NEUROCYSTICERCOSIS PRESENTING AS ISOLATED THIRD CRANIAL NERVE PALSY - A CASE REPORT

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Abstract: *Neurocysticercosis has varied clinical presentation amongst which isolated oculomotor nerve palsy is infrequently presenting clinical feature. Here, we report at case of acute unilaterial oculomotor nerve palsy. The diagnosis of neurocysticercosis was established by presence of characteristic granulomatous lesion in mid brain and strongly positive ELISA for cysticercal antigen in CSF and serum. Patient responded well to medical therapy.*

INTRODUCTION

Neurocysticerosis (NCC) is the most common neurological parasitosis and continues to be a serious health problem in several states of India with poor socioeconomic status and deficint civic facilities.cysticerci have a predilection for CNS,eyes and striated muscles. The clinical spectrum of NCC is variable and ranges from asymptomatic form to symptomatic forms.The commonest presentation is presentation is partial or secondary genesralized seizures.Here,we report a case of NCC presenting as acute onset isolated unilateral oculomotor nerve palsy.

CASE REPORT

A 22 year old right-handed man presented with three days history of headache followed by drooping of left eyelid (Fig.1). There was no history of vomiting, blurring of vision, fever or trauma. On examination patient was normotensive, general physical & musculoskeletal examination was unremarkabale. Neurological examination was normal except left side partial ptosis with unilateral dilated pupil which was sluggishly reactive to light, with restriction of adduction in left eye.

Routine Investigations like hemorgram, blood glucose and other biochemistry were normal X-ray or chest and soft tissues of thigh did not reveal any abnormality. ELISA for cysitcercal 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 hyper dense nodule within it and perilesional edema (Fig-2) M.R.I. brain confirmed it to be a cysticercus lesion.

The patients was primed with oral steroids (Prednisolone 40 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 u CT scan after eight weeks showed complete resolution of lesion.

DISCUSSION

The clinical spectrum of NCC is variable and depends on the location, size, number of lesions and the magnitude of hosts inflammatory re3action against parasite in brain[2]. Other proposed mechanisms are ischemia secondary to arterial inflammation and occlusion produced by cysticercal arachnoidities[5]. The common sites of involvement in brain are cerebral parenchyma, meninges and ventricles. Brain stem and cerebellum are infrequently affected and are usually part of disseminated form of neurocysticercos[4] is but the incidence of isolated solitary cyst in brainstem is rare[3,6,7,8].

The usual presenting features of NCC are partial or secondary generalized seizures. Signs and symptoms or raised intracranial pressure and mental changes are also been and clinical presentation like begin intracranial hypertension is not uncommon. Many patients show focal neurological



deficit, which is a result of parenchyma presence of cysticerci[4].

Cysticerci are seen in unusual locations with interesting clinical findings. Mesencephalic syndrome described by Sawhney et al9 has spectrum of clinical features including upward gaze palsy, convergence retraction nystagmus, eyelid retraction, pupillaryu abnormalities and skew deviation. The underlying pathology may be necrosis or location of cyst in dorsal mid brain itself.

Our patient was interesting in view of location of the lesion in dorsal mid brain resulting in acute onset partial coulomoter nerve palsy with papillary involvement The possible mechanism for deficit in this patient may be the lesion itself. The rapid improvement could because of reduction in perilesimal edema. Thought other common causes of acute third nerve palsy are diabetes mellitus, vacuities due to intra cranial infection and posterior communicating artery aneurysm. The papillary involvement in ptosis should prompt early neuron imaging studies to exclude the space occupying lesion.

REFERENCES

1. ***Delbrutto OH, Sotelo.*** *J Neurocysticercosis A update Rev. Infect Dis 1988 10.1057-87*
2. ***McCormick G.F.*** *Cysticercosis a review of 230 patients. Bull clin neurosici 1985:50,76-101*
3. ***Rajshekhar V.E. Joseph, R. Kachnara et al****-Neurocysticercosis presenting as a Solitary cyst in the brainstem-A case report: An of Indian Academy of neurology 1999 vol. 2:99-100.*
4. ***Del Brutto OH****: Cysticerosis- cerebrovascular disease, review: J Neurol, Neursourg, Psychiatry : 1992 55: 252-54.*
5. ***Levy, A.S. Lillehei, K.O Rubinstein, D Stears, J.C. subarachnoid*** *Neurocysticercosis with occlusion of major intracranial arteries. A Case report J. Neurosurgery 1995 36 183 186.*
6. ***Chotmongkolv' suranyauisuth K Limpaawettana P. et.al*** *superior divisional occulomotor palsy caused by midbrain neurocysticercosis. Parasitol Inmto 2006 sept 55(3) 223-225.*
7. ***Garg RK. Karak B****, Neurocycticrecosis presenting as midbrain syndrome, Journal AssocPhysicians India 2000 May: 48(5) 533-5*
8. ***Singh N N, Verma, R, Pankaj B K et al****: Neurocysticercosis presenting as weber syndrome, Neurology India 2003 Dec.51(4) 551-52*
9. ***Sawhney IMS, singh G, Lekhra OP et al****, Uncommon presentation of cysticerosis, J, Neurolsci: 1988: 154:94-100.*

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