CLINICAL IMAGE

Wernicke encephalopathy: (MRI) picture worth a thousand words

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abstract

A 61-year-old gentleman, with a long-standing history of alcohol abuse presented with altered sensorium and unsteady gait for 2 weeks. A diagnosis of Wernicke encephalopathy (WE) was considered. The magnetic resonance imaging brain showed features consistent with the WE, thus supporting the diagnosis. He was treated with parenteral thiamine with good outcome.

A 61 years old gentleman, who was a known case of alcoholic cirrhosis, was admitted to the hospital with 2 weeks history of confusion that had worsened few days prior to his presentation. He had a long-standing history of alcohol abuse. His family also reported unsteady gait for a month or so. There was no antecedent history of fever, neck stiffness or headache.

On examination his vital signs were within normal limits, however, he was disoriented to time, place and person. His family reported that he has been confabulating. His speech was not dysarthric. Cranial nerves examination revealed horizontal and torsional nystagmus. His gait could not be assessed properly as he tended to sway and potentially fall to either side.

His initial laboratory work up was unremarkable, as was a non-contrast computed tomography of the head. A diagnosis of Wernicke encephalopathy (WE) was made and a magnetic resonance imaging (MRI) of the brain was obtained to rule out other diagnoses such as an ischemic stroke. The MRI brain images are shown in Figs 1–4, as below, that showed features consistent with Wernicke encephalopathy, thus, supporting the diagnosis.

WE is an acute neuropsychiatric syndrome considered to be mainly due to underlying thiamine deficiency and is typically seen in patients with a long-standing history of alcohol abuse. Wernicke triad consists of an acute confusion, ataxia and ophthalmoplegia, however, this triad has low sensitivity and most patients will go undetected if clinicians use the classic triad to diagnose WE. Thus, the newer Caine criteria should be used instead, which is more sensitive and requires two of the following four signs; (i) dietary deficiencies, (ii) oculomotor abnormalities, (iii) cerebellar dysfunction and (iv) either an altered mental state or mild memory impairment [1].

Key diagnostic features on MRI of the brain include areas of symmetrical increased T2/Fluid attenuation inversion recovery (FLAIR) signal that can be seen involving bilateral medial thalami (85%), the mammillary bodies (58%), tectal plate (38%), periaqueductal area (65%) and around the third ventricle [2]. One of the differential diagnoses of these MRI findings is Leigh syndrome, also known as subacute necrotizing encephalomyelopathy, where the mammillary bodies are not involved typically and it presents during childhood. Another differential diagnosis would be Metronidazole induced encephalopathy, where additionally, one can appreciate symmetric signal-intensity alterations of the dentate nuclei, vestibular, abducens, red nuclei and splenium [3].

Prompt parental treatment is paramount and neuroimaging should never delay immediate treatment with intravenous (IV) thiamine as the administration of IV thiamine has little risks involved [4]. Some authorities suggest IV thiamine 500 mg thiamine three times a day for 2–3 days [5]. When left untreated or

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with inappropriately low doses of thiamine, it can be potentially lethal, with mortality rates up to 20% [6].

Although we could not test for ataxic gait in our patient, we were still able to make a diagnosis of WE clinically using the Caine criteria. Thus, even before obtaining MRI of the brain, the patient had received intravenous thiamine followed by oral thiamine therapy. His acute confusion and gait showed partial but significant improvement and he is on long-term follow up with a psychiatrist.
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