

Images in Neurology

Characteristic Neuroimaging Abnormalities of Korsakoff Syndrome

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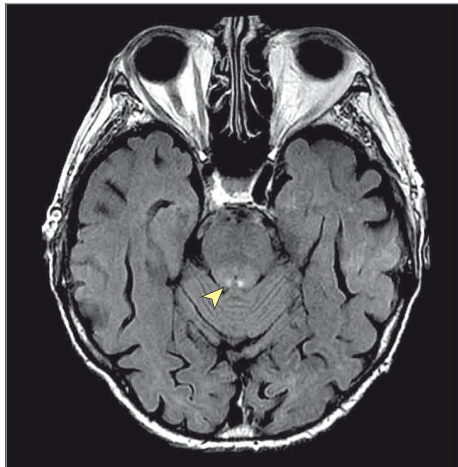
A man in his 70s with a history of chronically elevated lactate levels, alcoholic cirrhosis, and chronic cognitive decline attributed to hepatic encephalopathy despite lack of asterixis or response to lactulose presented with 1 week of confusion worsened from baseline. On examination, he had anterograde worse than retrograde memory deficits without asterixis, nystagmus, ophthalmoplegia, or ataxia. Findings on magnetic resonance imaging of the brain (Figure) were consistent with previously undiagnosed Korsakoff syndrome.¹ His subacute decline was attributed to a urinary tract infection, and the patient's orientation returned to baseline with antibiotics. His lac-

tate level returned to within a normal range with parenteral thiamine, although his memory deficits persisted.

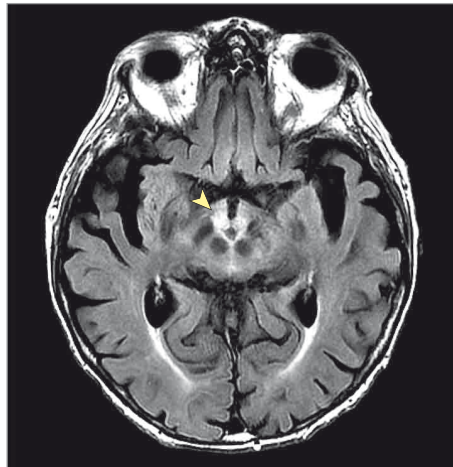
Korsakoff syndrome is an acquired chronic disease characterized by dense anterograde amnesia and short-term memory loss in the setting of otherwise intact cognition.¹ Korsakoff syndrome results from untreated or undertreated Wernicke encephalopathy, an acute, reversible condition caused by thiamine deficiency. Thiamine mediates pyruvate metabolism; its deficiency can result in elevated lactate.² Wernicke encephalopathy is most closely associ-

Figure. Magnetic Resonance Imaging Findings of Korsakoff Syndrome

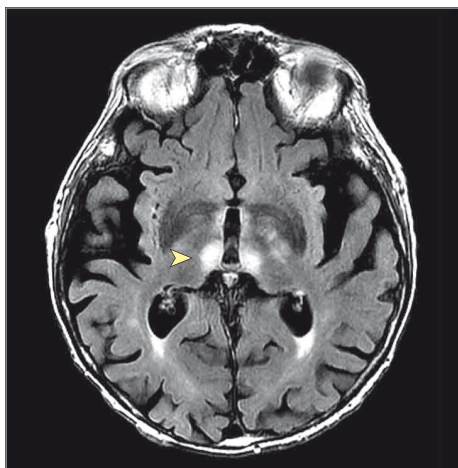
A Periaqueductal gray matter



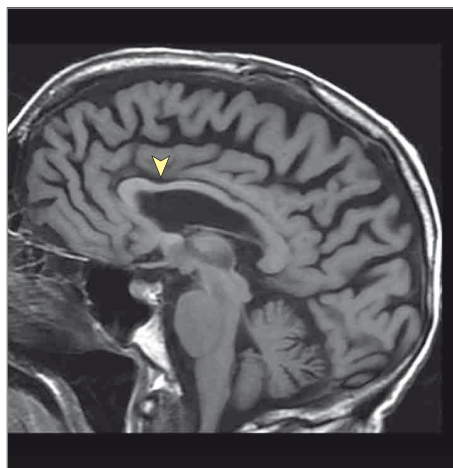
B Mammillary bodies



C Medial thalami



D Sagittal T1-weighted image



Magnetic resonance imaging shows bilateral fluid-attenuated inversion recovery hyperintensity (arrowheads) in the periaqueductal gray matter (A), mammillary bodies (B), and medial thalami (C). D, Sagittal T1-weighted image demonstrates volume loss in the corpus callosum and in the cortex with widening of the cerebral sulci (arrowhead).

ated with chronic alcohol abuse but can also arise from nonalcoholic causes of nutritional deficiency such as gastrointestinal surgery.¹

Only 16.5% of patients with Wernicke encephalopathy present with the classic triad of ataxia, ophthalmoplegia, and confusion.³ Diagnosis is also frequently confounded by concurrent explanations for a patient's cognitive or motor symptoms such as hepatic encephalopathy or intoxication. As a result, the diagnosis of Wernicke encephalopathy is often missed.³

Magnetic resonance imaging is the most valuable study to detect symptomatic thiamine deficiency.¹ Fluid-attenuated inversion recovery sequences demonstrate characteristic symmetric hyperintensities most frequently in the medial thalamus, periventricular region, periaqueductal gray matter, mammillary bodies, and mid-

brain tectum most likely reflecting edema, loss of neurons, and reactive gliosis.¹ These lesions can be accompanied by symmetrically distributed hyperintensities in atypical locations.⁴ When the disease is chronic, T1-weighted imaging can show volume loss in the mammillary bodies and corpus callosum along with enlargement of the sulci and ventricles suggesting atrophy and volume loss.⁴

Once a patient develops Korsakoff syndrome, the prognosis for recovery is poor. Dense amnesia and short-term memory loss are typically irreversible.¹ Disability from Korsakoff syndrome is best prevented by retaining a high index of suspicion when evaluating malnourished patients at risk for thiamine deficiency and immediately administering parenteral thiamine and glucose if Wernicke encephalopathy is suspected.^{1,3}

ARTICLE INFORMATION

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