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Wernicke's encephalopathy secondary to depression from systemic lupus erythematosus

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Abstract

Systemic Lupus Erythematosus (SLE) is a complex inflammatory disease, often involving several organs and systems. While it is uncommon for patients with SLE to present with neuropsychiatrist symptoms, the early detection and management of these symptoms is crucial. We present the case of a young SLE patient who developed severe depression soon after diagnosis, resulting in poor oral intake and consequently malnutrition. Imaging findings were consistent with Wernicke's encephalopathy. The patient was started on thiamine replacement with gradual improvement in symptoms. This case highlights the complexity associated with diagnosing and treating neuropsychiatrist conditions seen in SLE. Although literature is limited, providers should have a high index of suspicion of Wernicke's Encephalopathy as a differential in SLE patients with neuropsychiatric symptoms, especially given the high mortality rate if left untreated.

Keywords

Systemic lupus erythematosus; inflammatory disease.

Introduction

SLE is an autoimmune condition that is most prevalent among women. The reported prevalence of SLE in North America is 241 per 100,000 person years [1]. SLE can often have varying presentation and can have multi-system involvement. Some common manifestations of SLE include glomerulonephritis, arthritis, vasculitis, cutaneous symptoms, and CNS involvement. Depression and anxiety are common neuropsychiatric manifestations associated with SLE. Severe depression can lead to poor oral intake and nutrition associated diseases. We present the case of a young female with newly diagnosed SLE who developed poor oral intake and subsequent Wernicke's encephalopathy due to neuropsychiatric manifestation from SLE.

Case Presentation

Patient is a 38-year-old female with no significant past medical history who presented to the hospital initially for progressive dysphagia of both solids and liquids accompanied by 15-pound weight loss in the last 3 months. She denied any abdominal pain, nausea, vomiting, change in bowel habits, alcohol use or any other new onset issues. Vitals on admission showed temperature of 98.8^0F, heart rate of 105 beats per minute, respiratory rate of 14 breaths per minute and oxygen saturation of 97% on room air. Physical exam showed a thin African American female in no apparent distress. Oral thrush was noted. Normal bowel sounds were heard on physical exam with no masses palpated on abdominal exam. Labs showed Bun of 60 and creatinine of 1.9 mg/dL, from unknown prior baseline, normal bilirubin, and liver function tests. Complete blood count revealed pancytopenia with a hemoglobin of 10.5 g/dL, white blood count of 3300/µL, and platelet count of 93,000/µL. MCV was noted to be 90.8fl. LDH was elevated at 942 U/L. Ferritin was elevated at 2547 ng/mL. Reticulocyte was decreased at 0.33%. ANA was positive at >1:1280. Double stranded DNA was found to be positive at >300 IU/mL. C3 and C4 complement levels were significantly low at 31 mg/dL and 5mg/dL respectively. HIV test was negative. Patient underwent endoscopy which showed mild gastritis, but no esophageal lesions. Patient was diagnosed with SLE and discharged on prednisone 40 mg daily, fluconazole 100 mg daily and pantoprazole 40mg daily with plan for outpatient follow-up with rheumatology and hematology. Patient did not follow up with any physicians and was readmitted 4 weeks later for worsening dysphagia. Patient reported not taking all the medications on discharge as prescribed. Vitals on second admission showed tachycardia with heart rate of 122 beats per minute, temperature of 100.7^0F, blood pressure of 127/93 mm of Hg, respiratory rate of 18 breaths per minute and oxygen saturation of 98% on room air. Physical exam this time showed a severely depressed woman in no apparent distress. She was minimally verbal and had flat affect. Physical exam, including a complete skin examination, did not reveal any rashes of points of tenderness. Lactic acid was elevated at 2.4 mmol/L. Patient was given IV fluids and started on empiric antibiotics. Complete blood count showed macrocytic anemia with a hemoglobin of 8.5g/dL and MCV of 100.7fl. White blood count was decreased at 3500/µL and platelet count was normal at 204,000. Her bloods cultures were positive for methicillin sensitive *Staphylococcus aerues* (MSSA). CT abdomen done on admission and repeated few days after admission, did not show any occult abscess. Transthoracic and transesophageal echo did not show any valvular vegetations. Repeat endoscopy showed improving gastritis, but no other abnormalities. Due to her depressed mood and flat affect, an MRI of the brain with and without contrast was done and showed nonspecific signal abnormality within supratentorial white matter, medial thalami, mesial temporal lobes, central cerebellar hemispheres, periaqueductal gray matter, and mamillary bodies consistent with Wernicke's encephalopathy, as shown in the figure below:

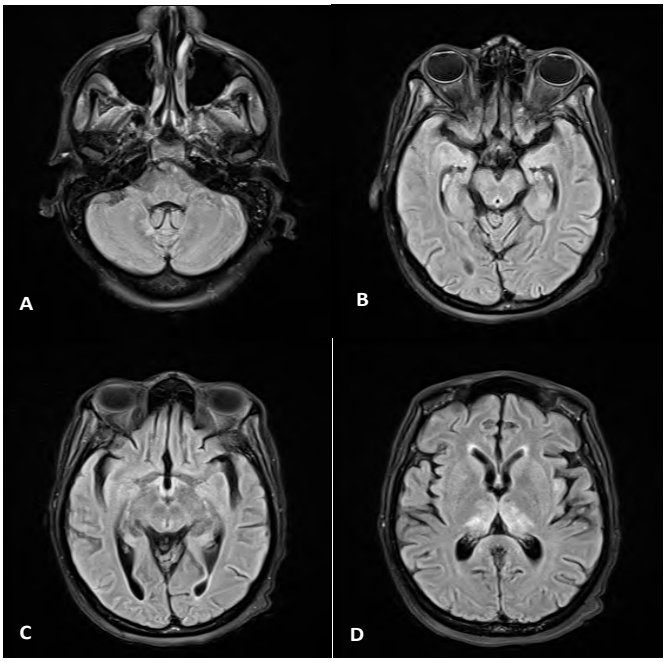

Figure 1: T2 FLAIR findsings on MRI showing high signal intensity at the cerebellum (A), mammillary bodies (B), periaqueductal gray matter (C) and medial thalami and periventricular region of the third ventricle (D)

She was started on high dose thiamine and her overall mood and affect improved. Due to concerns about continued malnutrition, a percutaneous enterogastric tube was placed and patient was discharged.

Discussion

Wernicke's encephalopathy is a neuropsychiatric condition that arise from vitamin B1 deficiency and is mostly commonly associated with alcohol abuse. Other causes of Wernicke's encephalopathy include systemic malignancy, anorexia nervosa and gastrointestinal disease or bariatric surgery. To further complicate things, the triad of oculomotor dysfunction, confusion and ataxia is only seen in about 10% of the patients with Wernicke's encephalopathy [2]. The presentation of the encephalopathy can be varying and can range from negligence to coma, depending on the length of the disease [3]. Our patient did not have any ataxia or oculomotor issues. She did however have encephalopathy characterized by severe indifference and inattentiveness.

We hypothesize that due to her poor nutritional intake as result of severe depression from SLE. As a result, patient was likely deficient of thiamine and as a result developed Wernicke's encephalopathy. Patients with SLE have been shown to have higher rates of depression and fatigue when compared to patients without SLE [4]. The extent of depression also seems to be associated with the extent of the disease [5]. Our patient had severely low complement levels and extremely high dsDNA levels, likely indicating high burden of SLE on first admission, which likely lead to the severity of her Wernicke's encephalopathy needing PEG tube placement.

Her MRI findings were also consistent with Wernicke's encephalopathy from a non-alcohol etiology. Patients with alcohol associated Wernicke's encephalopathy present with atrophy of the mammillary bodies, supratentorial regions and corpus callosum. In contrast, our patient had typical MRI changes

with symmetric enhancement in the thalami, mammillary bodies consistent with non-alcohol associated Wernicke's encephalopathy. Our patient also had signal abnormality in the central cerebellar hemispheres which has been documented as a rare and atypical finding in literature [6].

The treatment of Wernicke's encephalopathy is with the replacement of thiamine. However, there is no consensus on the route, dosage, or frequency of thiamine administration. The route of thiamine administration is dependent on the primary etiology and viability of the gut. A minimum of 100mg/day is recommended and even higher doses in alcoholics [7]. Others have proposed a 3–5 day course of IV thiamine 200mg-500mg/day followed by oral thiamine supplementation [8]. The response to treatment also seems to be relatively quick. Patients, on average recover and report improvement in about three days [9]. Our patient's mood started improving about 48 hours after administration of thiamine. She was given intravenous thiamine 100mg for 7 days, followed by oral thiamine supplementation.

Timely replacement of thiamine is essential in Wernicke's encephalopathy. Without replacement, Wernicke's encephalopathy can have a mortality rate of up to 17%. Additionally, without' treatment, Wernicke's encephalopathy can progress to Korsakoff syndrome and cause permanent memory impairment [10]. The timely replacement of thiamine lead to slow improvement in our patient's neuropsychiatric symptoms.

Conclusion

In conclusion, we present a patient with newly diagnosed SLE who presented with severe depression and developed poor oral intake subsequently. She was diagnosed with Wernicke's encephalopathy from her presenting symptoms and MRI findings. Her symptoms improved after thiamine administration as well. Our case demonstrates emphasizes that SLE can at times present with neuropsychiatric symptoms such as depression. In such cases, patients can be high risk of developing Wernicke's encephalopathy as a result. Physicians need to have a high index of suspicion for Wernicke's encephalopathy in patients with SLE who have depression symptoms. Early recognition and correction of thiamine levels in such cases can be potentially lifesaving.

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