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ACUTE HEMORRHAGIC LEUCOENCEPHALITIS

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ABSTRACT

Acute He morrhagic Leucoencephalitis (AHLE) is a rare fulminant demyelinating disorder. It is considered to be the most severe form of Acute Disseminated Leucoencephalomyelitis. Mortality occurs in most of the patients in this disease. We present here a case of 28 year male who presented to us with acute onset neurological deficits. MRI brain revealed flair hyper intensities with patchy haemorrhages involving right temporo-occipital lobe and both the cerebellar hemispheres. Our patient is surviving till date after the timely management

KEYWORDS: Acute Disseminated Leucoencephalomyelitis, Hurst's Disease, Respiratory Illness, Acute He morrhagic Leucoencephalitis is a Rare Fulminant Demyelinising Disorder

INTRODUCTION

Acute He morrhagic leucoencephalitis, also called Hurst's Disease, is a rare, rapidly progressive and mostly fatal demyelinating condition affecting the central nervous system. This disease was first described by Weston Hurst in 1941[1]. Since Hurst's first description, <100 adult[2-7] and 10 pediatric[8, 9] cases of AHLE have been reported. It is considered to be a fulminating variant of Acute Disseminated Leucoencephalomyelitis. It is usually preceded by a upper respiratory tract infection[1]

Case Report

A 28 year male presented with moderate grade febrile illness with stuffy nose and mild throat irritation. For this he was treated locally with some medicines and got relieved. 5 days after this episode one morning after getting up from bed, he noticed that he was unable to walk properly and had slurred speech. There was no history of seizures, unconsciousness. Boweland bladder habits were normal.

On examination, he was conscious, oriented with a GCS off 15/15. There was no neck rigidity or kernig's sign. No cranial nerve deficit. However, patient was unable to walk properly and was swaying to either side. Dysdiadochokinesia was present. Shin-heel test was abnormal. Finger-finger nose test, putting dots in a circle were abnormal. All other systemic examination revealed no abnormality.

On investigation, his blood parameters [Complete Blood Count, Liver Function Test, Renal Function Test, Lipid Profile] were normal. Cerebrospinal Fluid examination revealed 15 cells with 85% lymphocytes. CSF sugar and protein were within normal limits. CSF Immune-electrophoresis did not show oligoclonal band. MRI brain revealed patchy areas of FLAIR hyper intensity with associated oedema and patchy haemorrhages involving the right temporo-occipital lobe and both the cerebellar hemispheres. Focal lesions were also seen involving the thalami.

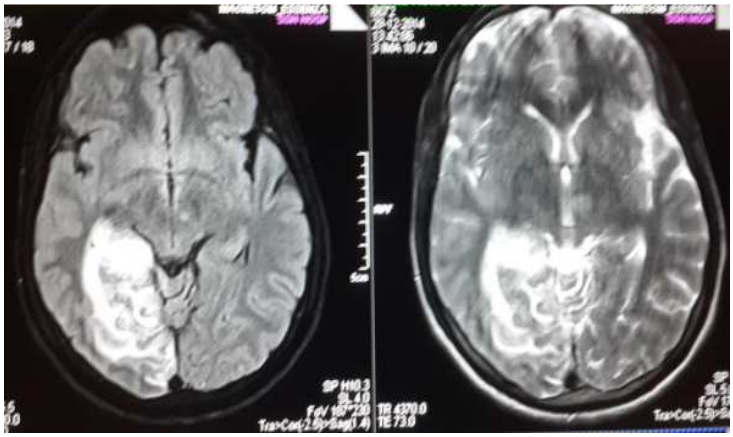

Figure 1

Patient was admitted and treated with high dose steroid therapy. After this patient was symptomatically betterand hence was discharged. 1month later patient came to the hospital with complaints of bitemporal hemianopia. Perimetry confirmed it. Again we started him on steroid therapy. 1month later the patient revisited the hospital with complaints of generalised tonic clonic seizures. Antiepileptic treatment, valproic acid, was administered. Presently the patient is stable and coping with residual neurological deficits. Speech therapy is being advocated for slurred speech.

DISCUSSIONS

Acute Hemorrhagic Leucoencephalopathy is a rapidly progressive, widespread demyelination of the white matter. It is usually preceeded by a viral upper respiratory tract infection. Pathogenesis is thought to be immunologically mediated. Pathological changes include perivascular oedema, vessel wall necrosis, fibrinoid degeneration, infiltrates of eosinophils and neutrophils with perivascular haemorrhage[1]. In the absence of brain biopsy, clinical and radiological evidence is the only source for making a diagnosis. The present case met most of the described criteria in literature. However the prognosis differed as the patient recovered upto a reasonable extent to lead a near normal life except the visual impairment which is difficult to explain as per the available literature.

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