

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.

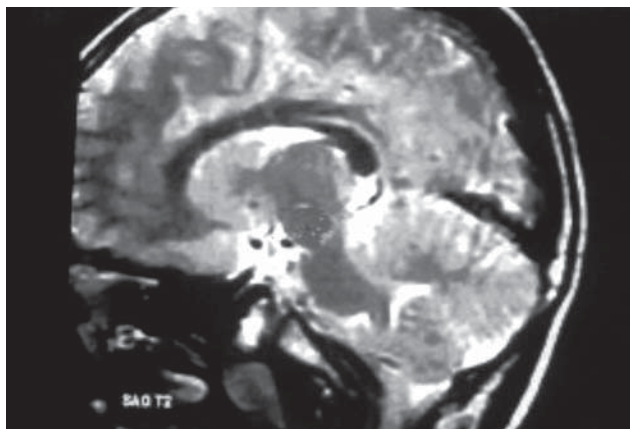


Fig.3: Ring enhancing lesion in pons.

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).

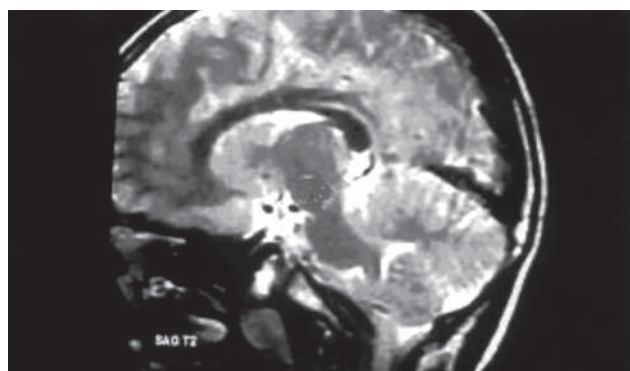


Fig.4: Resolution of the lesion

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