Paralysis Due to Thiamine and Copper Deficiencies Following Sleeve Gastrectomy Complicated by Oral Necrotizing Ulcers

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Worsening obesity and widespread availability of laparoscopic surgery has led to an increase in bariatric weight loss procedures with long term consequences including nutritional deficiencies more common after malabsorptive procedures such as gastric bypass and bilipancreatic diversion. This is a case of a restrictive surgery that produced unexpected nutritional deficiencies with lasting deleterious consequences. A 50 year old woman with a history of vertical sleeve gastrectomy 16 months prior had complications of oral ulceration due to several bouts of pancreatitis with prolonged hospitalization and total parenteral nutrition (TPN). Five weeks after surgery she developed ascending numbness and tingling of the feet leading to falls and inability to walk. She was treated for multiple vitamin deficiencies including Dry Bertiore and had several balloon dilations of her sleeve eventually tolerating enteral feeding again. She was improving with physical therapy but recurrence of oral ulceration led to progression of numbness, tingling, and weakness to include her face, hands, and abdomen causing her to present to the ED. Labs revealed deficiencies of copper and riboflavin with borderline thiamine levels as well as toxic levels of pyridoxine. Exam revealed motor and sensory polyneuropathies with an eczematous sign to the umbilicus and weakness of lower extremities with loss of vibration and proprioception. Nystagmus and memory loss were consistent with Wernicke's Encephalopathy as well. Endoscopy was unrevealing and gastrointestinal symptoms resolved prior to discharge six days later with improvement of the neuropathy of the hands and face after IV and oral thiamine and copper supplementation. Although not often associated with sleeve gastrectomy, copper deficiency is associated with zinc supplementation and use of TPN as in this case. Currently, the ASMB (American Society of Metabolic and Bariatric Surgery) only recommends checking levels in patients with suggestive symptoms who have had malabsorptive procedures, but not in restrictive. But in 2017, a small retrospective study of patients with gastric sleeves showed those with neuropathy displayed a pattern of low thiamine, copper, and riboflavin with toxic levels of pyridoxine which also causes neuropathy. Perhaps copper levels should be routine post-operatively in restrictive bariatric procedures. Consistent vitamin toxicities also speaks to the need for reevaluation of vitamin supplementation regimens.

Diagnostic Dilemma: Perforated Gastric Ulcer in the Excluded Stomach After Roux-en-Y Gastric Bypass

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Bariatric surgery has become an important therapeutic option for the treatment of severe obesity. There are multiple procedures to choose from, each with its own risks and benefits. Complications can occur such as micronutrient deficiency, dumping syndrome, and marginal ulceration. We report a case of perforated gastric ulcer occurring in the excluded stomach 15 years after Roux-en-Y gastric bypass highlighting the diagnostic and therapeutic challenges. A 50 year old female presented via phone call to the ER 44 yrs after female with history of Roux-en-Y gastric bypass for weight loss 15 years ago presented with worsening RUQ and epigastric pain present for 3 weeks. She had no fever, melaena, or hematemesis. There was no NSAID, alcohol, tobacco or drug use. She denied any past history of PUD or diabetes. PE: blood pressure 116/58, HR 71, RR 18, T 37°C, the abdomen was soft, nondistended with normal bowel sounds. Tenderness was noted in the right upper quadrant and epigastrium, but no rebound. Stool hemoccult was positive. Laboratory studies showed WBC count 8800, hgb 15.7. Liver enzymes, bilirubin, lipase, and creatinine were normal. On day 1 of admission, a CT demonstrated normal liver, gallbladder and biliary system, and pancreas, a normal gastric remnant and no leak or free air was found. On day 2, an EGD revealed no ulcer or erosion and normal effluent limb. On day 3, the patient continued to have pain. WBC became elevated despite IV antibiotics. On day 4, abdominal ultrasound of the liver and gallbladder was normal but a CCK HIDA scan revealed a gallbladder ejection fraction of 7%. On day 5, a laparoscopic cholecystectomy was attempted. On insertion of the scope was noted throughout the abdominal cavity. After conversion to an open procedure a perforation was found in the terminal ileum and a pinworm ova on the surface of the epithelium. The patient and family were treated with albendazole with increasing serum B12 levels and resolution of her symptoms. Pinworm infestation as a cause of tissue eosinophilia and Vitamin B12 deficiency would have been undiagnosed if the worm and ova had not been found on follow-up colonoscopy. Due to the low sensitivity of stool ova and parasite testing, B12 deficiency of undetermined etiology associated with tissue eosinophilia may warrant endoscopical evaluation, as pinworm infestation may be the missing link between the two findings. Additionally, more studies may be needed to evaluate if empiric antiparasitic treatment of children with B12 deficiency of undetermined etiology may be of benefit prior to the initiation of an expensive multi-specialty workup.