Fundamental Neurosurgery

Surgical remotion of a cysticercotic granuloma responsible for refractory seizures: A case report

Md. Shariful Hasan, Hamidon Bin Basri, Lim Poh Hin, Johnson Stanslas¹

Neurology Unit, ¹Pharmacotherapeutics Unit, Department of Medicine, Faculty of Medicine and Health Sciences, University Putra, Malaysia

E-mail: *Md. Shariful Hasan - shariful_hsn@yahoo.com; Hamidon Bin Basri - hamidon@medic.upm.edu.my; Lim Poh Hin - phlim@medic.upm.edu.my; Johnson Stanslas - rcxjs@medic.upm.edu.my

*Corresponding author

Received: 4 October 11

Accepted: 2 November 11

Published: 13 December 11

This article may be cited as:

Hasan MS, Basri HB, Hin LP, Stanslas J. Surgical remotion of a cysticercotic granuloma responsible for refractory seizures: A case report. Surg Neurol Int 2011;2:177. Available FREE in open access from: http://www.surgicalneurologyint.com/text.asp?2011/2/1/177/90698

Copyright: © 2011 Hasan MS. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Abstract

Background: Neurocysticercosis is the most common parasitic infestation of the central nervous system and an important cause of acquired epilepsy. Although endemic in developing countries, with an increased immigration from the endemic regions, it is also seen progressively in other parts of the world. Hence, there is an increased need for awareness of neurocysticercosis in the non-endemic areas.

Case Description: The case described here is of a 13-year-old girl who presented with refractory seizures. She had been on antiepileptic medication and had also received anti-parasitic treatment for neurocysticercosis. Surgical intervention was recommended because the seizures were resistant to treatment and also because the diagnosis could not be clearly established. Following surgery, the seizures have been under control and the patient has been doing well.

Conclusion: Neurocysticercosis can be a potential cause of refractory seizure even in non-endemic countries. Some cases may be difficult to diagnose. Clinical presentation of seizure and brain imaging should be given priority over blood investigations for diagnosing neurocysticercosis and advanced neurosurgical intervention can be considered in suitable cases for better outcome.



Key Words: Neurocysticercosis, refractory seizures, stereotactic craniotomy

INTRODUCTION

Humans are the definitive hosts while pigs are the intermediate hosts in the life cycle of *Taenia solium*. Cysticercosis occurs when human replaces the pig as an intermediate host by accidentally ingesting the eggs of *T. solium* through feco-oral transmission, generally seen in regions with poor hygienic and sanitary conditions. The eggs migrate and develop into cysts most often in the brain and less commonly in the muscles, subcutaneous tissues or eyes. The clinical manifestation may be varied

depending on the location, number, size, viability of the cyst and the host immune response. Focal or generalized seizure is the commonest presenting feature in parenchymal neurocysticercosis. The diagnosis is usually made by integration of the clinical picture, epidemiological data, and the evidence from immunological tests and neuroradiological investigations.^[2] Though it may not always be the case, the diagnosis is sometimes not clear. Nonspecific and varied clinical presentation, non-pathognomic neuroradiological findings, and low sensitivity and specificity of some serological tests can at

Surgical Neurology International 2011, 2:177

times make diagnosing neurocysticercosis a challenging task. In case of uncertain diagnosis and also in refractory cases, surgical intervention could be an important mode of treatment. The current case highlights this particular aspect in the management of neurocysticercosis.

CASE REPORT

The patient is a right-handed girl originally from India who migrated to Malaysia. At the age of 9 years, she had a simple partial seizure with shaking of her right hand that lasted for 1 minute, whilst in India. A computed tomography (CT) brain scan at that time showed a white fleck in the left frontal lobe with ring enhancement and surrounding edema. She was given carbamazepine 200 mg 12 hourly, but had a second seizure about 2 months later when she was in Sweden. At this time, neurocysticercosis was suspected, but serological test was negative. She continued taking carbamazepine. Seven months later. CT brain scan showed the same lesion in the left frontal lobe, but the edema had disappeared. Electroencephalogram (EEG) was done after the patient had been on carbamazepine for 1 year and 7 months. Since the EEG showed no epileptiform activity, the carbamazepine dose was decreased and then stopped. Following this, the patient was well for almost 2 years; she then had a third seizure. Magnetic Resonance Imaging (MRI) brain scan showed a signal void in the left frontal lobe with ring enhancement on contrast imaging and perilesional edema. The patient was then treated with an anti-parasitic agent albendazole 400 mg 12 hourly for 1 month and an antiepileptic agent levetiracetam. An MRI scan repeated after 1 month of treatment did not show edema; there was also lesser enhancement. The antiparasitic agent was stopped at this point. Following this, the patient was symptom free for 4 months; she then had four episodes of seizures on four consecutive days. An MRI brain scan [Figure 1] when compared with the

scan that was done after the third seizure showed that the enhancement around the left frontal lobe lesion had returned and there was increased edema. A surgical excision was recommended. On a CT-guided stereotactic craniotomy, the expected calcified lesion was not found at the target site. The navigator probe localized to a sulcus with an artery running through it. A cuff of surrounding tissue was removed. The patient awoke with no problems. Postoperative CT brain scan showed the lesion was still present. The stereotactic craniotomy was therefore repeated and a calcified lesion that was found anterior and slightly deeper to the resection site was removed. The patient had some numbress of the right arm and leg after the operation. A CT brain scan [Figure 2] showed no lesion, indicating its successful removal. The patient mobilized well and was discharged after 2 days. Within a couple of days, her numbness had reduced. Histological assessment showed a cystic structure surrounded by gliotic brain. It was entirely necrotic except for several small oval structures but no scolex or cuticle with spines (features of a tapeworm). The histological picture was suggestive of a necrotic parasite, unclassified. The patient had an EEG 2 months after the surgical intervention; no epileptiform activity was observed. The dose of levetiracetam was reduced from 500 mg in the morning and 1000 mg at night to 500 mg BID. The patient was advised to continue the antiepileptic for 2 years.

DISCUSSION

Neurocysticercosis is rare in Malaysia, but with an increase in the population migrating from the endemic regions, it has become important to be aware of this particular disease, especially in patients presenting with seizures.^[4] Though the diagnosis of neurocysticercosis is not always easy, a combination of clinical picture,

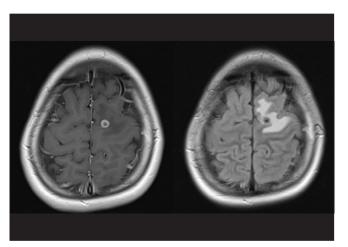


Figure I: MRI brain scan (TI with contrast and FLAIR)

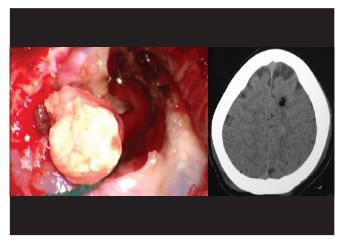


Figure 2: Intraoperative photograph showing the lesion next to the cortical incision and postoperative CT brain scan showing successful removal of the lesion

Surgical Neurology International 2011, 2:177

epidemiological data, serological tests, and neuroimaging can help to reach a diagnosis in most cases.^[2] Although in this particular case, the serological test was negative and the neuroradiological findings were not pathognomonic (cystic lesions with scolex), we can still consider the diagnosis of neurocysticercosis based on the typical clinical picture, a highly suggestive ring enhancing lesion on neuroimaging, and the fact that the patient belonged to an endemic region.^[1] Moreover, serological tests are known to be less sensitive in case of a single cyst.^[3] It has been established in previous studies that chronic calcific lesions are a cause of seizure activity, which was observed in the current case. Studies have shown an association between appearance of perilesional edema and recurrence of seizures,^[2,5] which is also seen in this case. Nevertheless, there remained some doubt about the diagnosis. Also, the seizures appeared quite refractory. Hence, it was thought that surgical intervention would be an appropriate choice of treatment. In addition, as the lesion was close to the surface, the surgical risk was not high. Removing it had a chance of curing the epilepsy, and would allow decreasing or even stopping the antiepileptic medication. Stereotactic craniotomy was therefore carried out which successfully removed the lesion. Surgical management can thus sometimes prove to be an important mode of treatment for parenchymal cysticercosis.

CONCLUSION

Though neurocysticercosis is not common in many countries, it still can be a potential cause of refractory seizure which at times may be difficult to diagnose. Clinical presentation of seizure and brain imaging should be taken as priority over blood investigations for diagnosing neurocysticercosis, and advanced neurosurgical intervention should be considered in suitable cases.

ACKNOWLEDGEMENT

The author thanks MakroCare for professional medical writing and editing services.

REFERENCES

- Del Brutto OH, Rajshekhar V, White AC Jr., Tsang VC, Nash TE, Takayanagui OM, et al. Proposed diagnostic criteria for neurocysticercosis. Neurology 2001;57:177-83.
- 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;72:3-9.
- Harrington AT, Creutzfeldt CJ, Sengupta DJ, Hoogestraat DR, Zunt JR, Cookson BT. Diagnosis of neurocysticercosis by detection of Taenia solium DNA using a global DNA screening platform. Clin Infect Dis 2009;48:86-90.
- Ibrahim N ,Azman Ali R, Basri H, Phadke P. Neurocysticercosis in a Malaysian Muslim. Neurol J Southeast Asia 2003;8:45-8.
- Nash TE, Del Brutto OH, Butman JA, Corona T, Delgado-Escueta A, Duron RM, et al. Calcific neurocysticercosis and epileptogenesis. Neurology 2004;62:1934-8.