

UNUSUAL CLINICAL PRESENTATION OF NEUROCYSTICERCOSIS – A REPORT OF 3 CASES

C.M. Sharma, Ravindra Singh, B.L. Kumawat, Shailesh Dixit, Anjani Sharma, Arvind Kankane
Department of Neurology, SMS Medical College, Jaipur, Rajasthan, India

Abstract : Neurocysticercosis is one of the common parasitic diseases of central nervous system with variable clinical presentation. Brainstem is infrequently involved in patients with neurocysticercosis. Usually it occurs in association with disseminated form of neurocysticercosis. We are reporting three cases who had large isolated cysticercus lesions in brainstem which presented as acute neurological syndrome. The diagnosis of neurocysticercosis was established by the presence of characteristic granulomatous intraxial lesions in the brainstem along with strongly positive ELISA for cysticercal antigen in cerebrospinal fluid as well as in serum. All the three patients responded well to corticosteroids and albendazole. Repeat CT scan showed significant alteration in size of the lesion.

INTRODUCTION

Neurocysticercosis is the most common parasitic disease of the central nervous system (CNS). It occurs when man becomes the intermediate host of *Taenia solium* and harbours the larval form, *Cysticercus cellulosae*. They have special predilection for the central nervous system, skin, skeletal muscle and the eyes. It is most common in the Indian subcontinent, Central and South America, Spain and Eastern Europe¹. Its incidence is rising in the non endemic areas because of rising rates of immigration from endemic countries². Clinical manifestations of the disease are varied and depend upon topography, number, and size of the lesions, as well as status of host's inflammatory reaction against the parasite. In brain, the common sites of involvement are cerebral parenchyma, meninges and ventricles. Brainstem and cerebellum are infrequently affected, and are usually part of disseminated form of cysticercosis. We report here 3 cases of isolated cysticercus lesions in brainstem who had acute presentation.

CASE 1

A – 22 year old right handed man presented with three days history of headache followed by drooping of left eyelid. There was no history of vomiting, blurring of vision, fever or trauma. On examination patient was normotensive general physical & musculoskeletal examination was unremarkable. Neurological examination was normal except left sided partial ptosis with unilateral dilated pupil which was sluggishly reactive to light, & there was restriction of adduction in left eye.

Routine Investigations like hemogram, blood sugar and other biochemistry were normal. X-ray of chest and soft tissues of thigh did not reveal any abnormality. ELISA for cysticercal antigen in CSF and serum was positive. Serological tests for tuberculosis and HIV were negative. C.T. scan brain showed a ring enhancing lesion in the left mid brain with hyperdense nodule within it and perilesional edema (fig. 1). M.R.I. brain confirmed it to be cysticercus lesion.

The patient was primed with oral steroids (Prednisolone 40

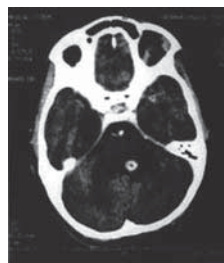


Fig.1: Ring enhancing lesion in left mid brain.

mg per day) & received albendazole (15 mg/kg/day). The neurological deficit started improving within seven days of treatment and complete recovery was seen after 15 days of treatment. Follow up CT scan after eight weeks showed complete resolution of lesion (Fig. 2).

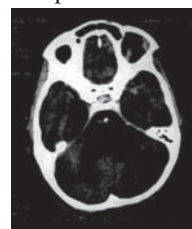


Fig. 2 : Resolution of the lesion.

CASE 2

A-9 year old right handed child presented with one day history of headache followed by drooping of right eyelid. There was no history suggestive of fever or raised intracranial pressure. General physical & neurological examination was unremarkable except right sided ptosis with dilated pupil which was sluggishly reactive to light. Routine hemogram and blood chemistry was normal and ELISA for cysticercal antigen in CSF and serum was positive. C.T. scan brain showed a ring enhancing lesion in the right mid brain with hyperdense nodule within it and perilesional edema. M.R.I. brain also confirmed, the cysticercal lesion. Similarly this patient was also primed with steroids & given albendazole. He had complete recovery within 3 weeks. Follow up CT after six weeks showed complete resolution of lesion.

CASE 3

A 18 year old right-handed female presented with two days history of headache followed by deviation of angle of mouth

Correspondence: Prof. C.M. Sharma

150, Hari Marg Civil Lines, Jaipur, Rajasthan, India, Ph-0141-2225684

toward right side, along with restriction of lateral movement of left eye. there was no history suggestive of fever or raised intracranial pressure. General physical examination was unremarkable & neurological examination revealed left lower motor neuron type of seventh nerve palsy along with left sixth nerve palsy. Routine hemogram & blood chemistry was normal & ELISA for cysticercal antigen in CSF and serum was positive. C.T. scan brain showed a ring enhancing lesion in the left pons with hyperdense nodule within it and perilesional edema (Fig.3). MRI brain also confirmed, the cysticercal lesion.

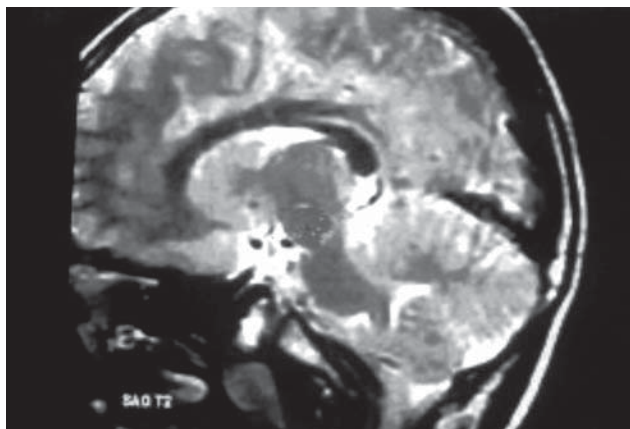


Fig.3: Ring enhancing lesion in pons.

The patient was put on oral steroids with albendazole. Their neurological deficit started improving within two weeks of treatment and follow up CT scan after eight weeks showed complete resolution of lesion (Fig.4).

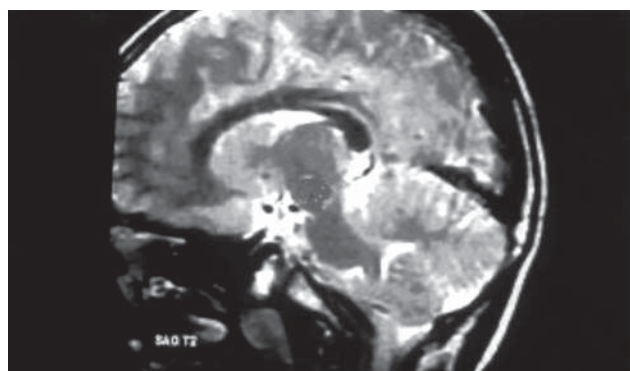


Fig.4: Resolution of the lesion

DISCUSSION

Cysticercosis, caused by taenia, is mainly a disease of gastrointestinal tract that is found predominantly in the third

world countries. Ramamurthi and Balasubramanian³ found the incidence of cysticercosis to be 1.25% of all intracranial space occupying lesion in south India. Wani et al⁴ reported an incidence of 2.5% among space occupying lesion. The common clinical features are seizures, raised intracranial tension and dementia. Uncommon presentation like pure motor hemiparesis, ataxic hemiparesis, homonymous hemianopia, cerebellopontine angle syndrome. Painful cervical radiculopathy have been reported in literature^{5,6,7}.

Focal neurological deficit is because of involvement of arterial system in subarachnoidal from the disease. The literature survey revealed involvement of brainstem along with multiple intracerebral lesions⁸. However, there are few documented cases of solitary lesion in the brainstem^{9,10,11,12}. The possible mechanism for deficit in these patients may be the lesion itself and the perilesional edema. The rapid improvement could be because of reduction in perilesional edema. The diagnosis of NCC by clinical features alone is difficult because of the varied clinical presentation. The neuroradiological features depends upon the stage of disease. The diagnosis in our cases are based on clinical data, neuroradiological features finding of specific antigen in CSF. The other common causes of acute cranial nerve palsies like diabetes mellitus, vasculitis due to intracranial infections and vascular aneurysms were ruled out. Immunoassays like ELISA and Western blot techniques are more often used these days as an adjuvant in the diagnosis because they are more specific. Purpose of our case reports is to make physicians /neurologist aware of these uncommon presentation of a treatable disease like cysticercosis so that a correct diagnosis can be made especially in tropical countries where diagnostic facilities are limited because of a paucity of resources.

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ERRATA The name of Prof. N.S. Neki, Professor of Medicine Government Medical College, Amritsar has been inadvertently missed in the list of peer reviewers published in October-December 2007 issue page 316. Prof. Neki has peer reviewed several articles in the quarterly issues of the year 2007.

Error is regretted.

Editor