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Non-alcoholic Wernicke's Encephalopathy

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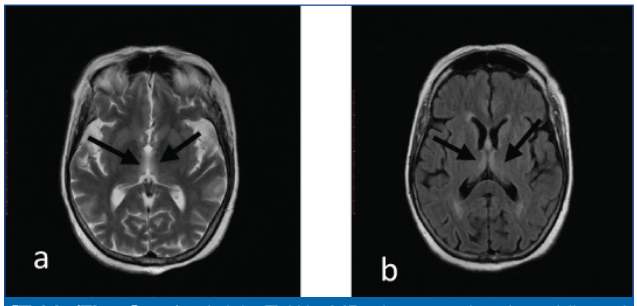
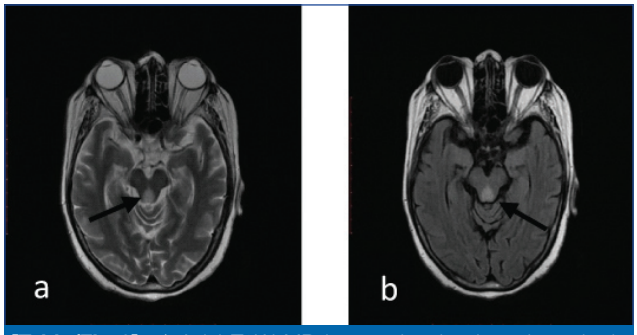
Keywords: Demyelination, Mammillary body, Thiamine deficiency

A 55-year-old female patient presented with history of abnormal eye movements and unsteadiness of gait since 15 days. There was no history of alcohol intake. General condition of the patient was poor with emaciated appearance. Neurological examination showed ataxic gait. Ophthalmological examination revealed bilateral nystagmus.

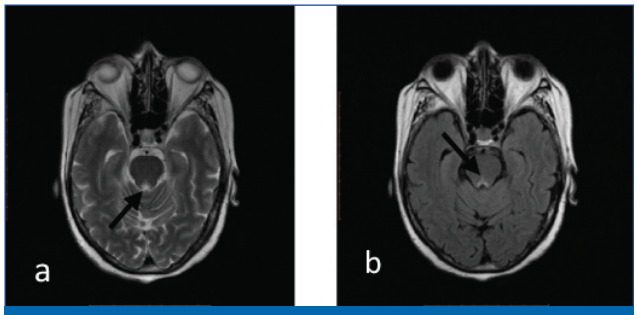
Magnetic resonance imaging of the Brain revealed: Bilateral, symmetric T2W & FLAIR hyperintensity in medial thalami [Table/Fig-1a and b], periaqueductal gray matter [Table/Fig-2a and b], upper dorsal pons [Table/Fig-3a and b] and hypothalamus [Table/Fig-4a and b]. Hyperintense signal noted on DWI [Table/Fig-5a,6a] with no reversal on ADC [Table/Fig-5b,6b]. No blooming on GRE. T1W hypointensity noted in the bilateral medial thalami and periaqueductal gray matter [Table/Fig-7a and b]. Ischemic foci noted in bilateral fronto-parietal lobes.



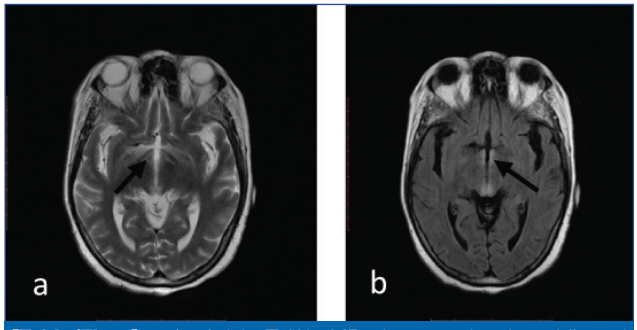
[Table/Fig-1]: a) Axial T2W MR image showing bilateral hyperintensity in medial thalami; b) Axial FLAIR MR image showing bilateral hyperintensity in medial thalami.



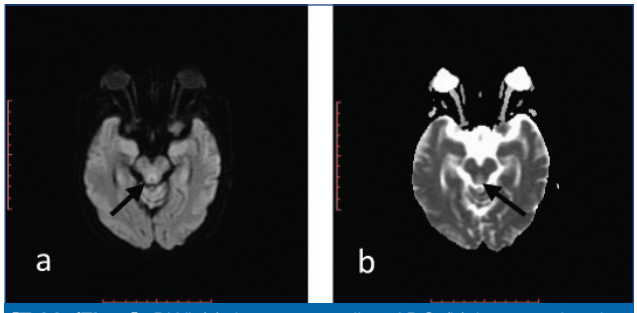
[Table/Fig-2]: a) Axial T2W MR image showing hyperintensity in periaqueductal gray matter; b) Axial FLAIR MR image showing hyperintensity in periaqueductal gray matter.



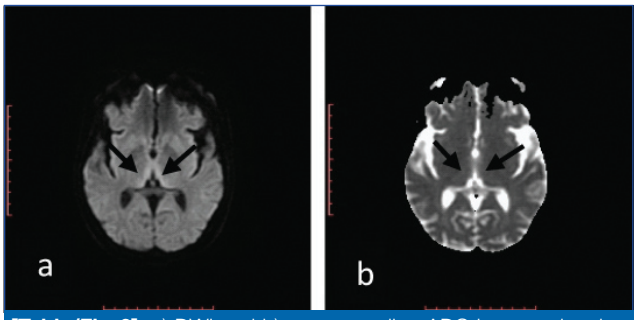
[Table/Fig-3]: a) Axial T2W MR image showing bilateral hyperintensity in upper dorsal pons; b) Axial FLAIR MR image showing bilateral hyperintensity in upper dorsal pons.



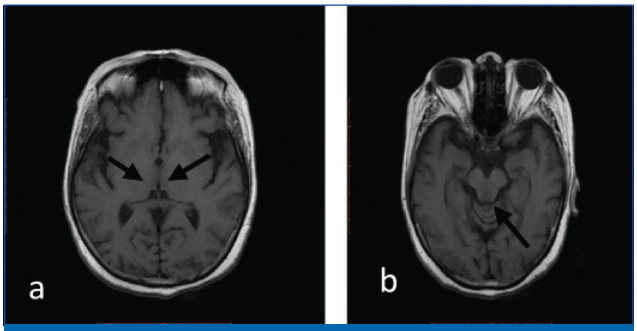
[Table/Fig-4]: a) Axial T2W MR image showing bilateral hyperintensity in hypothalamus; b) Axial FLAIR MR image showing bilateral hyperintensity in hypothalamus.



[Table/Fig-5]: DWI (a) & corresponding ADC (b) images showing hyperintensity in periaqueductal gray matter on DWI with no reversal on ADC.



[Table/Fig-6]: a) DWI and b) corresponding ADC images showing hyperintensity in medial thalami on DWI with no reversal on ADC.



[Table/Fig-7]: a) Axial T1W MR image showing hypointense signal in bilateral medial thalami; b) Axial T1W MR image showing hypointense signal in periaqueductal gray matter.

Laboratory investigations revealed thiamine (vitamin B1) deficiency – 1.6µg/dL (Normal range = 2.5-7.5 µg/dL).

Hence, a final diagnosis of non-alcoholic Wernicke's encephalopathy was made and treatment with rapid intravenous thiamine (vitamin B1) replacement was given.

Wernicke's encephalopathy is a rare neurological disorder caused by thiamine (vitamin B1) deficiency, either in chronic alcoholics or in the non-alcoholic setting. Thiamine (vitamin B1) deficiency in non-alcoholic Wernicke's encephalopathy occurs due to malnutrition secondary to hyperemesis gravidarum, reduced thiamine intake following bariatric surgery, eating disorders, prolonged parenteral nutrition and chemotherapy. Thiamine deficiency leads to disrupted osmotic gradients across cell membranes, lactic acidosis with intracellular and extracellular oedema [1].

Non-alcoholic Wernicke's encephalopathy is more common in adults however, it can occur in children also [2]. In acute stages, demyelination and petechial haemorrhages occur. In chronic Wernicke's encephalopathy, callosal necrosis and white matter rarefaction can be seen. Most commonly involved areas in brain are medial thalami, periaqueductal gray matter, tectum and hypothalamus. Less commonly involved areas are dorsal medulla, cranial nerve nuclei and splenium of corpus callosum [3]. Patients present with a clinical triad of nystagmus, ataxia and confused mental status. Some patients may have polyneuropathy. Korsakoff psychosis is sequelae of Wernicke's encephalopathy which includes severe retrograde amnesia, memory loss and confabulation [3,4].

CT scan has a low sensitivity in the diagnosis of Wernicke's encephalopathy. NECT may be normal in Wernicke's encephalopathy or there may be subtle bilateral hypodensities in midbrain and medial thalami. Contrast enhanced CT scan may show subtle enhancement of the involved areas. On T2W & FLAIR MR images, bilateral symmetric hyperintensity is seen in the medial thalami, periaqueductal gray matter and tectum. Bilateral, asymmetric cortical hyperintensities can be seen in some cases. On T1W MR Image, hypointense signal is seen around the third ventricle and cerebral aqueduct. Hyperintense signal on T1W images in medial thalami may be seen in cases with petechial haemorrhages.

Gradient echo sequences will show microhaemorrhages as areas of blooming in the affected areas [5]. DWI images show restricted diffusion in the areas with cytotoxic oedema. In areas with vasogenic oedema, hyperintense signal on DWI with no reversal on ADC is seen. On T1 post contrast images, enhancement of the periventricular and periaqueductal lesions will be seen [6,7].

Differential diagnosis of Wernicke's encephalopathy includes Artery of Percheron infarct, Deep cerebral venous thrombosis and viral infections like Influenza A and West Nile virus. Alcoholic Wernicke's encephalopathy is characterized by history of alcohol intake, atrophy of mammillary bodies and cerebellar vermis on imaging, which is absent in nonalcoholic Wernicke's encephalopathy [8].

CONCLUSION

MRI is the imaging modality of choice for early diagnosis of nonalcoholic Wernicke's encephalopathy.

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