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CASE REPORT

Bilateral leg oedema after bariatric surgery: A selenium-deficient cardiomyopathy

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Summary This report describes a case of selenium-deficient cardiomyopathy secondary to bariatric surgery (Roux-en-Y gastric bypass surgery). A 40 year-old woman presented with bilateral leg oedema nine months after the surgical procedure. Timely diagnosis of selenium-deficient cardiomyopathy was due to the recognition of symptoms of heart failure, increased NT pro-BNP level, detection of myocardial diastolic dysfunction and impaired left ventricular global longitudinal strain by echocardiography, and early identification of selenium deficiency.

Symptoms resolution, cardiac biomarkers and echocardiographic abnormalities normalization were observed after 3 months of oral selenium supplementation and conventional heart failure therapy.

Any sign of heart failure after bariatric surgery should require screening for a nutrient-deficient cardiomyopathy.

Introduction

The rate of obesity shows a concerning rise. Worldwide obesity has more than doubled since 1980. In 2014, more than 1.9 billion adults, 18 years and older, were overweight. Of these over 600 million were obese [1].

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Healthcare professionals are frequently confronted with individuals with severe obesity and with postoperative bariatric patients because obesity is associated with significant morbidity and increased mortality. The number of bariatric operations being performed is increasing tremendously as a result of increasing medical need and the evolution of safer surgical techniques.

Obesity produces a variety of hemodynamic alterations that may cause changes in cardiac morphology and left ventricular (LV) hypertrophy which predispose to impaired LV diastolic function and less commonly, LV systolic dysfunction. Many of these alterations are reversible with substantial voluntary weight loss [2].

The onset of heart failure as a late complication of bariatric surgery is unusual. A specific management is required as shown in this case report.

Case report

A 40 year-old woman was referred to the hospital after the onset of bilateral lower extremity pitting oedema. She described a two-week history of progressive exertional dyspnea. She had no cardiovascular risk factors. Her past medical history was limited to a bariatric surgery (Roux-en-Y gastric bypass (RYGB) surgery) performed nine months earlier without complication. Initial body weight was 100 kg (body mass index: 41 kg/m^2) and body weight was 54 kg nine months after surgery. She stopped micronutrient supplementation two months after surgery. Physical examination revealed normal pulse and blood pressure, normal heart sounds, no jugular venous distension, no hepatojugular reflux and no venous disease on the lower limbs. Blood count, C reactive protein, creatinine, liver enzymes and albumin level were normal; no proteinuria was detected. N-Terminal Pro-Brain Natriuretic Peptide (NT pro-BNP) value was elevated at 1617 ng/L (normal value <300 ng/mL). ECG and chest X-ray were normal.

Thoraco-abdominal CT scan revealed no post-surgical complications, abdominal or pelvic mass, venous thrombosis, pulmonary embolism or Budd-Chiari syndrome. Transthoracic echocardiography showed no pericardial disease, normal size and volume of the cardiac chambers, and normal left ventricular ejection fraction (60%). Left ventricular (LV) global longitudinal strain (GLS) was impaired (−12.8%). A ''pseudonormal'' transmitral flow pattern was recorded (Fig. 1). A diastolic dysfunction was evidenced by the measure of mitral lateral annulus Tissue Doppler Imaging velocities (E/E′ = 17); right ventricular function and pulmonary artery pressure were normal. Cardiovascular magnetic resonance (CMR) imaging confirmed normal left and right ventricular ejection fraction (56% and 47% respectively) and did not show any late gadolinium enhancement (Fig. 2). Computed tomography (CT) coronary angiogram was normal. Cardiomyopathy with impaired left ventricular (LV) global longitudinal strain and evidence of increased LV-filling pressures was diagnosed and nutrient deficiency due to bariatric surgery was suspected. The blood levels were normal for the following nutrients: vitamins A, B1, B9, B12, C, D, E and K, calcium, copper, iron, and zinc. Serum selenium concentration was low at 53 μg/L (normal values range from 89 to 150 μg/L). Oral supplementation (2 μg/kg/day) was started and continued for three months. The leg oedema resolved after three weeks of heart failure treatment (furosemide + ACE inhibitor) and selenium supplementation. After three months, the patient was weight stable (54.7 kg), continued to be asymptomatic, NT pro-BNP (130 ng/L) and selenium blood levels (118 μg/L) had returned to normal values. Echocardiographic parameters improved significantly (normal transmitral flow pattern, E/E′ = 8 and GLS was − 16.5%) (Fig. 1).

Discussion

Selenium is a trace element essential for humans and animals, through its potent antioxidant activity. Major dietary sources of selenium are plant foods (if grown on a soil rich in selenium), animal kidneys, seafood, egg yolk and Brazil nuts [3]. The primary sites of absorption are from throughout the duodenum. Virtually no absorption occurs in the stomach and very little takes place in the remaining two segments of the small intestine. Prolonged parenteral and enteral nutrition are well-known iatrogenic causes of selenium deficiency. Gastric bypass and laparoscopic adjustable gastric banding procedures, and the accompanying dietary restrictions significantly reduce selenium absorption. Dietary selenium deficiency usually appears within 3 months after bariatric surgery [4]. Selenium deficiency can lead to cardiomyopathy, myopathy and osteoarthropathy. This deficient cardiomyopathy results from the depletion of selenium-associated enzymes which protect cell membranes from damage by free radicals and exposes to myocardial necrosis and sudden death. The time course for the development of congestive heart failure varies from several days or weeks in patients with Keshan

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