Available online at http://www.ijims.com
ISSN: 2348 – 0343

Neurocysticercosis presenting as Hemiplegia: A rare case report

Gursimran Singh*, V.B. Vikhe, Harish Chaudhari
Dept. of General Medicine, Dr. D.Y.Patil Medical College, Pune, India
*Corresponding author :Gursimran Singh

Abstract
Neurocysticercosis is a common neurological illness in India. A variety of presentations have been reported in the literature, including weber syndrome, Millard Gubler syndrome. Neurocysticercosis, manifesting as Hemiplegia, have not been reported in literature. Therefore, we report a case presented to us with right sided hemiplegia due to frontal lobe neurocysticercosis and was treated successfully.

Keywords: hemiplegia, neurocysticercosis

Introduction

Neurocysticercosis is a parasitic infection caused by larval form of pork tape warm, TaeniaSolium. It is important cause of epilepsy, headache and other neurological signs when it is located in brain, optic, Nerve or spinal cord. [1] In India, cysticercosis is highly prevalent and CNS involvement is seen in 60%–90% of infested patient. [2] Cerebrum and cerebellum are common sites but may involve brainstem, basal ganglion, thalamus, and lateral sinus. [3] Seizures are the commonest presenting feature in majority but can present with headache, focal deficits, hydrocephalus, and raised intracranial pressure. [2] We report a case of frontal lobe neurocysticercosis presenting with right sided hemiplegia.

Case History

A 14 year old male patient presented with history of acute onset of involuntary jerky movements of right upper and lower limb since 3 days which used to subside after few minutes without loss of consciousness, followed by weakness which was progressive to complete hemiplegia and used to recover completely in next 5-6 hours. There were about 2-3 episodes per day. The patient presented in state of complete weakness after last episode to hospital with no recovery even after 24 hours. He complained 2-3 episodes of vomiting with throbbing headache, there was no history of uprolling of eyeballs, bowel and bladder incontinence, tongue bite, loss of consciousness, frothing from mouth, fever, trauma, and diplopia. There was no previous significant medical or family history (tuberculosis or epilepsy).

Examination

General examination: Thin built, moderately nourished, pulse 96/min regular, BP 110/76 mmHg no pallor, clubbing, cyanosis, icterus and lymphadenopathy. All Vital examinations (Chest, CVS, Abdomen) were within normal limits.

Neurological Examination: Revealed normal mental status, no cranial nerve palsies, motor examination showed right upper and lower limb power 0/5(no power), DTR exaggerated and plantar extensor, left side power was normal, no sensory deficits were present, no signs of meningeal irritation could be detected. No involvements of cranial nerve were detected.

Laboratory Investigations

Haematological reports: Haemoglobin-12.6%; Total Leucocyte Count- 7000, Nuetrophil-72,Lymphocyte-21,Eosinophil- 2, Monocyte- 5m; ESR -2mm ; RBC- appear normocytic normochromic; no hemoparasite; HIV

Non -reactive; Bilirubin – 0.6/0.3 (Direct); ALT – 19 IU; Alkaline Phosphatase – 26 IU; Urea – 35mg%; Creatinine-0.8mg%; Serum Proteins – 8.1gm(4.1+4.0); Serum Sodium – 138; Pottasium – 4.7; Calcium – 9.8 (mg); Phosphorus – 5.6 mg; Magnesium – 6.8 (mg); Uric Acid – 6.8 (mg).

The biochemical – and cytological examination of CSF revealed protein of 50mg%, sugar 40mg% and 9 cell, all lymphocytes. PCR analysis of mycobacterial DNA was negative both in CSF and serum. Enzyme linked immunosorbent assay (ELISA) for NCC was positive both in CSF and serum.

Radiological imaging

Chest X-ray was within normal limits.

EEG showed no abnormality.

MRI: MRI is considered the best neuro-imaging tool for the detection of degenerating and innocuous (viable) cysticerci, while CT is the best for calcified lesions. [4]

Image 1:

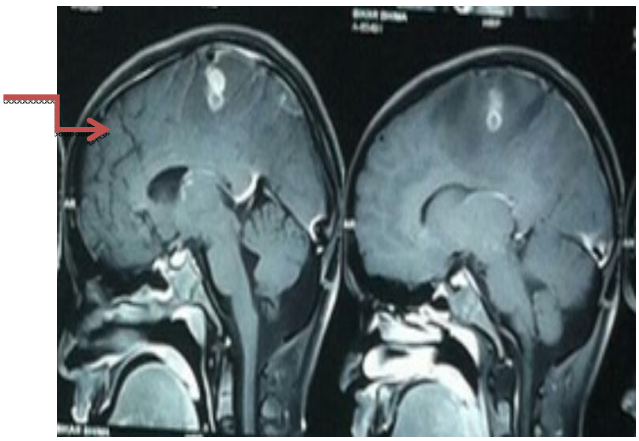
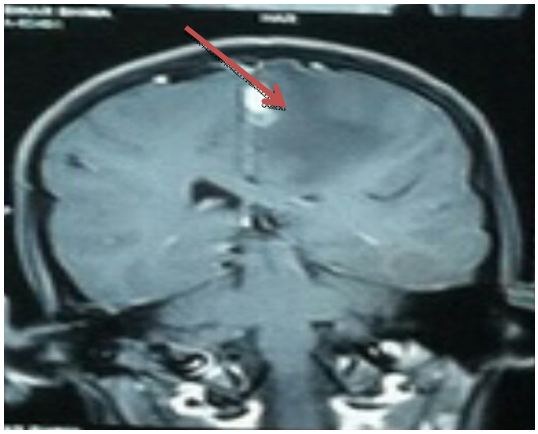


Image 2:



Two closely placed ring enhancing lesions of sizes 10 mm and 8 mm are seen in left high frontal lobe in parafalcine location. The smaller lesion is oval and shows a tiny mural nodule within. The large lesion is vertically oblongated and shows thick, irregular wall enhancemnent. No mural nodule is seen in this lesion. Significant periliesionaledema and effacement of the adjacent sulcal spaces in seen.

Whole spine screening with T2W sagittal images does not reveal any obvious abnormality. Spinal cord appears normal.

Treatment

Patient was treated conservatively [Albendazole (15mg/kg/day) in 2 doses for 8 days and Praziquantel (150mg/kg/day) for 15 days] and given systemic steroids, which resolved the symptoms completely within 7 days. No surgical intervention was carried out.

Discussion

Neurocysticercosis is a pleomorphic disease although it sometime produces no clinical manifestations. This pleomorphism is due to variations in the location of the lesions, the number of parasites and host immune response. [5] Many patients are asymptomatic, other report vague symptoms such as headache or dizziness. The

onset of symptoms is usually sub-acute to chronic with exception of seizures which present in acute fashion. [6] It's pleomorphic manifestations includes seizures, headache, and focal neurological deficits. Unusual manifestations are stroke, visual loss, ataxia, dystonia, dementia, and hydrocephalus. [2, 7] Cysticercal granulomas as cause of Weber's syndrome and ptosis have also been reported. [8] Cerebral infarction may result from obstruction of small terminal arteries or vasculitis. Intraventricular (5%–10%) and meningeal cysticerosis are associated with hydrocephalus, signs of meningeal irritation, and raised intracranial pressure. Neuroretinitis and occipital lobe syndrome have been reported in four patients of neurocysticercosis. [9]

Our patient presented with mimic palsy due to neurocysticercosis.

Patients with active cysts remain at risk of serious complication. Therefore, all patients with multiple cysts should receive treatment with steroid to reduce intracranial pressure and edema; thereafter cysticidal drug, i.e., albendazole. Efficacy of treatment should be monitored by repeat CT/MRI after 3 months.[10]

Our patient improved and became asymptomatic with steroid treatment and albendazole. Recognizing this clinical entity would avoid unnecessary medical treatment and surgical intervention. Follow-up CT scan /MRI at 4 months revealed complete resolution of the granuloma. Patient is still asymptomatic.

Summary: Based upon history, imaging results and high prevalence of disease in the region, the findings were suggestive of mimic palsy due to neurocysticercosis.

Recommendation: A point of medical and public health interest is that cysticercosis andneurocysticercosis are not reportable diseases in India. This case may serve as encouragement for authorities to reconsider the importance of this disease to be added to the list of reportable diseases

References

1. H Foyaca-Sibat, L Ibañez-Valdés Cerebellar Neurocysticercosis and Novel Hypotheses. The Internet Journal of Neurology. 2012; 14 (1): 1-60.

2. Hawk MW, Shahlaie K, Kim KD, Theis JH. Neurocysticercosis: A review. Surg Neurol. 2005; 63:123–32

3. Singhi P, Ray M, Singhi S, Khandelwal N. Clinical spectrum of 500 children with neurocysticercosis and response to albendazole. J Child Neurol.2000; 15:207–13.

4. Garcia H H and Del Brutto O H. A Imaging findings in neurocysticercosis; ActaTropica 2003;87:71–78.

5.Chaoshuang L, Zhixin Z, Diaohong W, Zhanlian H, Zhiliang G. Clinical Analysis of 52 cases of neurocysticercosis. Trop Doct. 2008; 38(3): 192-4.

6. Sawhney IMS. Singh G, Lokhra OP, Mathuriya SN, Parihar PS, Prabhakar S. Uncommon presentations of neurocysticercosis. Journal of the Neurological Sciences. 1998; 154(1): 94-100.

7. Jha S, Kumar R, Kumar R. Clinical pleomorphism of neurocysticercosis in children: Some unusal manifestations. J Pediatr Neurol. 2006;4:103–9.

8.Singh NN, Verma R, Pankaj BK, Misra S. Neurocysticercosis presenting as Weber's syndrome. Neurol India. 2003;51:555.

9. Muralidhar R, Sharma P. Optic nerve cysticercosis: Case report and review of current management. Orbit. 2005;24:159–62.

10. 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.