CASE REPORT

CENTRAL PONTINE AND EXTRAPONTINE MYELINOLYSIS IN ENCEPHALITIS

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ABSTRACT

A 15 years male presented with fever, convulsion, unconsciousness and dystonia of acute onset. He was investigated and treated. MRI brain shows central pontine and extrapontine myelinolysis. Osmotic injury, Wilson’s disease and other possibilities were excluded by clinical findings with supportive investigations. Patient was improved satisfactorily with treatment.

Key words: encephalitis, pontine myelinolysis, extrapontine myelinolysis.

BACKGROUND

Central pontine and extrapontine myelinolysis are rare phenomenon. It commonly occurs in osmotic demyelination syndrome (ODS), alcoholism, encephalitis (Japanese encephalitis-JE, West Nile encephalitis-WNE), and Wilson’s disease. In our case the patient was presented with features of encephalitis with extrapyramidal features like dystonia, tremor and rigidity and referred to ID & BG Hospital with suspicion of tetanus.

CASE REPORT

A 15 years old Muslim male (A. Sk) of Nadia district of West Bengal was referred to I.D. & B.G. Hospital, Kolkata with suspicion of tetanus due to rigidity of neck, unable to open mouth as a part of generalized muscle rigidity with history of small cut injury four weeks back. We examined the patient and tetanus was ruled out due to impairment of sensorium, negative spatula test and absence of provocative muscle spasm. He was ill for seven days and presented with fever, headache, vomiting, convulsion, abnormal posturing and movements of the limbs with subsequent loss of consciousness. He was nonalcoholic, of average built with normal developmental mile stones.

Examinations:

The patient was unconscious and on Ryle’s tube feeding, spatula test was negative with absence of provocative muscle spasm. Patient was normotensive (blood pressure 110/70 mm Hg) with rigid neck, generalized increased muscle tone, tremor, dystonic movement of head, mouth and limbs, fever (102°F). His respiratory, cardiovascular and abdominal findings were within normal limit. There was no jaundice, skin rash, lymphadenopathy or features of arthritis. Conjugate movements of eyes were disturbed without quadriplegia.

Investigations:

Patients investigation reports were as follows-hemoglobin 10.7 g/dl, total leukocytes count 7000/cumm with differential count as N78 L20 M1 E1 B0, ESR 97 mm, bilirubin (total) 0.62 mg %, SGPT 44IU/L, urea 40mg%, creatinine1.1mg%, Na 132mEq/L, K 3.2mEq/L, PO 4 3.9 mEq/L, Mg 1.6 mEq/L, Ca 10.1 mEq/L. Cerebrospinal fluid: cell count 04 cells/cumm, all mononuclear cells, sugar 84 mg/dl (N=45-70), protein 61.44 mg/dl (N=15-45); Ultrasonography whole abdomen and chest X-Ray showed normal findings; CT scan brain –normal study; MRI brain shows bilateral symmetrical altered signal intensity (hypointense in T1, hyperintense in T2 and FLAIR) in both basal ganglia, central pons and similar signal intensity in posterior aspect of body of corpus callosum and splenium- features were suggestive of central pontine and extrapontine myelinolysis. Twenty four hours urinary copper excretion was in normal range (20μg). KF ring was absent on slit lamp examination. Serological markers of HSV and JE virus were negative in blood and CSF.

Treatment and course

Patient was treated with acyclovir, trihexyphenidol hydrochloride (paciten), clonazepam with other supportive measures he was improved satisfactorily. He gradually became conscious and aphasia, dysarthria,
dysphagia, dystonia, tremor were improved. He was discharged on 16th day of admission in stable condition.

DISCUSSION

Encephalitis commonly presents with fever, impaired consciousness, focal or diffuse neurologic symptoms and signs with or without meningeal signs. CT or MRI may be normal but sometimes diffuse edema may be observed. Focal abnormalities are seen in eastern equine encephalitis, severe Japanese encephalitis, often with hemorrhagic thalamic lesions.1 In herpes simplex encephalitis, MRI abnormalities are found in 80-90% patients with predominant involvement of temporal lobe as increased signal intensity in T2weighted, FLAIR images. In West Nile virus encephalitis about 60% patients have MRI abnormalities involving deep brain structures like thalamus, basal ganglia and brain stem.2

Central pontine myelinolysis can occur in rapid correction of low sodium levels, alcoholism, hematopoietic stem cell transformation, underlying severe liver disease, hepatic transplantation, severe burn, malnutrition, AIDS, hyperemesis gravidarum, chronic renal failure, dehydration, acute hemorrhagic pancreatitis.3,4 Extrapontine myelinolysis commonly involves putamen, caudate nucleus, midbrain, thalamus, subcortical white matter.5 Osmotic demyelination syndrome (rapid correction of sodium level) and chronic alcoholism are responsible for more than 75% cases.6 Pons and basal ganglia involvement are commonly seen in osmotic injury, hypoxia, Wilson’s disease, Leigh disease etc but more specific for osmotic demyelination syndrome. Differential diagnosis of central pontine myelinolysis includes infarct, glioma, multiple sclerosis and encephalitis.7

In our case presentation was like encephalitis, MRI shows pontine and extrapontine myelinolysis, other causes were excluded from clinical findings and investigations. Limitation of this case is etiological diagnosis of encephalitis.

REFERENCES