

Background

- Anorexia nervosa can result in complications across many organ systems with malnutrition and weight loss acting as the key etiologic factor.
- Poor dietary intake can lead to deficiencies of thiamine and the classical triad of encephalopathy, oculomotor dysfunction, and gait ataxia.
- Presented here is a case of a young patient suffering from anorexia nervosa who developed thiamine deficiency and a literature review regarding non-alcoholic Wernicke encephalopathy.

Case

- A 15-year-old female with no medical history presented with a new diagnosis of anorexia nervosa, restricting type, at a BMI of 12.38.
- The patient was admitted for nutritional rehabilitation and refeeding syndrome monitoring.
- On hospital day 6, the patient was noted to have increased confusion.
- Overnight EEG was within normal limits.
- The next day, the patient was experiencing worsening agitation and hallucinations with nystagmus noted on exam.
- No gait disturbances were observed. MRI revealed mild diffuse parenchymal loss consistent with anorexia nervosa and mild cerebral hypoperfusion.
- The patient was managed with two days of IV thiamine 1500mg followed by five days of 250mg IV thiamine then 100mg oral thiamine daily.
- Mentation returned to baseline by time of discharge and the patient was continued on thiamine upon discharge.

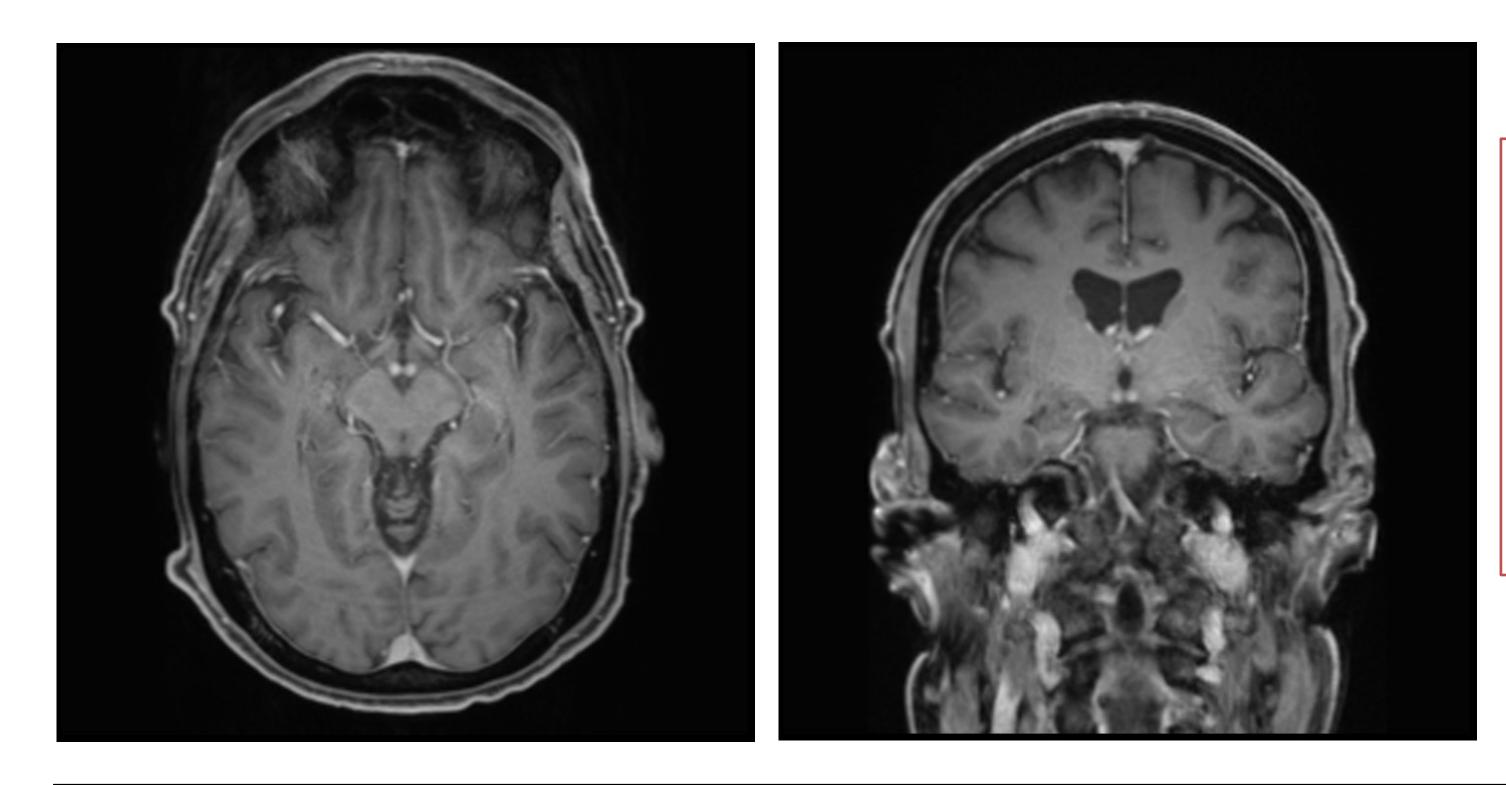
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Images

Image 1: Case courtesy of Dr Zach Drew, Radiopaedia.org, rID: 71702 Image 2: Case courtesy of Dr Zach Drew, Radiopaedia.org, rID: 71702

Not always alcohol: A case of restriction-induced Wernicke Encephalopathy Samantha DeSilva, BS, and Meghan Schott, DO, FAPA



Discussion

- In one autopsy study, non-alcoholics counted for nearly one-quarter of Wernicke encephalopathy.² A retrospective observational cohort study found non-alcoholics took longer to diagnose over alcoholic patients (4 vs 1 days) and required longer hospitalizations (23 vs 13 days).³
- The extent and severity of neuropsychiatric symptoms of Wernicke encephalopathy often more severe in AN than alcohol-induced.
- Cohort studies and systematic reviews show ocular signs were more common and cerebellar signs were less common in non-alcoholic patients.^{3,4}
- Fittingly, another retrospective autopsy study found that the classic triad was only recorded in 17% of patients.⁵
- Neurologic symptoms often seen outside the triad include diplopia, unresponsive pupils, general paresis Mood symptoms often seen in thiamine deficiency include depressive symptoms, emotional liability, cognitive difficulty, and loss of appetite, complicating the diagnosis of anorexia nervosa.
- In this case, nystagmus and altered mental status were present and cerebellar gait ataxia was not observed.
- These key differences all contribute to the need for more attention to subtle clinical characteristics of

Conclusion

Wernicke encephalopathy can present subtly, oftentimes not meeting all facets of the classical triad and with symptoms that overlap with mood disorders. The index of suspicion for this dangerous syndrome should remain high when assessing non-alcoholic patients with other causes of poor nutrition, including eating disorders.

Image 1. MRI, Axial T1 C+. Mammillary bodies are a region of high oxidative metabolism, and thus, enhancement can be seen in thiamine deficiency.

Image 2. MRI, Coronal T1 C+ . Mammillary body enhancement seen in thiamine deficiency.

Wernicke encephalopathy rather than seeking the classic triad and supplementing thiamine as indicated.