Wernicke’s Encephalopathy: A case report.

CLINICAL HISTORY: A 28 year old female patient, belonging to low socio-economic status with 25 weeks of pregnancy was referred for MRI of brain. Patient presented with history of vomiting for past 3 months, imbalance and recent onset of confusion.

PLAIN MRI FINDINGS: Symmetrical areas of altered signal intensity were noted in the periaqueductal grey matter, tectal plate, both medial thalami, dorsal pons and in mamillary bodies, showing hyperintense signal on T2WI and FLAIR sequences. Restricted diffusion was seen in the medial thalami.

No evidence of foci of hemorrhage seen.
TOF venogram performed did not reveal any filling defects in the dural sinuses.
FINAL DIAGNOSIS: Acute Wernicke’s Encephalopathy

DISCUSSION: Wernicke’s encephalopathy (WE) is a medical emergency clinically characterised by sudden onset of ataxia, changes in consciousness and abnormal eye movements. However, the classic clinical triad is present in only a minority of patients, making early clinical diagnosis challenging.

Pathogenesis: WE is caused by a deficiency in thiamine (vitamin B1), a water-soluble compound essential for carbohydrate metabolism. However, the disease may not manifest in all patients with thiamine deficiency because genetic susceptibility may be involved in some patients who develop the disorder. Moreover, blood serum levels of thiamine may not be reduced at the time of clinical presentation, limiting the value of a therapeutic test under certain circumstances. Clinical presentation is usually subtle especially in non-alcoholic patients and in those with deep coma whose neurological evaluation is often limited. Non-alcoholic WE manifests in many different clinical settings, such as gastrointestinal tumours, hyperemesis gravidarum, chemotherapy, acquired immunodeficiency syndrome, prolonged therapeutic fasting, prolonged parenteral nutrition and bariatric surgery, anorexia nervosa and can even be secondary to socioeconomic factors.

Imaging features: In WE the blood brain barrier is defective in the periventricular region in which there is a high rate of thiamine related metabolism. Hence MR abnormalities are classically reported in the literature as bilateral and symmetrical lesions around the third ventricle, in the dorsomedial portions of the thalami and the peri-aqueductal region of the mid-brain, characterised by high signal intensity on T2 weighted and FLAIR sequences. Mamillary bodies are frequently involved in WE. Contrast-enhanced images, on the other hand, may disclose breakdown of the blood–brain barrier only in the mamillary bodies and sometimes their involvement is more conspicuous on such enhanced images. Besides the more frequently reported structures involved in WE, there are many other less typically affected locations that can show abnormal signal intensity on MRI. Such structures include the caudate nucleus, perirolandic cortex and posterior putamina. Cranial nerve nuclei may also be involved. Reversible cytotoxic edema is considered the most distinctive lesion of WE, and it is easily shown on MR images.

MESSAGE:
- Wernicke’s encephalopathy is an acute neurological syndrome resulting from thiamine (Vitamin B1) deficiency. Timely thiamine supplementation can reverse the clinical features of the disease.
- Distinct pattern of MR alterations including symmetrical alteration in the thalami, mamillary bodies, tectal plate and peri-aqueductal area.
- Neuroimaging studies are powerful tools in supporting the diagnosis of WE and can also help to distinguish WE from other neurologic disorders.

Regards,

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N.B: This case is authentic and from the archives of Radiance Diagnostics. For any queries/suggestions / feedback write to us at radiance@radiancediagnostics.in. Case of the month can also be accessed anytime online at VIEW BOX at www.radiancediagnostics.in