

Occipital Lobe Syndrome Due To Giant Intraparenchymal Neurocysticercosis

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Citation

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Abstract

We report a case of radiologically proven multifocal active NCC (vesicular stage) and giant intraparenchymal cyst on the left occipital lobe also in the same stage, presenting as Occipital Lobe Syndrome.

INTRODUCTION

Neurocysticercosis (NCC) is a parasitic infection of central nervous system (CNS) caused by the larval stage (*Cysticercus cellulosae*) of the pig tapeworm *Taenia solium*. It is well known that after human ate undercooked infected pork meat then viable cysts are implanted on the small intestine, where, digestive enzymes and bile cause evagination of the scolex and attaches to the mucosa by their suckers and hooklets (Taeniasis), humans are the only known host for the adult cestodes. If humans ingest *Taenia* eggs from contaminated water/food or from oral/fecal contamination, then they will acts as intermediate host and their will develop cysticercosis, this is the most common helminthes to produce CNS infection in human being. The occurrence of acquired epilepsy or the syndrome of raised intracranial pressure in a person living in or visiting a region where Taeniasis is endemic or even in one living in close contact with people who have taeniasis should suggest a diagnosis of cysticercosis; the NCC may remain asymptomatic for months to years and sometimes its diagnosis is made incidentally when neuroimaging is performed. Symptoms and signs are related both to the parasite, and to the inflammatory-immunological response of the host. NCC is the most common cause of acquired epilepsy worldwide and most of the patients taking phenytoin or carbamazepine for a proper control of their seizures, respond very well. For interested peoples, other aspects concerning to NCC from our region, full text available at URL₁234556789*101112

CASE REPORT

A 34-years-old man presented two weeks history of altered mental state characterized by delirium (talking non sense),

weakness on the right hemibody (arm grip of hand was poor-dropping things)

He drags the right leg on walking and episodes of falling being complete unable to walk without support, he also noticed deviation of his mouth toward to the left. Intermittent frontal headache (non responsive to analgesia) vomiting, and visual hallucinations (seen goats) were also reported. No history of trauma and his family history were unremarkable. Past medical history: Secondary epilepsy since 1996 on Carbamazepine 400mg PO 8 hourly. He's been seizure free since 1997, no other chronic illnesses. On examination: He was alert, aware but confused in time and place, no meningeal signs are seen, muscle power 4/5 on the right hemibody, subtle facial nerve palsy on the right. Dysnomia for blue and red, palignosia on the left eye and visual agnosia and dyslexia were confirmed.

ELISA test for cysticercosis and other blood tests were normal. A multiplanar reconstructed CT scans of the brain revealed a right oval shaped huge cyst (106 x 69 mm) on the left parafalx temporo-parietal-occipital lobe, a similar mass (26 mm) in the right parietal lobe, masses eccentric with small calcification and 3 other mass, midline shift to the right, No enhancement was detected in any part of the calcified mass. A large cystic mass on the left occipital lobe. Some cyst had density similar to CSF, others show a turbid fluid. No anatomical structures were recognized between the cyst and the ventricular system. There was a marginal rim around the cysts and weak pericyclic oedema in the left occipital lobe. Electroencephalographic studies revealed a theta burst in the right temporal region and superimpose delta activity on the contra lateral occipital lobe, but all light

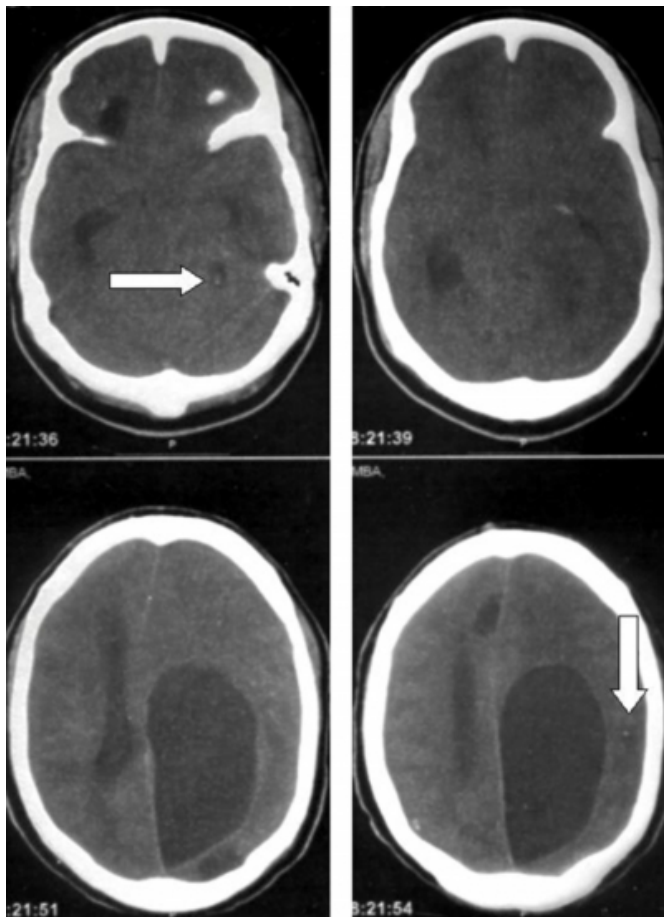
stimulation program failed to provoke additional photosensitive paroxysmal response. The results of his CSF investigations were normal.

Patient was on 500 mg of valproic acid per os 8 hourly, albendazole 800 mg per os daily for a week, and prednisone 40 mg per os daily for ten days.

CT scan of the brain done 3 weeks later revealed a only one single cyst (48 x 31 mm) on the occipital lobe and 5 weeks after CT scan showed unremarkable findings.

Figure 1

Figure 1: CT scan of the head shows a 106 mm intraparenchymal cyst on the left cerebral hemisphere and its scolex on the caudal view (horizontal white arrow); beside to this cyst another cystic lesion is also seen (vertical white arrow). NCC in colloid and calcified stage are also seen.



DISCUSSION

In our case the cysticercus appeared to be in the early degerating stage because it showed perifocal oedema, mild turbidity of the intracystic fluids, and mild enhancement of the capsule in the big one and some of them. Usually the size of cysticercus cellulosae range from 4 to 20 mm in some cases up to 50mm but 106 mm should be considered as a

giant cyst. We could not confirm on CT scan, the presence of internal septae as has been reported by Agarwal Prachi et al,^{13,14} on MRI, they believed that folding of the cyst wall gave the appearance of septation in MR imaging, we have not argument in favour or against it but we suggest that a large series should be tested to confirm this hypotheses. The presence of scolex in the cyst is a strong evidence to support a diagnosis of intraparenchymal cyst while its absence suggests: a basal cistern /subarachnoid / racemose NCC and for the other hand, we still believe that CT scan is a reliable test to identify the scolex of *Taenia solium* which can be used to rule out other diagnosis such as: arachnoidal cysts, porencephalic cyst, schicencephaly, cystic tumours particularly giant dermoid cyst (Frequently occur in the midline)¹⁵, congenital epidermoid cyst, congenital giant inter-hemisphere cysts with or without dysgenesis of the corpus callosum, and cerebral hydatid cyst.

In our patient to differentiate giant occipital cystic NCC from giant occipital hydatid cyst was only possible by identification of the scolex of *Taenia solium* at the caudal region of the cyst on CT scan.

Primary hydatid disease of the brain is a rare entity but may pose various diagnostic problems,¹⁶ we believe that the occurrence of this zoonosis that resemble cysticercosis and does not respond well to Albendazole,¹⁷ should be considered in patients from endemic areas but as was before-cited identification of scolex is decisive and its patognomonic of NCC.

PATHOGENESIS

On arrival at the left occipital lobe the cysticercus is viable and unchains inflammatory reaction in the surrounding tissues. Probable this parasite remained there for 10 or even more years (nobody knows) and because of weak immunological response of the patient to this invasion the parasite is still alive (scolex is identified, vesicular fluid is clear and membrane looks normal) but decrease resistance from the surrounding tissues cyst continued growing up gradually until get giant's dimensions causing mechanical compression over those tissues and clinical manifestations of cerebral lobe syndrome. If due to natural causes, anti-parasitic treatment (without anti-inflammatory protection) or because a complex immunological attack from the patient these parasites die, then we will expect more complications. In that situation the clear vesicular fluids will be replaced by viscous and turbid fluid (as can be seen in other samples on the same CT scan), scolex will disappear, and a thick

collagen capsule will replace the current capsule-membrane, also intense astrocytic gliosis, vasogenic oedema, microglial proliferation, perivascular mononucleocytosis and oligodendrocyte's dysfunction will be present, all these changes will cause neuronal damage and severe dysfunction of the electrical and chemical transmission with its corresponding consequences. Cysticercus on the brain in different stages are well documented by CT scan, we believe that there are parasites in different stages because they did not arrive to CNS at the same time therefore they were not affected by the immunological system in the same way, while some of them are under protection from inhabitation of the classical complement activation pathway (antigen B-C1q) other remains unprotected from ineffective prostaglandins and many other mediators.

To obtain more information about current aspects of neurocysticercosis, to review Sotelo and Del Brutto's¹⁸ article is recommended.

Based in our report giant intraparenchymal neurocysticercosis can cause incomplete occipital lobe syndrome therefore its should be included in the list of aetiological diagnosis and treatment of choice is albendazole. Other optional therapeutical approach should be considered only after albendazole's failure.

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References

1. Foyaca SH, Ibañez V, LdeF, Awotedu A, Fernandez MC. Neurocysticercosis in critical stage. Third International Congress of Critical Care Medicine on Internet. Full text available at URL: <http://www.uninet.edu/cimc2001/comunicaciones/foyaca/index.html>
2. Foyaca SH, Ibañez Valdes. Clinical trial of praziquantel and prednisone in rural patients with neurocysticercosis presenting recurrent epileptic attacks. The Internet Journal of Neurology 2002;1(2) Full text available at URL: <http://www.ispub.com/ostia/index.php?xmlFilePath=journals/ijn/vol1n2/ncc.xml>
3. Foyaca SH, Ibañez -Valdes. LdeF, Awotedu A, Fernandez MC. Neurocysticercosis in the former Transkei. 7th Internet World Congress for Biomedical Sciences INABIS 2002. Full text available at URL: http://www.inabis2002.org/poster_congress/area_01/01011/010117.pdf
4. Foyaca SH. Tapeworm and the brain. Science in Africa. June 2002;XVIII:(3) Full text available at URL: <http://www.scienceinfrica.co.za/2002/june/worm.htm>
5. Foyaca SH, Ibañez-Valdes LdeF. Intraventricular neurocysticercosis. II Virtual Congress of Neurosurgery. Online at: <http://www.neuroc.sld.cu/papers/tl-neurocys.htm>
6. Foyaca SH, Ibañez-Valdes LdeF. Intraventricular neurocysticercosis in HIV positive patients. The Internet Journal of Neurology. 2003;2(1) Full text available at URL: <http://www.ispub.com/ostia/index.php?xmlFilePath=journals/ijn/vol2n1/ncc.xml>
7. Foyaca S.H, Ibañez-Valdes LdeF. Vascular dementia type Binswanger's disease in patients with active neurocysticercosis. Electron J Biomed 2002;1(1):1-12 Full text available at URL: <http://www.uninet.edu/biomed/rebio/reb/2002/n1/foyaca.htm>
8. Foyaca S.H, Ibañez-Valdes LdeF. Enfermedad de Binswanger en pacientes con neurocisticercosis activa. IV Internet Congreso de Psiquiatria Interpsiquis 2003. Full text available at URL: <http://www.psiquiatria.com/interpsiquis2003/9638>
9. Foyaca SH, Ibañez-Valdes LdeF. Binswanger's disease and neurocysticercosis. The Internet Journal of Neurology. 2003;2(1) Full text available at URL: <http://www.ispub.com/ostia/index.php?xmlFilePath=journals/ijn/vol2n1/bins.xml>
10. Foyaca SH, Ibañez-Valdes LdeF. Pseudoseizures and Epilepsy in Neurocysticercosis. Electron J Biomed 2003;1(2):79-87 Full text available at URL: <http://www.uninet.edu/biomed/2003/n2/2foyaca.html>
11. Foyaca-Sibat H, Ibañez-Valdes LdeF. Neurocysticercosis in HIV-positive patients. The Internet Journal of Infectious Diseases 2003;2(2):1-10 Full text available at URL: <http://www.ispub.com/ostia/index.php?FilePath=Journals/ijid/current.xml>
12. Foyaca-Sibat H, Del Rio AR, Ibañez-Valdes LdeF, Vega-Novoa E, Awotedu AA. Neuroepidemiological survey for Epilepsy and Knowledge about Neurocysticercosis at Sidwadweni Location, South Africa. Electron J Biomed 2004;2(1):40-48 Full text available at URL: <http://www.uninet.edu/biomed/2004/n1/foyaca.html>
13. Suh DC, Chang KH, Han MH, Lee SR, Han MC, Kim CW. Unusual manifestation of neurocysticercosis. Neurology 1989;31:396-402
14. Agarwal Prachi P, Gaikwad SB, Garg A, Gupta V, Mishra NK, Mehta VS. Giant intraparenchymal neurocysticercosis: Unusual MRI findings. Neurol India 2004;52(2):259-260
15. Carbanz, Kemerdere R, Ocal E, Tanriverdi. Intracranial dermoid cyst mimicking a giant thrombosed aneurysm. Neurol India 2004;52(4):524-525
16. Miabi Z, Hashemi H, Ghaffarmour M, Ghelichnia H, Media R. Clinico-radiological finding and treatment outcome in patients with intracranial hydatid cyst. Act Med Iran. 2005;43(5):359-364
17. Ciurea AV, Vasilescu G, Nuteanu L, Carp N. Cerebral hydatid cyst in children. Experience of 27 cases. Child's Nervous System 1995;11(12):679-685
18. Sotelo J, Del Brutto O. Review of neurocysticercosis. Neurosurg focus. 2002;12(6):1-6

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