EJCRIM
European Journal
of Case Reports
in Internal Medicine

SEVERE HYPOALBUMINAEMIA AFTER ROUX-EN-Y GASTRIC BYPASS: A DIAGNOSIS OF EXCLUSION

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Received: 31/07/2025
Accepted: 11/08/2025
Published: 25/08/2025

Conflicts of Interests: The Authors declare that there are no competing interests.
Patient Consent: Written informed consent was obtained from the patient for publication of their clinical history and associated data.
Acknowledgements: The authors wish to thank all individuals who provided administrative and technical assistance during this work. We also acknowledge the support of the institution for the resources made available, which contributed to the successful completion of this study.
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How to cite this article: Santos Reis C, Lopes AF, Santos Dias F, Soares Carreira M, Almeida J. Severe hypoalbuminaemia after Roux-en-Y gastric bypass: a diagnosis of exclusion. *EJCRIM* 2025;**12**:doi:10.12890/2025_005741

ABSTRACT

Bariatric surgery has emerged as a highly effective treatment option for individuals with obesity. Severe hypoalbuminaemia is a feared complication after a Roux-en-Y gastric bypass. It is characterised by a low serum albumin level of <25 g/l, neither explained by renal losses, protein-losing enteropathy nor by liver disfunction, and is associated with high morbidity and mortality. Prompt workup leading to the diagnosis and aggressive treatment are recommended to prevent rapid clinical worsening; however, evidence-based recommendations and specific treatment approach are lacking. Most cases can be managed with dietary counselling and protein-enhanced oral supplements.

We report a case of a female patient in her 50s with class II obesity, who underwent a Roux-en-Y gastric bypass eight months earlier, who presented to the Emergency Department with new onset of asthenia and lower limb oedema. Her biochemistry panel revealed severe serum hypoalbuminaemia, with a level of 18.4 g/l. After a thorough examination, complete laboratory tests and imaging that excluded renal and gastrointestinal losses and hepatic disfunction, she was presumed to have developed severe hypoalbuminemia after bariatric intervention. She managed to increase her oral intake of protein-enhanced products, according to nutritional counselling. At a six-month follow-up visit the patient regained 1% of the weight and had a slow and progressive increase in the serum albumin level. Our report highlights the importance of continuous nutritional monitoring and a multidisciplinary approach in the post-operative period of bariatric surgery to improve long-term outcomes.

KEYWORDS

Obesity, bariatric surgery, Roux-en-Y gastric bypass, protein malnutrition, hypoalbuminaemia

LEARNING POINTS

- Severe hypoalbuminaemia caused by bariatric surgery requires exclusion of alternative aetiologies.
- Surgical reversal can often be avoided by appropriate dietary interventions.

EFIM
European Federation of Internal Medicine

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INTRODUCTION/BACKGROUND

Bariatric surgery has emerged as a highly effective treatment option for individuals with obesity. The growing body of evidence regarding the benefits of bariatric surgery has significantly contributed to its increasing popularity, particularly due to its ability to reduce comorbidities associated with obesity, improve quality of life and decrease obesity-related mortality. However, one of the most serious, though rare, complications that may arise is protein malnutrition. This condition is the primary cause of macronutrient deficiency following bariatric surgery[1].

Protein malnutrition is defined as a nutritional deficiency resulting from inadequate intake or absorption of protein, leading to measurable biochemical and clinical consequences. One of its key laboratory indicators is hypoalbuminaemia, which reflects the patient's protein status. Hence, protein malnutrition could be characterised according to serum albumin levels, generally below 35 g/dl, with the normal range being 35–50 g/l. Albumin levels between 30–35 g/l are typically classified as mild, 25–30 g/l as moderate, and levels below 25 g/l as severe hypoalbuminaemia[1,2]. Overall, it is estimated that protein malnutrition may occur in up to 1.7% of cases, with the median incidence varying among patients undergoing bariatric surgery[2]. Dietary changes, along with subsequent physiological alterations in the gastrointestinal tract, place these patients at risk for nutritional deficiencies[3]. Medical complications related to the procedure emerge when albumin levels drop below 25 g/l, correlating with an annual hospitalisation rate of 1%[4]. Patients with hypoalbuminaemia also exhibit higher mortality compared to those without hypoalbuminaemia (0.4 vs 0.14%), with a rehospitalisation rate of 10.87%[5]. These patients require close nutritional support, including the introduction of oral nutritional supplements with high-quality protein content, as the most common cause of hypoalbuminaemia remains insufficient oral intake of protein.

Although other causes must be excluded in these patients, extensive diagnostic workups have consistently ruled out underlying infectious, metabolic, autoimmune liver diseases or inflammatory bowel disorders. However, in cases of reduced intake, protein intolerance, excessive malabsorption or gastrointestinal complications, patients are at high risk of rapidly becoming protein deficient. If left untreated, severe hypoalbuminaemia can lead to anaemia, oedema, Kwashiorkor disease and acute liver failure[2].

The treatment approach varies depending on the severity of the condition, with several treatment options available including high-protein supplements, enteral feeding or parenteral nutrition. However, evidence-based recommendations for a specific treatment approach within these regimens are not currently available[1].

High-protein feeding can be beneficial but carries risks, especially in individuals with severe protein and energy depletion. In these patients, the breakdown of endogenous proteins for glucose production may lead to hyperammonaemia. Elevated ammonia levels in post-bariatric patients with hypoalbuminaemia can indicate a urea cycle defect, which is often congenital but can go unnoticed until adulthood, particularly when stressed by nutritional deficiencies in essential amino acids. Urea cycle defect is more common in post-bariatric patients[6].

In some cases, patients may not respond to conservative treatment and may require surgical revision to optimise absorption or even restore normal anatomy, although such procedures are rarely performed[2].

CASE DESCRIPTION

A 50-year-old Caucasian woman with grade II obesity (weight 81 kg, BMI 36 kg/m^2) with complications (medicated arterial hypertension) underwent a Roux-en-Y gastric bypass (RYGB) eight months earlier. According to the intraoperative report, the course of surgery was uneventful, and her Roux and biliopancreatic limbs were 110 cm. In the next months, she achieved weight loss down to 49 kg, her blood pressure normalized, and her antihypertensive medications were discontinued. She presented again to the emergency department with complaints of fatigue and bilateral lower limb swelling, progressively worsening in the previous four months. She denied gastrointestinal symptoms, and her bowel movements were regular, although she was not adhering to physical exercise and was not following the prescribed diet. Her medication was a daily multivitamin. On physical examination, her blood pressure was 92/62 mmHg with no difference between the limbs, heart rate was 85 beats/min and oxygen saturation was 98% on room air. Pulmonary and cardiac auscultation were normal, with no murmurs or gallops; there was no jugular venous distention. A symmetric pitting oedema below the knees (Godet scale grade 2) was noted, and limb signs of arterial ischaemia and venous thrombosis were ruled out. The electrocardiogram showed no signs of right-sided heart overload or conduction abnormalities. Laboratory results showed a blood glucose level of 84 mg/dl, hypoalbuminaemia of 18.4 g/l, hypoproteinaemia of 36.4 g/dl, and renal, thyroid and liver function, as well as coagulation, within the normal range. She was admitted as inpatient, and a thorough evaluation and additional diagnostic tests were performed. Serum alpha-1 antitrypsin levels were also within the normal range. Urine sediment showed no albuminuria, and there were no vitamin deficiencies. A 24-hour urine collection showed no proteinuria. Bacteriological and parasitological stool tests were negative and immunological studies were negative. Abdominal ultrasound showed no signs of liver disease; abdominopelvic computed tomography (CT) scan angiography and lower limb doppler ultrasound showed no signs of obstruction. Upper and lower gastrointestinal endoscopic examinations showed normal mucosa, with unremarkable gastric and duodenal biopsies.

After excluding other diagnoses, the patient was assumed to have severe hypoalbuminaemia following her previous gastrointestinal intervention. The decision to reverse the gastric bypass was postponed by the multidisciplinary team,

and an attempt of conservative medical treatment was made. The patient managed to intensify the oral protein supplementation under the guidance of a nutritionist and her specialised oral diet, with dietary recommendations, included 1,500 kcal and 60 g of protein per day, in addition to oral supplementation of minerals and vitamins. After she was discharged from hospital, weekly follow-up visits for the first month and monthly thereafter were scheduled. Her body weight remained stable with no fluid retention, and there was an improvement in her laboratory results, showing a slow and progressive increase in serum albumin levels.

DISCUSSION

Obesity is a concept whose definition has evolved over time. Clinical obesity is now recognised as a chronic and systemic disease that can lead to severe target organ damage, resulting in life-altering and potentially life-threatening complications. Therefore, it should not be defined solely by body mass index. Its management involves a comprehensive approach including nutritional therapy, physical activity and, in selected cases, pharmacological treatment. When these therapeutic strategies fail, bariatric surgery becomes a viable treatment option. Despite its low mortality rate, bariatric surgery is associated with an increased risk of surgical, metabolic and nutritional complications[7,8].

Hypoalbuminaemia is currently regarded as one of the most significant modifiable predictors of serious post-operative complications following bariatric surgery. Implementing strategies to identify and optimise preoperative serum albumin levels has emerged as a key measure to reduce post-operative risk. A preoperative weight loss of >10%, when combined with moderate to severe hypoalbuminaemia, synergistically increases the risk of severe malnutrition and must be addressed prior to surgical intervention[9].

Severe hypoalbuminaemia is linked to increased morbidity and mortality and requires high clinical suspicion and prompt intervention. Although commonly related to inadequate dietary intake or acute precipitating factors, a thorough diagnostic workup is necessary to identify underlying causes and guide appropriate management, as demonstrated in our report.

Close and long-term post-operative monitoring is essential to detect this deficiency, as it can manifest several years after the procedure. However, adherence to long-term follow-up is often suboptimal, as patients frequently report feeling well and are satisfied with their weight loss outcomes. Moreover, nutritional deficiencies may remain asymptomatic for extended periods, and by the time clinical signs emerge, micronutrient and macronutrient stores are typically severely depleted[1-3]. In our case, the most plausible causes of hypoalbuminaemia were a combination of reduced oral protein intake and a degree of metabolic resistance to protein absorption following RYGB.

Currently, dietary guidelines for post-bariatric surgery patients recommend an average daily protein intake of 60–120 g/day or 1.1–1.5 g/kg of ideal body weight[2]. For patients undergoing biliopancreatic diversion, a further 30% increase in protein intake is recommended.

A case series has shown that continuous 24-hour feeding via a nasal-jejunal tube delivering medium-chain triglycerides, combined with frequent pancreatic enzyme supplementation, was effective in all patients with severe post-bariatric hypoalbuminaemia and was not associated with adverse effects. This approach involved continuous enteral feeding enriched with essential amino acids and medium-chain triglycerides, along with pancreatic enzyme supplementation every three hours to enhance digestion and nutrient absorption within the alimentary limb[6]. The risk of protein malnutrition is generally lower following restrictive procedures and increases proportionally with the extent of the malabsorptive component. Following standard RYGB, the incidence is relatively low for most patients[1]. In one case series, five out of six patients developed initial complications such as anastomotic ulcers, chronic diarrhoea or intestinal obstruction after standard RYGB[6].

Laparoscopic reversal of RYGB is a technically demanding procedure but can be safely performed in selected patients with severe complications, including excessive weight loss, dumping syndrome and postprandial hypoglycaemia refractory to all other therapeutic options. Given the non-negligible risk of complications, surgical reversal should be reserved for well-selected cases[9]. In rare cases of treatment-refractory conditions, surgical reversal becomes mandatory, with protein malnutrition being the leading indication for this intervention (12.3%)[5]. Besides that, a trial of conservative management was implemented in our case.

Moreover, the length of the biliopancreatic limb (BPLL) plays a crucial role in minimising post-operative nutritional complications, although the optimal length remains controversial. Nutritional deficiencies are typically observed when the BPLL is 200 cm or longer. Reports in the literature have documented cases of hepatic failure and death in the setting of hypoalbuminaemia associated with BPLL lengths of 200 cm[10]. Our patient had a BPLL of 110 cm.

Besides the clinical suspicion, diagnostic workup and conservative management of severe hypoalbuminaemia after RYGB, our case also highlights the importance and benefit of post-discharge follow-up with regular outpatient care, leading to gradual improvement in laboratory results, eventually approaching normal reference values. This outcome emphasises the need for specialised care in the prevention and management of nutritional complications following bariatric surgery.

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