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# Secondary Kwashiorkor Disease in a Patient with Gastric Bypass Surgery and Short Gut Syndrome

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Data Collection B  
Statistical Analysis C  
Data Interpretation D  
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**Conflict of interest:** None declared

**Patient:** Female, 60-year-old  
**Final Diagnosis:** Malnutrition  
**Symptoms:** Edema  
**Medication:** —  
**Clinical Procedure:** —  
**Specialty:** General and Internal Medicine

**Objective:** Rare disease

**Background:** Kwashiorkor disease is a subtype of severe acute protein malnutrition characterized by peripheral edema associated with hypoalbuminemia and ascites. It can result from both protein deficiency and protein loss. In resource-poor countries, the disease often is caused by inadequate dietary intake, but in resource-rich countries, it can be seen as a rare complication of severe malabsorption.

**Case Report:** We present the case of a 60-year-old woman who presented with 1 week of progressive anasarca in the setting of decreased dietary intake and poor tolerance of total parenteral nutrition (TPN). She had a history of Roux-en-Y gastric bypass surgery which was complicated by a strangulated internal hernia that required an exploratory laparotomy and small bowel resection. She subsequently developed short gut syndrome with TPN dependence. Work-up revealed hypoalbuminemia with several micronutrient deficiencies consistent with secondary kwashiorkor disease. With a multidisciplinary approach that included Gastroenterology, Pharmacy, and Nutrition, she was treated with albumin, furosemide, nutritional supplementation, and ultimately rechallenged with TPN. At discharge, her swelling had improved, her weight had decreased, and her albumin improved to the normal range.

**Conclusions:** This case is a unique presentation of secondary kwashiorkor disease. In our patient, the combination of gastric bypass surgery and short gut syndrome with poor TPN tolerance likely led to severe protein malabsorption. This underscores the importance of recognizing the signs and symptoms of kwashiorkor disease and understanding the associated complications so that treatment can be instituted promptly. Furthermore, the case demonstrates how an interdisciplinary approach to management can increase the chance of a successful outcome.

**Keywords:** Gastric Bypass • Kwashiorkor • Malnutrition

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## Background

Kwashiorkor disease is a subtype of severe protein malnutrition characterized by peripheral edema associated with hypoalbuminemia and ascites [1-3]. It is an underrecognized cause of protein-energy malnutrition that can be seen in adults as a result of both protein deficiency and protein loss [3-5]. In resource-poor countries, this is often the result of inadequate dietary intake and subsequent caloric deficit. In resource-rich countries, however, the causes are malabsorption, chronic alcoholism, kidney disease, and even bariatric surgery [4,5]. Common features include symmetric peripheral edema, apathy, a “moon” appearance of the face, peeling of the skin, loss of hair, ascites, micronutrient deficiency, and increased risk of infection [4]. We present the case of a 60-year-old woman with progressive anasarca from secondary kwashiorkor disease in the setting of gastric bypass surgery and a strangulated internal hernia that was treated with small bowel resection and complicated by short gut syndrome and dependence upon total parental nutrition (TPN).

## Case Report

A 60-year-old woman presented to the Emergency Department (ED) with a 1-week history of progressive anasarca. She had undergone Roux-en-Y gastric bypass surgery 14 years previously, which was complicated by volvulus with a strangulated internal hernia. Seven years later, she developed diffuse gangrene, which required emergent exploratory laparotomy and resection of her small bowel from the gastrojejunal anastomosis to the terminal ileum with sparing of her pancreaticobiliary limb. She subsequently developed short gut syndrome (45 cm of remaining small intestine) and was dependent upon TPN.

Before presenting to the ED, the patient had been eating 7 small meals per day and was only tolerating 3 hours of intermittent TPN per day. Her TPN formula consisted of 300 g of dextrose, 50 g of amino acids, and 65 g of soybean oil, medium-chain triglycerides, and olive and fish oil (SMOF). She denied fevers, chills, shortness of breath, chest pain, orthopnea, abdominal pain, nausea, vomiting, or diarrhea. She also denied any changes in diet, bowel movements, weight, or medications.

Upon presentation, the patient was afebrile with a heart rate of 100 bpm and a blood pressure of 90/76 mmHg. Her body mass index was 18.03 kg/m<sup>2</sup>. Physical examination revealed a thin, cachectic, and listless elderly woman with diffuse swelling of her upper and lower extremities, abdomen, and face (Figure 1). She also had skin peeling off her hands and feet (Figure 2). Her cardiac, pulmonary, and abdominal examinations were unremarkable.



**Figure 1.** Image showing bilateral facial swelling or a “moon” appearance of the face, which is a common finding associated with severe acute malnutrition or kwashiorkor disease.

The initial work-up revealed an albumin level of 1.6 g/dL with normal renal and hepatic function. A complete blood count; basic metabolic panel; levels of calcium, magnesium, phosphorus, amylase, lipase, triglycerides, iron, folate, vitamin B<sub>12</sub>, thyroid stimulating hormone, and thiamine; and studies of stool samples all were unremarkable. The patient had low levels of copper (41.9 µg/dL), zinc (39.1 µg/dL), vitamin D (14 ng/mL), and total protein (1.0 g/dL). Her brain natriuretic peptide level was 461 pg/mL. Her alpha-1 antitrypsin level was 238 mg/dL with 24-hour stool levels >1.13 mg/g (reference range 0-0.5 mg/g). A total stool volume was not obtained. Urinalysis showed no proteinuria. Chest X-ray and computed tomography scans of the patient’s abdomen and pelvis were unrevealing. A transthoracic echocardiogram showed no congestive or high-output heart failure.

Gastroenterology, Pharmacy, and Nutrition all were consulted. The patient was treated with albumin, furosemide, and nutritional supplementation (copper, zinc, and vitamin D), and ultimately rechallenged with TPN, which was compressed to 12 hours per day. She passed a swallow evaluation and tolerated enteral feeding. Her course was complicated by *Lactobacillus* and *Escherichia coli* bacteremia, for which she received 7 days of ceftriaxone at a dose of 2 g per day. On discharge on hospital day 26, the patient’s swelling had improved, her weight decreased from 120 to 91 lb, and her albumin level had improved to 3.7 g/dL.

At clinic follow-up 10 months later, she was tolerating 3 meals and 12 hours of TPN per day. Her TPN formula consisted of 190 g of dextrose, 30 g of amino acids, and 45 g of SMOF for



**Figure 2.** Images showing bilateral peeling of the skin on the patient's **A:** hands and **B:** feet, which is a common finding associated with severe acute malnutrition or kwashiorkor disease.

a total of 1266 kcal per day. Her weight and albumin level remained stable at 100 lb and 3.5 g/dL, respectively. Repeat testing for copper, zinc, vitamin D, and total protein showed that all of those levels had normalized.

## Discussion

This patient met both the European Society of Clinical Nutrition and Metabolism and American Society of Parenteral and Enteral Nutrition diagnostic criteria for malnutrition and the peripheral edema associated with hypoalbuminemia and ascites that were suggestive of kwashiorkor disease [1,2]. Secondary kwashiorkor disease is an underrecognized cause of severe protein malnutrition [4,5]. Common features include symmetric peripheral edema, apathy, listless affect, a “moon” appearance of the face, peeling of skin, loss of hair, and a distended abdomen [4]. Kwashiorkor disease also is associated with hypothermia, hypotension, and bradycardia [4]. Complications include dehydration, impaired nutrient absorption, vitamin deficiencies, impaired pancreatic exocrine function, fatty liver disease, and infection [4].

The pathogenesis of kwashiorkor disease involves protein deficiency and hypoalbuminemia, which causes third spacing of fluids into the interstitium [6]. However, emerging evidence suggests a relationship with the intestinal microbiome [7]. The diagnosis is clinical and the work-up should include routine laboratory tests; tests for albumin, iron, thiamine, zinc, and fat-soluble vitamin levels; and an investigation for causes of malabsorption [8]. The differential diagnosis includes other causes of symmetric peripheral edema (cardiac, hepatic, and renal dysfunction) and protein loss or malabsorption (pancreatic insufficiency, protein losing enteropathy, and nephrotic syndrome). Treatment includes carefully restarting adequate feeding, correcting fluid imbalances, replenishing any micronutrients that are deficient, and preventing or managing complications [4,8]. While kwashiorkor disease can be life-threatening if left untreated, most adults will recover from it [5,8].

It is interesting to discuss the etiology of malnutrition in this case. The patient had 2 major risk factors: gastric bypass surgery and short gut syndrome. While gastric bypass surgery is associated with several deficiencies in nutrients such as vitamin B<sub>12</sub> (19-35%), calcium (10%), vitamin D (25-73%), and thiamine (49%), it is less commonly associated with protein deficiency (7-21%) [9]. However, current recommendations for patients who have undergone gastric bypass surgery include a high-protein diet of 60 to 20 g/d [9]. Similarly, short gut syndrome is associated with deficiencies in several nutrients, including fat-soluble vitamins, vitamin B<sub>12</sub>, zinc, copper, and selenium [10-12]. However, protein deficiency is less common, and studies have shown minimal impact on amino acid and protein absorption and metabolism [13]. While 5 to 6 small meals per day are recommended for patients with short gut syndrome, they often require no change in dietary protein intake [12,13]. Therefore, in our patient's case, it is likely that the combination of gastric bypass surgery and short gut syndrome with poor TPN tolerance led to both protein deficiency and protein loss and resulted in severe protein malnutrition.

This case demonstrates that patients with a history of bariatric surgery, which is increasingly being used to treat morbid obesity, are at higher risk for protein malnutrition. It highlights some of the long-term nutritional complications of the procedure and the need for longitudinal, multidisciplinary care involving surgeons, internists, gastroenterologists, pharmacists, and dietitians to ensure a healthy nutritional status, especially in patients who have additional risk factors for malnutrition.

## Conclusions

This case is a unique presentation of secondary kwashiorkor disease. While it is an extremely rare complication of Roux-en-Y gastric bypass surgery [5,9], in our patient, the combination of gastric bypass and short gut syndrome plus poor TPN tolerance likely caused severe malnutrition. Her presentation underscores the importance of recognizing the signs

and symptoms of malnutrition, such as kwashiorkor disease, and understanding the associated complications so that treatment can be initiated promptly. Furthermore, this case demonstrates how an interdisciplinary approach to management involving Gastroenterology, Pharmacy, and Nutrition can increase the chance of a successful outcome.

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## Conflicts of Interest

None.