Nutritional Deficiencies Causing Altered Mental Status: Complicated Case of Non-Alcoholic Wernicke's Encephalopathy in a 55-year-old Woman with Persistent GI Distress and Rapid Weight Loss

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Abstract

Objective: This case report underscores the importance of recognizing and promptly treating Wernicke's encephalopathy in the setting of conditions leading to nutritional deficiencies.

Context: Wernicke's encephalopathy (WE), is a well-known complication of thiamine deficiency. It may be overlooked in 94% of patient's without alcoholism and 68% of those with alcoholism. It is crucial that this diagnosis not be missed as the consequences are severe and permanent.

Case Summary: 55 yo female with a history of prolonged nausea, vomiting, and weight loss presented with confusion and ataxia. Treatment for WE with IV thiamine lead to improvement, but patient continued to have long term effects.

Comparison to literature: Vomiting is a very common cause and symptom of WE. Diagnosis should be made according to the Caine criteria. Prompt treatment with IV thiamine is crucial

Introduction

- WE is a condition due to thiamine deficiency, traditionally associated with alcohol use.
- Triad: mental status changes, ophthalmoplegia and gait ataxia but seen in a minority of cases.
- Caine criteria highly sensitive and specific for WE; 2 of 4 signs including classic triad and dietary deficiencies[1].
- WE most often diagnosed at autopsy; >80% of true cases not diagnosed during life[2].
- In thiamine deficiency, toxic intermediates in the Krebs cycle, glycolysis, and prevents synthesis of nucleic acids through PPP[4] (Figure 1).

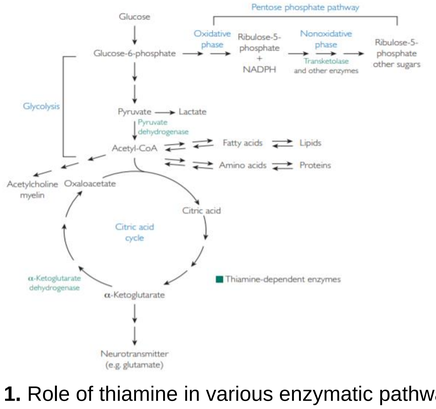

Figure 1. Role of thiamine in various enzymatic pathways

Case Description

- 55 yo female with history of NIDDM and bipolar disorder
- Recurrent ED visits and hospital admissions over the previous 2-3 months for nausea, vomiting, and abdominal pain. 45 lb weight loss in the last 3 months.
- Presented on this visit with nausea, vomiting, and generalized weakness/fatigue and was found to have electrolyte abnormalities (Table 1).
- GI symptoms were assumed to be due to diabetic gastroparesis, though never confirmed with a gastric emptying study, so treated with Reglan and IVF.
- Initially psychiatric medications including Abilify, Cogentin, and Trazodone were restarted while Zoloft and Klonopin were held.
- Patient became confused and Psychiatry was consulted.
- Unable to follow instructions. Had muscle weakness without clonus or rigidity, stuttering speech, and hallucinations.
- Although not showing many of the symptoms, there was some concern for NMS, benzodiazepine withdrawal, and less likely serotonin syndrome, so psychiatric medications were held except for resumption of Klonopin.
- With minimal improvement, Neurology was consulted.
- Exam was limited due to mental status, but demonstrated ataxia.
- Suspicious for WE given the electrolyte abnormalities in the context of weight loss and likely malnutrition.
- IV Thiamine was started after drawing lab (Table 1).
- MRI obtained showed subtle FLAIR hyperintensities of the dorsomedial thalamus, periaqueductal gray and tectal plate consistent with Wernicke's encephalopathy. (Figure 2).
- Ataxia and sensorium improved. With improved ability to follow instructions, ophthalmoplegia was observed.
- On discharge patient was given oral Thiamine supplements.
- Post-hospitalization, patient continued to decline with progressive nausea, vomiting, ataxia, bilateral leg weakness, sleep-wake disorder, AMS, and hallucinations.

Results

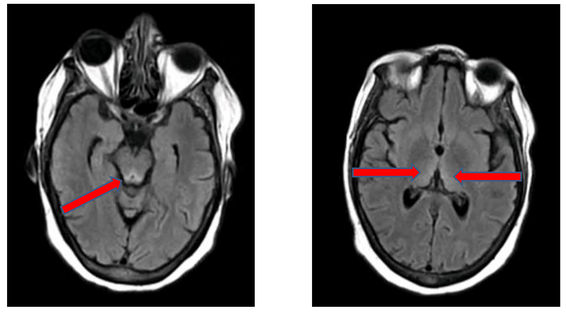

Figure 2. T2 FLAIR hyperintensity in the periaqueductal area (left) and bilateral medial thalamic area (right)

Results

Lab Studies	Result
K	2.2
Na	150
Ca, corrected	8.6
Ammonia	<10
Albumin	2.7
AST	149
ALT	184
CK	1,470
Thiamine	20.3

Table 1. Labs showing electrolyte abnormalities and nutritional deficiencies

Discussion

- One of the largest reported series of clinically diagnosed non-alcoholic WE analyzes 50 cases of non-alcoholic WE in Northern India where polished rice, which is thiamine deficient, is a dietary staple.
- Clinical diagnosis was made according Caine criteria - 2 or more of the following: (i) dietary deficiencies, (ii) eye signs, (iii) cerebellar dysfunction, and (iv) either AMS or mild memory impairment.
- Preceding/recurrent vomiting present in 42 patients with avg. duration of vomiting of 27.2 days (range 2-90 days)
- Vomiting is both a cause and a symptom of WE. It was the most common symptom reported (Table 2).

Symptom/sign	Number of patients (%)
Altered mental status	30 (60)
Memory impairment	15 (30)
Ophthalmoparesis	11 (22)
Seizures	4 (8)
Ataxia	18 (36)
Triad (confusion, ophthalmoplegia, ataxia)	5 (10)
Nystagmus	35 (70)
Anorexia	9 (18)
Abdominal pain	9 (18)
Nausea and vomiting	45 (90)
Fatigue	8 (16)
Weakness of lower limbs	35 (70)
Pedal edema	7 (14)
Tachycardia	6 (12)
Congestive heart failure	2 (4)

Table 2. Clinical features of nonalcoholic patients with WE

- <15% of patients had a major disease known to be associated with WE.
- GI disorders constituted the largest identifiable associated disorder in this study (Table 3)[3].

Clinical condition	Number of patients (%)
Acute gastritis	6 (12)
Acute calcular cholecystitis	2 (4)
Cholelithiasis	4 (8)
Duodenal ulcer	2 (4)
Chronic diarrhea	1 (2)
Pregnancy/peurperium	3 (6)
Hyperemesis gravidarum	1 (2)
Chronic liver disease	1 (2)
Recent major surgery	2 (4)

Table 3. Clinical conditions in the patients with possible association with development of WE

Discussion

- Development of thiamine deficiency occurs via one of four mechanisms: decreased availability, accelerated usage, impaired utilization, and increased loss[2].
- Of the 20 patients who underwent brain MRI, 16 had abnormal findings, with the most common lesions being bilateral medial thalamic, periaqueductal, and tectal hyperintensities.[3]
- Although there are no biomarkers that can confirm diagnosis or quantify disease, MRI has 53% sensitivity and 93% specificity with most common lesions found in dorsomedial thalamus, mammillary bodies, periaqueductal area, and tectal plate[1].
- All patients were treated with IV thiamine for 5-10 days followed by oral maintenance.
- Oral thiamine should not be used in the treatment of hospitalized patients with suspected WE due to absorption in the small intestine in a rate-limited, saturable process; parenteral thiamine is transported across blood brain barrier allowing for quick delivery of high doses of thiamine.
- 49 patients showed some degree of improvement though 1 patient developed Korsakoff psychosis.
- 9 patients had residual symptoms, the most common being lower limb weakness and ataxia.

Conclusions

- Several medical conditions predispose individuals to developing WE, particularly GI disorders.
- If WE is suspected, *must* treat promptly with **IV thiamine** as WE is a largely reversible condition and benefits of treatment outweigh risks.
- WE is a clinical diagnosis.
- Plasma thiamine levels do not correlate with disease, but MRI can help support diagnosis.
- Without prompt treatment, will progress to permanent brain damage and irreversible Korsakoff syndrome.
- Structure and function are reciprocally interrelated; in the setting of WE with alteration in brain structure leads to poor functioning.

References

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