PICTORIAL CME's

Wernicke’s Encephalopathy

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Fig. 1: Symmetric T2-FLAIR hyperintensities in postero-medial thalamus.

Fig. 2: Symmetric T2-FLAIR hyperintense signal changes in peri-aqueductal areas.

A 35 year old chronic alcoholic presented with progressively increasing erratic behaviour and emotional instability for the preceding 72 hours. On examination, he was afebrile and had normal vitals (BP: 110/86 mm of Hg). Neurological examination revealed disorientation to time and place, indifference and inattention. He also had impaired memory with poor recall. The pupils were unequal and had a sluggish reaction. The extra-ocular muscle movements were normal with fine nystagmus on horizontal gaze. The gait was slow, wide based with short spaced steps. He had no asterixis and plantars were downgoing.

The random capillary glucose was 58 mg/dl and he was given 25 gm. of glucose intravenously and maintained on glucose-saline drip. Baseline investigations including complete blood count and comprehensive metabolic panel were non-contributory. CT scan brain, CSF analysis, serum ammonia, HIV serology and thyroid study didn’t reveal any abnormality.

The patient developed progressive clinical deterioration over the course of next 48 hours in the form of increased drowsiness and stupor. He developed hypotension (BP: 88/64 mm of Hg). Pupils became small and unreactive. Plantars remained flexors bilaterally. MRI brain (T2 weighted FLAIR images) revealed hyperintense signal changes in both the thalamus (Figure 1) and peri-aqueductal gray matter (Figure 2) symmetrically without contrast enhancement.

The history, clinical findings and neuroimaging suggested Wernicke’s encephalopathy (WE). The patient was given thiamine (100 mg IV daily), with marked clinical improvement in the next four days. However, residual memory defects persisted to some extent.

Thiamine (vitamin B-1) deficiency can result in WE, a serious neurological disorder. The components of the classic triad of WE are encephalopathy, ataxic gait, and some variant of oculomotor...
dysfunction. Thiamine is a cofactor for several essential enzymes in the Krebs cycle and the pentose phosphate pathway and thus plays a vital role in carbohydrate metabolism. Because thiamine-dependent enzymes play an essential role in cerebral energy utilisation, thiamine deficiency may cause brain tissue injury by inhibiting metabolism in brain regions with higher metabolic demands and high thiamine turnover. MRI images during the initial days of acute WE show symmetric involvement of the mamillary bodies, the tectal plate, the peri-aqueductal gray matter and the periventricular region of the third ventricle including the paramedian thalamic nuclei. Studies have shown that signal hyperintensities on T2-weighted sequences, FLAIR and DWI within the posteromedial thalami and surrounding the third ventricle is the commonest abnormality and was also present in this patient. It is said that damage to the dorsomedial thalamic region correlates closely with the memory loss and in general the patients have poor recovery from their mental dysfunction. This was also evident in our case.

To conclude, acute WE is often underdiagnosed and it should be considered in all patients with unexplained impairment of cognition of acute or subacute onset. Treatment should be instituted at the earliest possible time to avoid persistent brain damage. MRI is not only a reliable means of diagnosing WE, it can provide additional information and thus is a valuable adjunct to clinical suspicion.

References
