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Severe Right Ventricular Dilation Due to Selenium Deficiency in a Patient After Gastric Bypass

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Abstract

Selenium deficiency is a rare but reversible cause of cardiomyopathy increasingly recognized as a late nutritional complication of bariatric surgery, yet it is not consistently included in routine postoperative surveillance. This report describes a 40-year-old woman who presented three years after Roux-en-Y gastric bypass (RYGB) with progressive dyspnea, weakness, lower extremity edema, and dizziness in the setting of nonadherence to postoperative nutritional supplementation. She was hypotensive and bradycardic with jugular venous distension, elevated B-type natriuretic peptide (BNP), and chest imaging showing cardiomegaly with a right-sided pleural effusion. Echocardiography revealed severe, isolated right ventricular and atrial dilation with reduced right-sided systolic function, and further evaluation identified a markedly low serum selenium level (25 µg/L) with normal thyroid function and other trace elements. She was treated with intravenous selenium repletion (150 µg/day) alongside inotropic and diuretic support, with rapid hemodynamic improvement and complete resolution of right ventricular and atrial dilation on repeat echocardiography within one week. This case illustrates that selenium-deficiency cardiomyopathy can present with isolated right ventricular involvement, distinct from the left-ventricular predominance classically described, and underscores the importance of considering trace element deficiency in unexplained cardiomyopathy with a remote history of bariatric surgery. Prompt recognition and repletion can produce dramatic, complete reversal of cardiac dysfunction.

Categories: Cardiology, Pulmonology, Internal Medicine
Keywords: gastric bypass surgery, heart failure with reduced ejection fraction, nutritional deficiency, pulmonary critical care, reversible heart failure, right heart failure, selenium deficiency

Introduction

Bariatric surgery has become a cornerstone intervention for patients with severe obesity and its associated metabolic disease. Among available procedures, Roux-en-Y gastric bypass (RYGB) produces durable weight loss, reverses many obesity-related comorbidities such as type 2 diabetes mellitus, and is associated with roughly a 30% reduction in long-term all-cause mortality compared with non-surgical management [1,2]. As the number of bariatric procedures performed worldwide continues to increase, long-term postoperative complications, particularly nutritional deficiencies, have become increasingly relevant in clinical practice [3].

By constructing a small proximal gastric pouch and routing nutrient flow past the duodenum and proximal jejunum, RYGB produces a combined restrictive and malabsorptive physiology [3]. Standard postoperative supplementation lowers rates of the better-recognized deficiencies (iron, vitamin B12, folate, calcium, and vitamin D), but trace elements receive far less routine surveillance and can become deficient without early symptoms [4]. Selenium deficiency is rare but potentially life-threatening, particularly due to its association with reversible cardiomyopathy [5].

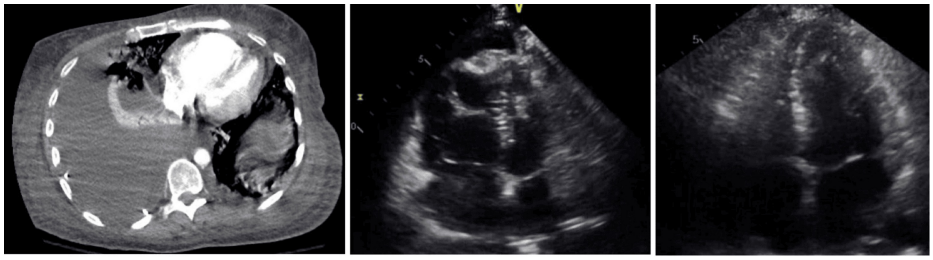
As a trace element, selenium is incorporated into a family of selenoproteins, among them glutathione peroxidases, thioredoxin reductases, and iodothyronine deiodinases, that together support antioxidant defense, redox regulation, thyroid hormone metabolism, and myocardial protection [5,6]. Severe deficiency has classically been associated with Keshan disease, an endemic cardiomyopathy first described in selenium-deficient regions of China, characterized by ventricular dilatation, heart failure, and increased mortality [7].

Although selenium-deficiency cardiomyopathy is well documented in malnutrition, chronic diarrhea, inflammatory bowel disease, bowel resection, and selenium-deficient parenteral nutrition [8,9], it remains an underrecognized cause of heart failure in post-bariatric surgery patients [10]. We present a case of severe, isolated right ventricular dilation and cardiogenic shock secondary to selenium deficiency occurring three years after RYGB, with rapid and complete reversal following selenium repletion.

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Case Presentation

A 40-year-old woman who had undergone RYGB three years earlier presented with one week of progressively worsening shortness of breath and generalized weakness, accompanied by increasing bilateral lower extremity edema and dizziness. She had not been taking her prescribed postoperative nutritional supplements. She denied relevant toxic or occupational exposures and had no significant family history of cardiac disease. On arrival, she was hypotensive (60/40 mmHg) and bradycardic (48/min). Examination revealed jugular venous distension, bibasilar crackles with decreased tactile fremitus at the right base, and bilateral non-tender pitting edema of the lower extremities. B-type natriuretic peptide (BNP) was elevated at 3500 pg/mL. The CTA chest showed bilateral vascular congestion and cardiomegaly with right-heart enlargement, no pulmonary embolism, and a right-sided pleural effusion (Figure *1A*). The electrocardiogram showed right axis deviation, deep S waves in the lateral leads, and a prominent R wave in lead V1.



(Fig 1a.) (Fig 1b.) (Fig1c.)

FIGURE 1: CTA chest and transthoracic echocardiography findings

a: bilateral congestion with cardiomegaly due to right ventricular enlargement and right-sided effusion; b: severely dilated right ventricle and atrium with diminished right-sided systolic function; c: resolution of right atrial and ventricular dilatation

Transthoracic echocardiography demonstrated a severely dilated right ventricle (basal diameter 51 mm, RV mid cavity diameter 41 mm) and right atrium, with reduced right ventricular systolic function (tricuspid annular plane systolic excursion (TAPSE) 14 mm; fractional area change (FAC) 20%; RV S' 8 cm/s). Left ventricular ejection fraction (LVEF) was 55% with normal left ventricular dimensions; interventricular septal flattening was not present. Tricuspid regurgitation (TR) was trace, with a peak TR jet velocity of 2.9 m/s and an estimated pulmonary artery systolic pressure of 22 mmHg. There was no significant left-sided valvular disease (Figure *1B*).

Competing causes of right ventricular dilation were systematically evaluated. Pulmonary embolism was excluded by CT pulmonary angiography. An agitated saline contrast study showed no intracardiac shunt, and no atrial septal defect or anomalous pulmonary venous return was identified. Troponin was elevated (280 ng/L) without ischemic ECG changes. The low-normal estimated pulmonary artery pressure did not support pre-capillary pulmonary hypertension as the cause of right ventricular failure.

The patient was started on dobutamine and intravenous furosemide and underwent right-sided thoracentesis. Further evaluation showed normal thyroid function and normal copper, manganese, zinc, calcium, iron, and vitamin levels, but a markedly low serum selenium concentration of 25 µg/L (reference ~70-150 µg/L). Intravenous selenium 150 µg/day was started and continued for 14 days, followed by oral selenium 100 µg/day for four weeks at discharge.

Hemodynamics improved, and inotropic support was weaned over the following week. Repeat echocardiography on day 7 showed resolution of right atrial and right ventricular dilation (basal diameter 35 mm; TAPSE 20 mm; FAC 40%), with LVEF 60% (Figure *1C*). Repeat serum selenium was 88 µg/L on day 20.

Discussion

Selenium deficiency-associated cardiomyopathy is a rare but reversible cause of heart failure. Historically, it has been described in association with Keshan disease, where endemic selenium depletion, combined with viral triggers such as Coxsackievirus, resulted in dilated cardiomyopathy and high mortality rates [7,11]. Beyond these endemic settings, deficiency has also been documented in patients receiving prolonged selenium-deficient parenteral nutrition, those with severe malabsorptive disease, and individuals who have undergone bariatric surgery [8,10].

Selenium is a critical component of at least 25 human selenoproteins [6]. Among the most relevant to cardiac function are glutathione peroxidases and thioredoxin reductases, which protect cardiomyocytes from oxidative injury and regulate intracellular redox signaling [6,12]. Iodothyronine deiodinases modulate thyroid hormone activation and indirectly influence myocardial contractility [6].

Deficiency results in impaired antioxidant defense, accumulation of reactive oxygen species, mitochondrial dysfunction, endothelial injury, and eventual myocardial structural remodeling [12,13]. Experimental and observational studies have demonstrated myocardial fibrosis, chamber dilatation, and reduced contractile function in selenium-deficient states [12,14]. Importantly, the cardiomyopathy is often reversible if detected early and treated promptly [13].

While selenium deficiency most commonly presents with left ventricular or biventricular systolic dysfunction, right ventricular involvement can occur in the setting of global myocardial oxidative stress [12]. In our patient, isolated severe right ventricular dilation with preserved left ventricular function suggests early or selective myocardial involvement. The thin-walled right ventricle may be more vulnerable to volume loading and oxidative injury, which could explain why it was affected first. Because isolated right ventricular dilation is atypical for nutritional cardiomyopathy, attribution to selenium deficiency rests on two findings: the exclusion of the usual causes of right heart failure and the complete structural recovery after repletion.

Isolated right ventricular dilation should prompt a structured search for more common causes before a nutritional etiology is accepted. In our patient, pulmonary embolism was excluded by CTPA, an agitated saline study showed no intracardiac shunt, the estimated pulmonary artery systolic pressure of 22 mmHg did not suggest significant pulmonary hypertension, tricuspid regurgitation was trace, and there was no evidence of ischemia or structural valvular disease. With these causes excluded and a profoundly low selenium level, selenium deficiency was the most plausible explanation.

After RYGB, micronutrient deficiencies arise frequently, driven by bypass of key absorptive segments, diminished gastric acid secretion, reduced oral intake, and inconsistent supplement use [3,4]. Studies evaluating micronutrient status after bariatric surgery have reported measurable rates of selenium deficiency, particularly in patients with poor supplement adherence [10].

Although standard postoperative monitoring guidelines have traditionally emphasized iron, vitamin B12, folate, calcium, and vitamin D, selenium has not historically been universally included in surveillance panels [3]. Notably, a 2025 American Society for Metabolic and Bariatric Surgery (ASMBS) postoperative care pathway update now recommends routine selenium screening every three months until stable, then every six to 12 months [15]. Our patient's documented non-compliance with nutritional supplementation likely contributed to progressive depletion over three years. Her serum selenium level of 25 µg/L (normal ~70-150 µg/L) confirmed severe deficiency [16].

Selenium-deficiency cardiomyopathy may present with progressive dyspnea, peripheral edema, hypotension, arrhythmias, and cardiogenic shock [13]. Elevated natriuretic peptides support a heart failure physiology but lack specificity, so identifying this entity depends on maintaining a high level of clinical suspicion, especially among patients with malabsorptive risk factors.

In patients with a remote history of bariatric surgery who present with unexplained heart failure, a micronutrient panel that includes selenium should be sent early in the hospitalization, alongside the standard evaluation for ischemia, valvular disease, pulmonary embolism, and congenital abnormalities. Selenium results often take several days, so sending the test at presentation avoids delaying repletion while other causes are being excluded. Prior case reports have demonstrated dramatic improvement in ventricular function following selenium supplementation, supporting the reversibility of this condition [8,13], whereas delayed treatment risks progression to irreversible myocardial fibrosis [12,14].

For adults, selenium intake of 55 µg/day meets the recommended dietary allowance (RDA) [16], while therapeutic repletion in deficiency states is typically dosed at 100-200 µg/day [16]. Our patient received 150 µg/day intravenously with rapid hemodynamic recovery and complete reversal of right ventricular dilatation within one week.

The speed of recovery should be interpreted with caution. Our patient received inotropic support, intravenous diuresis, and thoracentesis alongside selenium repletion. Rapid reduction of preload and right ventricular wall stress likely contributed to the early decrease in right heart dimensions within the first week. Decongestion alone, however, rarely normalizes a severely dilated and hypokinetic right ventricle so quickly in the absence of a correctable driver. We therefore view the recovery as the combined effect of hemodynamic unloading and restoration of selenoprotein-dependent antioxidant defense, rather than selenium repletion alone.

The dramatic improvement observed highlights the reversibility of selenium-deficiency cardiomyopathy when identified early. Delayed recognition may lead to irreversible myocardial remodeling or death [7,13].

Conclusions

This case highlights selenium deficiency as a rare but readily reversible cause of severe, isolated right ventricular dilation and cardiogenic shock in patients who underwent RYGB years earlier, particularly when postoperative supplementation has been inconsistent. Because selenium has not traditionally been part of

routine postoperative micronutrient surveillance, deficiency can progress silently for years before manifesting as advanced heart failure; this case underscores the rationale behind a 2025 ASMBS update that now recommends routine selenium screening after RYGB. Clinicians evaluating unexplained right ventricular dilation or cardiomyopathy, especially in patients with a history of bariatric surgery, malabsorption, or poor supplement compliance, should maintain a high index of suspicion for selenium deficiency and check serum levels early in the diagnostic workup. Because isolated right ventricular dilation is atypical for this entity, pulmonary embolism, intracardiac shunting, pulmonary hypertension, and valvular disease should be excluded in parallel, and the contribution of concurrent decongestive therapy to early recovery should be recognized. As illustrated in this patient, prompt selenium repletion was followed by rapid, complete reversal of ventricular dysfunction, whereas delayed recognition risks irreversible myocardial remodeling or death. Incorporating selenium into long-term post-bariatric nutritional monitoring may help identify at-risk patients before progression to overt cardiomyopathy.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

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