Hallucinations as a presenting symptom of non-alcoholic Wernicke-Korsakoff syndrome



BACKGROUND

Wernicke-Korsakoff syndrome (WKS) is a neuropsychiatric syndrome caused by thiamine deficiency that consists of two overlapping disorders, Wernicke encephalopathy (WE) and Korsakoff's syndrome (KS)

Symptoms of Wernicke encephalopathy include oculomotor abnormalities, cerebellar dysfunction, alterations in mental state, and mild memory impairments

Korsakoff's syndrome is a residual syndrome resulting from untreated WE, is characterized by global amnesia

| Causes | Mechanisms of thiamine deficiency |
|-----------------------------|---|
| Alcohol use | Low uptake of thiamine Low mucosal thiamine absorption Impaired thiamine utilization |
| GI procedure/starvation | Low uptake of thiamine |
| Hyperemesis gravidarum | Low uptake of thiamine Increased demands of pregnancy Loss of thiamine |
| Chemotherapy | Low uptake of thiamine Decreased thiamine availability Inactivation of thiamine Cachexia |
| Hyperthyroidism | Raised thiamine metabolism |
| Infectious and inflammatory | Low uptake of thiamine Raised thiamine metabolism Inhibition of intestinal uptake |
| Genetic diseases | Inactivation of thiamine transporter |

Though it is classically associated with alcohol use, non-alcoholic causes are possible, increasingly recognized, and have increased likelihood in medically-hospitalized populations

One barrier to effective recognition is that the symptomatology and findings of non-alcoholic WKS appears to be distinct from alcoholic and poorly understood and characterized

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CASE CC: 38 y/o female with no known past psychiatric history Admitted for a 2-week history of hallucinations HPI/Exam: Isolated tactile hallucinations of rats biting her legs with no hallucinations in other sensory modalities • Generally poor historian, unfocused, and inconsistent Anterograde amnesia and executive dysfunction present Otherwise normal exam with limited evidence of attention deficits, mania, disorganized thinking suggestive of delirium or a primary psychotic disorder **Past Medical History:** • History of epilepsy and hypertension Had an admission 5 months prior for LE weakness and confusion that had been preceded by several months of nausea, vomiting, decreased oral intake, and 35-pound unintentional weight loss

- Previous work-up showed thiamine and folate deficiencies, which were repleted
- Rest of work-up normal, including head imaging, EEG, and blood chemistries

Hospital Course:

- Thiamine was again found to be deficient
- Similar to the prior admission, the remainder of work-up was negative
- Started on high-dose intravenous thiamine supplementation and gabapentin for suspected neuropathic pain in her legs
- Discharged several days later after resolution of tactile hallucinations



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DISCUSSION

While the symptomatology of alcoholic WKS has been welldocumented, the pattern of symptoms and neuroimaging findings in non-alcoholic WKS has been more diverse. To our knowledge, there have been no descriptions of hallucinations in patients with nonalcoholic WKS. In this case, the nature of the patient's hallucinations remained unclear, though their improvement with the use of gabapentin suggests that tactile hallucinations was the misinterpretation of peripheral neuropathy, a known symptom of thiamine deficiency (Hamel, 2018).

CONCLUSIONS

This case adds to our understanding of non-alcoholic WKS's clinical phenotype and serves to alert consultation-liaison psychiatrists to consider this diagnosis in patients with new-onset hallucinations following significant weight loss and nutritional deficiencies.

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