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Copper deficiency-induced neuropathy after bariatric surgery disguised as demyelinating disease

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Abstract

Neuropathy may arise from many different etiologies–from diabetes and nerve compression, to viral infections and chemotherapy side effects; many patients suffer from neuropathic symptoms. While some etiologies produce irreversible neuropathy, others, such as vitamin and mineral deficiencies, lead to a possibly reversible disease process once treated. Clinicians should strive for early and prompt diagnosis of neuropathy whenever possible. We present a case of a 73-year-old female with past medical history of cobalamin deficiency, post-surgical hypothyroidism, anxiety, hypertension, and surgical history of Roux-en-Y Gastric Bypass 20 years prior, who was diagnosed with acquired copper deficiency induced neuropathy, initially disguised as a demyelinating disease.

Introduction

Bariatric surgery has arisen as a tremendously helpful modality for improving weight loss and other comorbidities; however, various post-surgical complications may occur. Commonly, patients develop deficiencies of specific vitamins and minerals absorbed throughout the gastrointestinal tract. These include but are not limited to cobalamin, folate, thiamine, copper, vitamin D, and other fat-soluble vitamins. It is essential to monitor levels of these vitamins and minerals in post-bariatric surgical patients so that any deficiencies can be promptly corrected [1]. Of the mineral deficiencies, copper deficiency presents in 9.6% to 18.8% of post-Roux-en-Y Gastric Bypass (RYGB) patients. This can be particularly troublesome if not appropriately diagnosed and treated due to its hematologic and neurologic manifestations [2].

Copper distribution is mediated by binders such as ceruloplasmin and albumin. About 30-40% of dietary copper is typically absorbed throughout the gastrointestinal tract [3]. Most copper absorption occurs in the small intestine, particularly in the duodenum; however, some copper can be absorbed in the stomach as well. After certain bariatric surgeries, such as RYGB, ingested food bypasses the duodenum, resulting in significantly reduced copper absorption.

Copper is essential for the process of erythropoiesis; therefore, copper deficiency may cause anemia, neutropenia, or pancytopenia [4]. Copper deficiency may lead to symptomatic anemia, presenting with weakness, shortness of breath, dizziness, paleness, or fatigue. In addition, the neurological effects of copper deficiency, such as gait difficulty (due to dorsal column dysfunction), lower limb spasticity, and neuropathy, could potentially mimic the presentation of demyelinating disease [4]. Although less reported, copper deficiency has been shown to cause cerebral demyelination, which may further lead to diagnostic mimicking and potential misdiagnosis [4].

Case report

73-year-old female with past medical history of cobalamin deficiency, post-surgical hypothyroidism, anxiety, hypertension, and surgical history of Roux-en-Y Gastric Bypass 20 years prior, presented to the emergency department for difficulty ambulating due to painful neuropathy. She had severe pins and needles in her feet, worsening for the past three months. The sensation had progressed to involve her mid-shins and her bilateral fingertips. She stated that she often bumped into things and had fallen twice last week due to an inability to feel where she was stepping. She was informed that her cobalamin levels were low on initial investigation; however, her symptoms did not improve with cobalamin supplementation. Her primary care physician had prescribed pregabalin and gabapentin, which improved her symptoms slightly. She also had recently completed outpatient nerve conduction studies (NCS) and electromyography (EMG), which supposedly suggested that the patient had a demyelinating peripheral neuropathy. She denied fevers, chills, recent illness, or diarrhea. Her vital signs on admission were within normal limits. On exam, she was pleasant and well-appearing. She had multiple bruises, cuts, and wounds on her left lower extremity, decreased proprioception bilaterally, and decreased sensation to light touch to her mid-shins bilaterally and symmetrically. Sensation was intact in her bilateral upper extremities, hands, and fingers. Admission labs were significant for TSH 56.600 micro IU/mL, Vitamin B12 1414 pg/mL, methylmalonic acid 140 nmol/L, iron 19 ug/dL, iron saturation 5.15 %, ferritin 43.1 ng/mL, hemoglobin 11.7 g/dl.

She was admitted to the general medical floor and was discharged two days later to the acute rehabilitation floor, where further workup was completed. A lumbar puncture resulted in normal CSF studies, and she was given five days of intravenous immune globulin (IVIG) for empiric treatment of possible chronic inflammatory demyelinating polyradiculoneuropathy (CIDP). When her copper level and ceruloplasmin resulted after several days of admission, they were both low (copper 27 mcg/dL and ceruloplasmin 14 mg/dL),

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consistent with copper deficiency. She was then started on intravenous copper supplementation, which resulted in slight improvement in her neurologic symptoms. She was discharged on day 15 to complete a two-week course of oral copper supplementation. One month after discharge, her symptoms were reassessed via follow-up phone call. Her symptoms were still present, however, they did not significantly change or worsen, indicating overall slight improvement.

Discussion

It is vital for physicians to have a good understanding of the anatomy and physiologic complications of different types of bariatric surgery. Copper deficiency can lead to significant morbidity in the post-bariatric surgery patient population. Although the mechanism underlying neurological damage due to copper deficiency is unclear, the enzymes and pathways that require copper are well established. Copper plays a critical role in pathways such as electron-transport chain and oxidative phosphorylation, and serotonin synthesis [4].

Our patient went through extensive workup, including a nerve conduction study, EMG, lumbar puncture, a five-day course of IVIG, and 15-day hospital admission, to diagnose a mineral deficiency that could have potentially been discovered on screening lab tests. In this case, the diagnosis was further delayed due to diagnostic anchoring on an outpatient diagnosis of a demyelinating disease such as CIDP, and the fact that the serum copper and ceruloplasmin tests took several days to result.

In 2016, the American Society for Metabolic and Bariatric Surgery updated their integrated health nutritional guidelines for the surgical weight loss patient to include a Grade C recommendation for at least annual screening of copper status with serum copper and ceruloplasmin levels in post-RYGB patients, even in the absence of clinical signs or symptoms of deficiency [5]. This is even more important in post-biliopancreatic diversion/duodenal switch (BPS/DS) patients, in whom copper deficiency has been reported up to 90% [5].

Treatment of copper deficiency myelopathy has shown to lead to slight symptom improvement and prevention of worsening disease process [6]. Once the proper diagnosis of copper deficiency was established, our patient's distressing neuropathy slightly improved with intravenous copper supplementation once the proper diagnosis was made; however, most importantly, further neurologic deterioration was likely prevented.

Conclusion

Early diagnosis of copper deficiency in post-bariatric surgical patients by regular screening of copper and ceruloplasmin levels can prevent significant morbidity, as well as potentially unnecessary and invasive testing.

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