CASE REPORT

MRI FINDINGS IN ACUTE WERNICKE'S ENCEPHALOPATHY, CAUSED BY HYPEREMESIS GRAVIDARUM

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A 25-year old pregnant female with history of confusion and drowsiness for 02 days was referred by neurophysician for MRI brain. MRI demonstrated T2W/FLAIR hyper intensities in medial thalami, peri-aqueductal areas with variable diffusion restriction, apparent as hyper intense signal on DWI and no signal change on ADC mapping that was typically consistent with Wernicke's encephalopathy. A high index of suspicion is necessary, as delayed or lack of treatment can lead to high morbidity and mortality.

Keywords: Wernicke's encephalopathy, MRI brain, Hyperemesis gravidarum
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INTRODUCTION

Wernicke's encephalopathy (WE) is a neuropsychiatric syndrome, caused by nutritional deficiency of thiamine/vitamin B1. Its deficiency leads to brain lesions in the region of medial thalami, mammillary bodies, periaqueductal region and floor of fourth ventricle.[1,4,5]

We present a case of 25-year pregnant female who presented with acute onset of confusion and drowsiness and was diagnosed as WE based on characteristic MRI findings.

CASE REPORT

A 25-year old female patient with 18-weeks twin pregnancy presented in emergency department of Combined Military Hospital (CMH) Multan in a state of coma with two days history of confusion and drowsiness. Earlier, she had been treated for hyperemesis gravidarum with antiemetics and IV fluids for two weeks and there is also history of weight loss of 18 kg in last 01 month. She was unable to obey the command; her pupils were bilaterally equal and reactive.

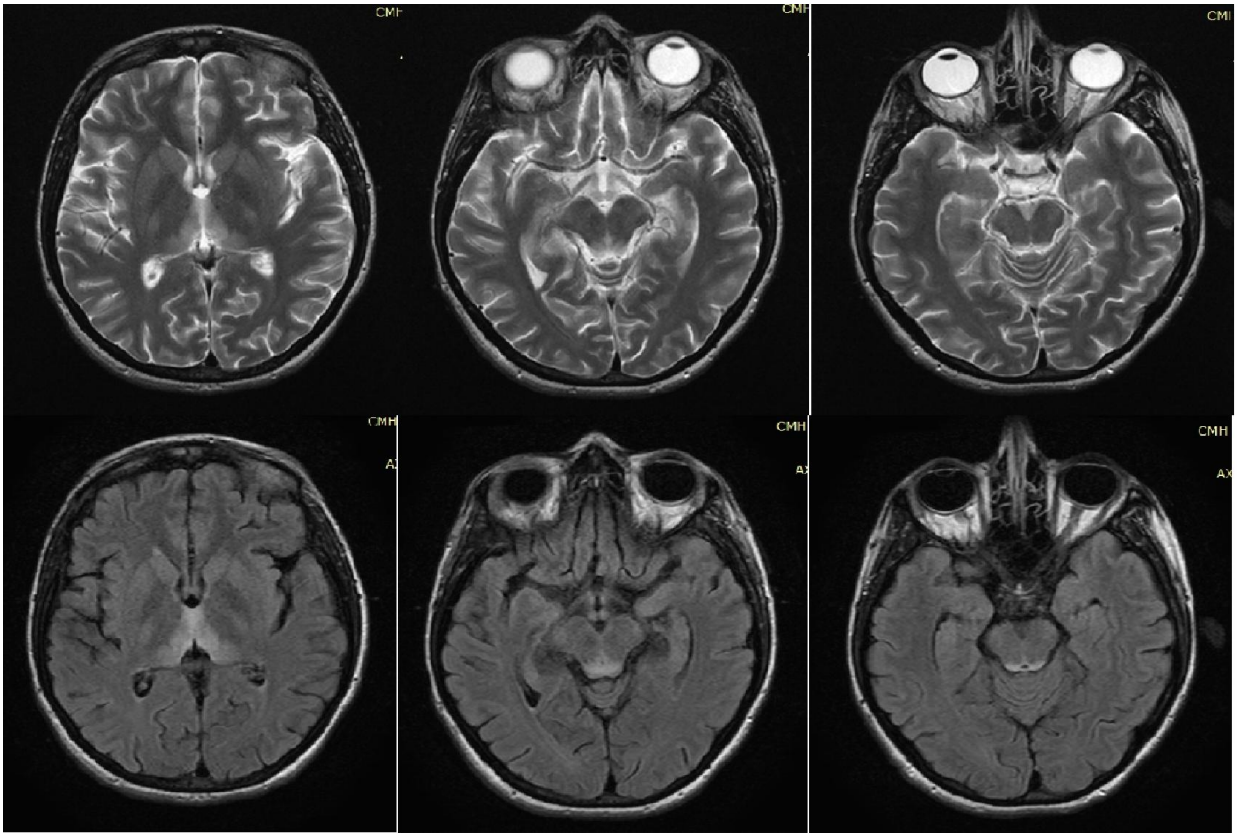

Figure-1: Brain MRI. Axial T2W and FLAIR images showing hyperintense signal in medial thalami and peri-aqueductal region

She had preserved tone in all four limbs and no other neurological deficit was noted. An obstetric ultrasound examination showed intrauterine twin gestation of 18 weeks.

She was referred for MRI brain, which demonstrated hyper-intensities on T2W/FLAIR (Fluid Attenuated Inversion Recovery) sequences in medial thalami and peri-aqueductal areas (Figure-1). These areas demonstrated variable diffusion co-efficient restriction by appearing hyperintense on DWI (Diffusion Weighted Imaging) and no corresponding signal change on ADC (Apparent Diffusion Coefficient) mapping. A diagnosis of WE was suggested and she was started with thiamine replacement and as result the encephalopathy resolved.

DISCUSSION

Wernicke Encephalopathy (WE) is caused by thiamine deficiency. Pyrophosphate is active form of thiamine, which is an essential co-enzyme in the metabolism of glucose in the brain.[1–3] WE is characterized by the classic triad of encephalopathy, ophthalmoplegia or nystagmus and ataxia.[2,4,5] It is important to note that most WE patient do not present the classic triad and thiamine levels can be normal in some patient, whereas MRI brain almost always shows characteristic features which cannot be disregarded. The typical MRI findings include T2W/FLAIR hyperintense signals in the areas around aqueduct, medial thalami, 3rd ventricle and mammillary bodies. The pathologic changes leading to these MRI findings are complex and consist of mechanism that can lead to ischemia like changes in thalami (Cytotoxic/Vasogenic oedema). Hyperintense signals on DWI can be due to restricted water diffusion in the lesion.[3,4] We presented this case in order to highlight the hyperemesis gravidarum as a cause of WE and characteristic MRI features for the diagnosis of a rare cause of acute encephalopathy.

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