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Optic Chiasm Involvement With Concurrent Typical Wernicke Encephalopathy Magnetic Resonance Findings: A Case Report

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The variable clinical presentation of Wernicke encephalopathy often complicates interpretation. Prompt and accurate diagnosis relies on a constellation of *typical* and *atypical* magnetic resonance imaging (MRI) findings, which are not always simultaneously present. Our case demonstrates concurrent presentation of all *typical* Wernicke encephalopathy MRI findings with additional signal abnormalities involving the optic chiasm and optic tract. We suggest that optic pathway involvement may be considered among several *atypical* MRI manifestations, reinforcing the prompt diagnosis of the potentially life-threatening encephalopathy.

Introduction

Wernicke encephalopathy is a rare neurologic condition resulting from the dietary depletion of thiamine.[1] Symptoms are often acute in onset and usually involve a variation of the classic triad: opthalmoplegia, ataxia, and altered consciousness. By far, the populace most susceptible to thiamine deficiency (and subsequent Wernicke encephalopathy) are chronic alcohol abusers.[2] However, there are a number of other etiologies that may also predispose to thiamine deficiency such as chemotherapy, hyperemesis gravidarum, or a history of gastrointestinal surgery.[3,4]

The classic triad symptomatology of Wernicke encephalopathy is often incongruent, leading to delayed or missed diagnosis.[5] This can be clinically unfavorable, as prognosis is contingent upon early administration of intravenous thiamine.[6] Postmortem studies in AIDS patients demonstrated that Wernicke encephalopathy was missed in approximately 75%-80% of cases.[7] For all comers, the estimated mortality rate has been reported as 17% with 80% of cases progressing to Korsakoff syndrome, which is characterized by severe loss of working memory.[8]

Although computed tomography cerebral imaging of patients with Wernicke encephalopathy may transiently demonstrate focal areas of parenchymal hypoattenuation, acute Wernicke encephalopathy does not typically present with abnormal computed tomography imaging findings.[9] Magnetic resonance imaging (MRI) is the most effective modality for correctly diagnosing Wernicke encephalopathy owing to its high specificity, reported at upwards of 93%.[5,10] Brain MRI should be considered in malnourished patients who present with the sudden onset of any isolated triad symptom, especially alterations of consciousness.[6]

Case Report

A 44-year-old man with a past medical history of alcohol abuse and hypertension presented to the emergency department with chest pressure, dizziness, and left arm tingling which began that morning. The patient was admitted but later discharged home when further diagnostic tests and labs were unremarkable. One month later, the patient returned to the emergency department with intermittent delirium, recurrent dizziness, and new onset diplopia. Physical examination revealed unilateral medial strabismus of the left eye with continual horizontal nystagmus and bilateral upper extremity dysdiadochokinesia. Although the patient was alert and oriented, he demonstrated an unusual flattened affect. Differential consideration was given for an acute cerebral process, and an MRI of the brain was ordered after computed tomography imaging showed no territorial infarct or hemorrhage.

Multisequence MRI of the brain was performed without and with intravenous paramagnetic contrast. Axial T2 and FLAIR imaging demonstrated abnormal signal hyperintensity involving the bilateral medial thalami and surrounding the third ventricle (Fig 1A and B), whereas postcontrast images showed symmetric enhancement of the mammillary bodies (Fig 1C and D). There is hyperintensity seen within the periaqueductal gray matter (Fig 2A), and minimal contrast enhancement of the midbrain tectal plate (Fig 2B). Additionally, postgadolinium imaging demonstrated enhancement of the optic chiasm (Fig 3A) with a slight left predilection, with mild enhancement of the left optic tract (Fig 3B).

A diagnosis of Wernicke encephalopathy was determined, and intravenous thiamine was promptly initiated. Within 12 hours of beginning thiamine therapy, there was improvement in the

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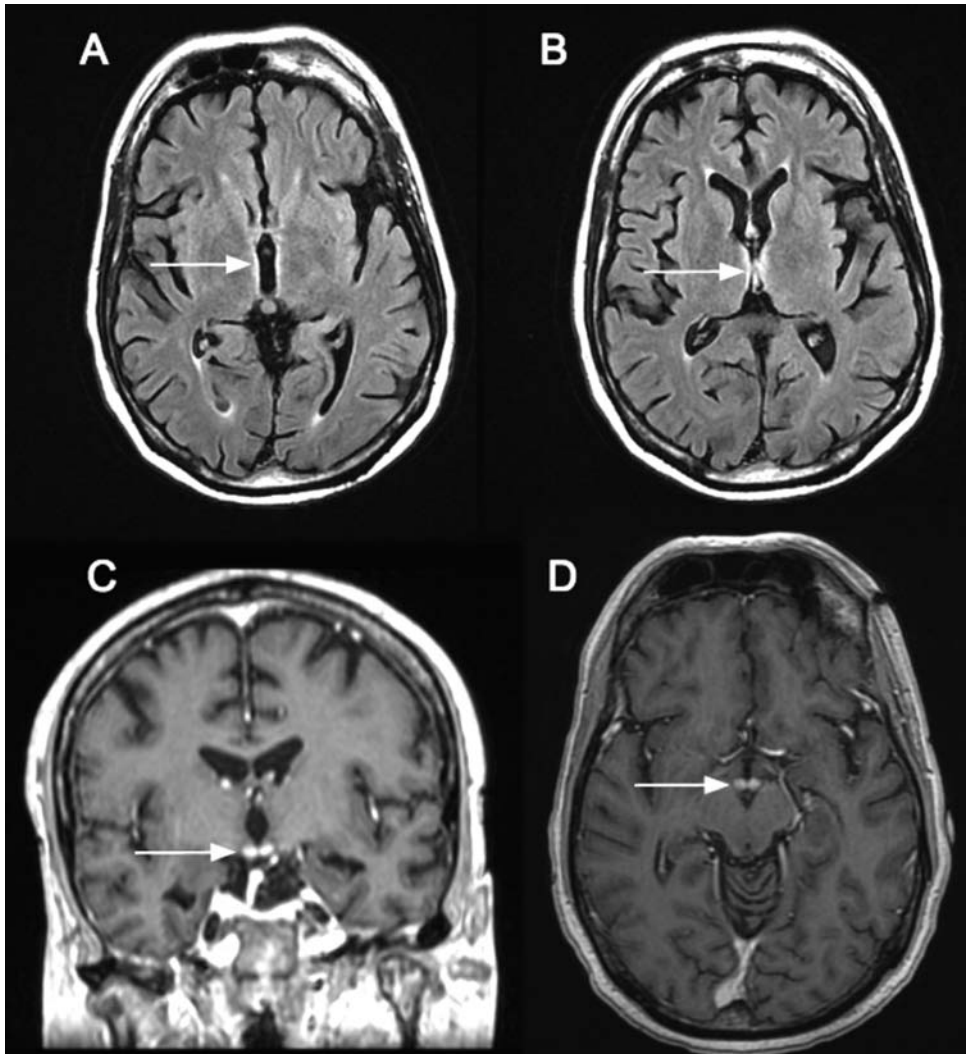

FIG 1. Axial T2-weighted FLAIR images at the level of the thalamus, demonstrating hyperintensity of the perithird ventricle (A) and medial thalami (B). Postgadolinium coronal and axial images demonstrating symmetric enhancement of the mammillary bodies (C and D). Mammillary body enhancement is one of the more notable findings in Wernicke encephalopathy, and on occasion, may be only image finding manifest in the disease.

patient's ophthalmic symptoms and ataxia. Over the next 24 hours, his symptoms continued to subside, and the patient was discharged home with a detailed plan for alcohol cessation.

Discussion

A number of distinct structures within the central nervous system maintain a cellular osmotic gradient with particular sensitivity to thiamine levels.[11] When thiamine levels are depleted, as is the case in Wernicke encephalopathy, these structures often exhibit abnormal MRI signal intensity,[12] with the most well-documented structures being the mammillary bodies, medial thalami, midbrain tectal plate, and periaqueductal area.[9] Although these MRI findings are called *typical*, they are not always present in Wernicke encephalopathy. For example, some case studies demonstrate the abnormal MRI manifestation of the medial thalami at 80%-85%, periaqueductal area at 59%-65%, mamillary bodies at 38%-45%, and tectal plate at 35%-38%.[9,13] Another study claimed that typical pattern of MRI findings is only present in 58% of cases.[14] To the best of our knowledge, there are no published cases or statistics addressing the simultaneous presentation of all *typical* Wernicke encephalopathy findings, which are demonstrated in this case report.

Various additional MRI findings have been found to coexist with these 4 typical findings of Wernicke encephalopathy. These have been termed *atypical* MRI findings and range in location from the myelencephalon to the telencephalon, but most often are associated with typical findings. Additional atypical findings within the brainstem include the dorsal medulla, pons, red nucleus, substantia nigra, as well as the nuclei of several cranial nerves.[15] Associated structures in the cerebellum are the dentate nuclei, vermis, and paravermian regions.[9] In the telencephalon, the corpus callosum, fornix, and parieto-frontal cortex are also atypical locations associated with Wernicke encephalopathy.[15]

Throughout the brain, the rate of oxidative metabolism varies significantly.[16] Typical sites involved in Wernicke encephalopathy possess a particularly high rate of oxidative metabolism.[17] The demonstrated variability of expressed atypical findings leads us to question what additional central nervous system regions are susceptible to thiamine deficiency, and may potentially manifest on MRI.

Clinical manifestations do not always correlate with MRI findings. For example, the periaqueductal grey matter is known for its central role in descending pain modulation yet, in acute Wernicke