Case Report

Wernicke encephalopathy

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Abstract Wernicke encephalopathy is an uncommon, severe neurological disorder classically characterised by triad of altered state of consciousness, occular dysfunction and ataxia. It is a manifestation of dietary thiamine deficiency (i.e. vitamin B1) either alcohol or non alcohol related.
Keywords: Wernicke encephalopathy.

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Email: vlaxmath579@gmail.com
Received Date: 11/05/2016 Revised Date: 20/06/2016 Accepted Date: 13/07/2016

Access this article online

Quick Response Code:

Website: www.medpulse.in

DOI: 22 July 2016

INTRODUCTION

Wernicke encephalopathy is also known as Wernicke korsak off syndrome. It results from vitamin B1 deficiency. Thiamine represents an essential co enzyme in intermediate carbohydrate metabolism but is also an osmotic gradient regulator[1] Its deficiency may cause swelling of intracellular space and local disruption of bood brain barrier. This disease is a life threatening condition and its prognosis depends on prompt diagnosis, followed by intravenous administration of thiamine[2]**.** If left untreated severe amnesic deficit, korsakoff psychosis and even death may follow the acute phase of the disease.[3]

CASE REPORT

Forty years old male patient was brought by his relatives in a state of unconsciousness. History as narrated by his partner reveals that the patient had binge alcohol intake since three days prior to the onset of altered state of consciousness. He was a chronic alcoholic. Consulting Physician advised imaging study. Hence the patient was subjected to MRI brain plain study with basic sequences [T1W, T2W, FLAIR, DWI]. The MRI revealed the following features.

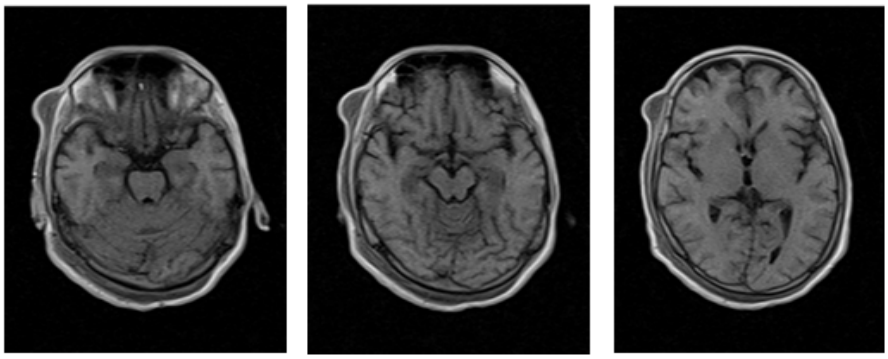

Figure 1: T1W images: There is evidence of hypointensities at bilateral hippocampi,bilateral medial temporal lobes and in parafalcinebilateral frontal lobes,bilateral straight gyri. There isassociated evidence of generalised mild cerebral and cerebellar atrophy. Incidental finding of sebaceous cyst at right fronto-temporal region

How to site this article: A M Deodhar, Valmik Kadpe, Vijayalaxmi Math. Wernicke encephalopathy. *MedPulse – International Medical Journal.* July 2016; 3(7): 672-674. http://www.medpulse.in (accessed 24 July 2016).

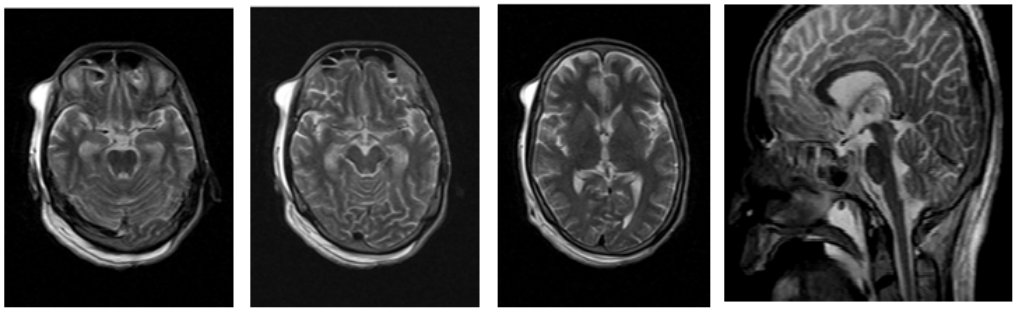

Figure 2: T2W images:-The above corresponding areas show hyperintensities within. There is evidence of hyperintense signal also noted in the splenium of corpus callosum

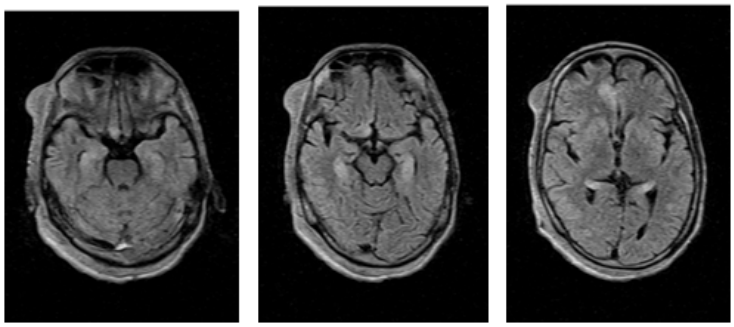

Figure 3: T2FLAIR images: Hyperintensities seen in corresponding areas

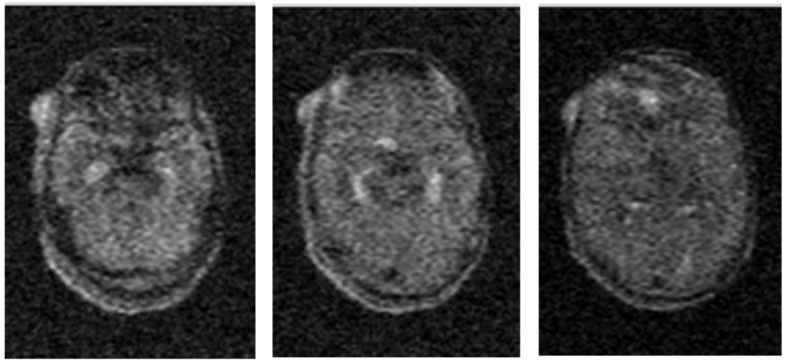

Figure 4: DW Images show restricted diffusion in the corresponding areas. The above features are suggestive of Wernicke encephalopathy

DISCUSSION

Wernicke encephalopathy manifests as a classical triad of ataxia, ophthalmoplegia and altered state of consciousness[4]. Korsakoff syndrome is the term used for late manifestation. Pathologic findings in WE include intracellular edema, demyelination, petechial hemorrhages and astrocyte and microglial proliferation. There is selective vulnerability of medial thalamic nuclei, third ventricular floor, tectal plate, periaqueductal region, mammillary bodies and periventricular region.

Neuroimaging

Imaging especially MRI is playing an increasingly importnant role in the early diagnosis of this condition. It is much more sensitive than computed tomography is now imaging of choice.[5] T1W images show hypointensities around the third ventricles and cerebral aqueduct. In severe cases petechial hemorrhages are seen and they cause hyperintensities in medial thalami and mammillary bodies. T2W and FLAIR images show hyperintensities in affected areas.Bilateral symmetric lesions in the mammillary bodies and around the third ventricle are typical. Less commonly bilateral but asymmetric cortical hyperintensities are present.[6] DWI shows corresponding areas of restricted diffusion[5]. Some areas may show isolated focus of diffusion restriction in corpus callosum splenium. In about half of all cases, post contrast study reveals enhancement of periventricular and periaqueductal lesions. Strong uniform enhancement of mammillary bodies is seen in upto 80% of acute cases and is considered pathognomic. CT:- CT has a low

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sensitivity for the detection of this condition. NECT scans in acute WE are often normal. Subtle findings include bilateral hypodensities around the third ventricle and midbrain. CECT may show subtle enhancement in the affected areas.[7]

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Source of Support: None Declared
Conflict of Interest: None Declared