CLINICAL VIGNETTE

Dry Beriberi and Wernicke's Encephalopathy Presenting as Dysphagia

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Case Description

A 54-year-old man was admitted from the Emergency Department with worsening dysphagia. He stated his mouth felt very dry, like he had no saliva and food would become stuck in his throat, which would "close up" and stick "like glue" when he ate or drank anything. According to his family, his symptoms began about 3 weeks ago, after he received his first dose of the Moderna COVID-19 vaccine. He expressed fear about what was injected into his body, subsequently reported difficulty swallowing both solids and liquids, and eventually stopped eating all-together due to dysphagia, with significant weight loss.

His past medical history is notable for dementia, anxiety, panic disorder, post-traumatic stress disorder, gastritis, and multinodular thyroid. Paranoia began at age 48 associated with jaundice, confusion, and auditory/visual hallucinations. He believed he was being poisoned and stopped eating and drinking to the point of becoming emaciated. Prior to this, he was a high functioning individual and completed a post-graduate degree. He has been living at a rehabilitation center for the past 3 years after a hospitalization for oropharyngeal dysphagia and acute encephalopathy. He was diagnosed with non-alcoholic Korsakoff psychosis with grave debilitation. Per his brother, he slowly recovered to the point of speaking fluently, reading, writing, walking, and was independent in his activities of daily living until this recent episode. He has never smoked, does not drink alcohol, nor uses any illicit drugs. Daily medications include olanzapine 7.5 mg, thiamine 100 mg, cholecalciferol 1000 units, mirtazapine 15 mg nightly, and a multivitamin. However, he stopped taking all of his medications three weeks prior when he developed dysphagia.

Upon admission, he reported worsening dysphagia over the past 10 days as well as some nausea and weakness. He denied odynophagia, abdominal pain, and vomiting. He also reported recent numbness and tingling of his lower extremities, which had resolved. His labs were notable for hypernatremia, blood urea nitrogen to creatinine ratio greater than 20:1, and high urine specific gravity, as well as elevated unconjugated bilirubin, low total protein, and vitamin D deficiency, consistent with significant dehydration and malnutrition. Thyroid stimulating hormone, vitamin B12, and folate levels were normal and head CT showed no acute intracranial abnormality. On physical exam, he was jaundiced and with temporal wasting and BMI 17.9. Abdomen was non-tender with negative Murphy's sign. He had dysphonia, soft speech, alogia, and increased speech latency, though was alert and oriented to person, place, time, and situation, with intact comprehension and recall. His MoCA score was 25, indicating mild cognitive impairment without documentation of baseline with history of prior neurologic insult resulting in dementia. He had horizontal nystagmus, but otherwise intact cranial nerves. Neuro exam revealed he had 4/5 strength in his upper and lower extremities, intact sensation to light touch, positive Tinel's sign of the left wrist, and intact reflexes. Gait was slow and unsteady.

He initially refused to participate in a swallow evaluation due to fear of food becoming stuck in his throat, but agreed to participate if he was allowed to self-administer the liquids and solids. He demonstrated an intact swallow and consumed an entire cup of gelatin and water without any difficulties. The following day, he agreed to restart oral medications and gradually increased his consumption of food and beverages. Gastroenterology, Neurology, and Psychiatry were consulted. However, his surrogate decision maker declined to pursue further studies and imaging including barium esophagram, endoscopy, a brain MRI, and electroencephalogram due to concern for worsening his paranoia. Occupational and physical therapy consulted for weakness and unsteady gait. Thiamine level resulted as less than seven nmol/L and raised concern for dry beriberi. He was switched from oral to intravenous (IV) 500mg TID for 3 days thiamine due to possible Wernicke's encephalopathy given the horizontal nystagmus and unsteady gait. He continued to express paranoia about being poisoned, especially with IV medications and refused a few doses of IV thiamine and only completed two days of treatment. His strength, energy, and food consumption improved after initiation of IV thiamine. He demonstrated no confabulation to suggest Korsakoff's syndrome. He was discharged back to his prior rehab center for completion of Wernicke's treatment with thiamine 250 mg IV daily for an additional five days followed by daily oral thiamine.

Discussion

Severe thiamine deficiency may cause preventable, life-threatening conditions such as dry beriberi, Wernicke's encephalopathy (WE), and Korsakoff syndrome (KS). These

conditions have historically been associated with alcoholism, however, they can result from any cause leading to an inadequate supply of thiamine including starvation, malnutrition, bariatric surgery, hyperemesis gravidarum and anorexia.[1] The body stores minimal thiamine, so severe depletion may occur within 18 days on a thiamine deficient diet.[2] Similar to this case, there are documented cases of Wernicke's and Korsakoff syndrome in nonalcoholic patients with psychiatric disorders.[3] Our case illustrates dry beriberi and possible non-alcoholic Wernicke's encephalopathy arising in the setting of neuropsychiatric illness.

Dry beriberi may present as peripheral neuropathy with paresthesia, patchy sensory loss, weakness, and gait difficulty.[4] Areflexia, foot drop, and wrist drop may also occur.[5] It can be associated with WE and KS depending on the degree and duration of thiamine deficiency. About 80% of cases of WE are made at autopsy.[6] The poor rate of early diagnosis may be due to the wide range of nonspecific symptoms of severe thiamine deficiency. These include fatigue, irritability, poor memory, sleep disturbances, anorexia, abdominal discomfort, and constipation.[7] Furthermore, the classic WE triad of confusion, ocular abnormalities, and ataxia may not be present in up to 90% of patients.[8] This patient initially presented with progressive dysphagia with globus sensation with a broad differential, often focusing on structural versus motility issues. On further examination, he had multiple features concerning for thiamine deficiency, including dysphonia, subtle nystagmus, symmetric motor weakness with unsteady gait, and reported lower leg numbness and tingling. His underlying psychiatric illness was apparent through paranoia during his initial swallow evaluation. After establishing patient-provider trust, he had an intact swallow with solids and liquids, lowering the suspicion for true dysphagia. His improvement in eating and drinking throughout his hospitalization further supported psychiatric dysphagia. His underlying psychiatric paranoia of being poisoned, with self-starvation resulting in dry beriberi with possible WE. The focus of his care shifted toward preventing devastating neurologic consequences of severe thiamine deficiency such as KS, which the patient had already suffered in the past with residual cognitive impairment.

Dry beriberi, WE, and KS remain clinical diagnoses. Thiamine deficiency can be confirmed with a serum thiamine level, however, this may take several days to result. It is important for physicians to be familiar with both the risk factors for thiamine deficiency and its signs and symptoms since delayed diagnosis may lead to permanent cognitive impairment and physical disabilities. Such irreversible consequences can be prevented by prompt recognition and immediate thiamine repletion. Clinicians should have a high index of suspicion of thiamine deficiency in nonalcoholic malnourished patients with a psychiatric history.

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