Abstract Form

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Project Title:	Wernicke's Encephalopathy Following Bariatric Surgery

Research Category (please check one):							
☐	**Original Research**	☒	**Clinical Vignette**	☐	**Quality Improvement**	☐	**Medical Education Innovation**

Abstract

Introduction: The obesity epidemic has led to steady increases in the number of bariatric procedures performed. A wide array of post-operative complications can occur following bariatric surgery, including nutritional deficiencies. These deficiencies can have a wide range of clinical impacts, from mild to profoundly debilitating and irreversible. Here, we present a case of Wernicke's encephalopathy (WE) following gastric bypass surgery.

Case Report: A 32-year-old female with a history of hypothyroidism and gastric bypass due to morbid obesity was brought to the emergency room for abnormal behavior and altered mental status. Three days earlier, she was discharged from another hospital but did not return to her normal baseline. Normally she is conversant, independent with all her activities, and an engaged mother. Shortly after returning home her mental status suddenly changed. Changes from baseline included answering questions inappropriately, ambulating with a walker, and showing a lack of attention towards her child. On presentation, she did not show any signs of fever, vomiting, abdominal pain, diarrhea, constipation. Per family she had no recent travel or sick contacts. She had a sleeve gastrectomy 3 months prior, after which she suffered poor oral intake due to throat pain and nausea. She underwent 3 esophageal dilations in the last 3 weeks, which helped her advance from clear liquids only to eating oatmeal and pureed vegetables over the last week. Her only medications are levothyroxine and pantoprazole, both of which she was adherent to. On initial evaluation she was oriented to name only, and unable to meaningfully answer questions or engage in an interview. Extraocular movement severely limited in the vertical plane, and she had bilateral gaze palsy. She was initially unable to cooperate for a gait exam but was later found to have profound ataxia. Complete blood count and renal function were within normal ranges. She had a Vitamin B -12 level resulted normal at 400 pg/mL. Computed tomography of the head was normal. A subsequent magnetic resonance imaging of the brain showed bilateral thalamic enhancement consistent with WE. Prior to confirmatory imaging or laboratory workup she was treated empirically for Wernicke's Encephalopathy given her poor oral intake after bariatric surgery. High-dose IV thiamine (500 mg every 8 hours) was started within hours of presentation with daily folic acid. Her mental status improved over the first few days fully oriented, then to fully conversational. Her ophthalmoplegia and ataxia showed gradual improvement. She was able to ambulate with assistance from physical therapy on hospital day 5 and was discharged to an acute rehabilitation facility with continued daily thiamine and folic acid supplementation.

Discussion: WE is an acute neuropsychiatric syndrome with a classic triad of mental status change, ataxia, and eye movement disorders. It is classically associated with chronic alcohol consumers who supplant alcohol for calories but may occur in any situation that facilitates thiamine deficiencies such as sequelae of bariatric surgery (1). Typically, bariatric patients are given preventative multivitamins including thiamine supplementation. In our patient, however, she was unable to tolerate oral medications or supplements. When WE is suspected, clinicians should administer 500 mg of parenteral thiamine three times a day until symptoms of improve (2). Most WE patients after bariatric surgery often have delayed time to diagnosis and treatment during which their symptoms may progress and can ultimately lead to the irreversible Korsakoff syndrome (3). With the increasing prevalence of bariatric surgeries, clinicians should be aware of this potential complication to facilitate rapid diagnosis and treatment.

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