992 Jul;22(4):471-7. doi: 10.1016/0020-7519(92)90148-e.

19. Michelet L, Fleury A, Sciutto E, Kendjo E, Fragoso G, Paris L, et al. Human neurocysticercosis: comparison of different diagnostic tests using cerebrospinal fluid. J Clin Microbiol. 2011 Jan;49(1):195-200. doi: 10.1128/JCM.01554-10.

20. Rajshekhar V, Murthy JM, Prabhakar S, Modi M, Khandelwal N, Garcia HH. A diagnostic and therapeutic scheme for a solitary cysticercus granuloma. Singh G. Neurology. 2010 Dec 14; 75(24):2236-45.

21. Sotelo J, Diaz-Olavarrieta C. Neurocysticercosis: changes after 25 years of medical therapy. Arch Med Res. 2010 Jan; 41(1):62-3.

22. Nash TE, Mahanty S, Garcia HH; Cysticercosis Group in Peru. Corticosteroid use in neurocysticercosis. Expert Rev Neurother. 2011;11(8):1175–1183. doi:10.1586/ern.11.86