

Acute liver failure after bariatric surgery

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Abstract

Obesity is a crucial health problem that is common today and may lead to systemic organ dysfunctions. In its treatment, bariatric surgeries are applied with increasing frequency and provide patients lose weight. However, it causes some complications in the post-operative period. Two of these rarely complications are acute liver failure and long-term chronic liver disease. Here, we present a case that died due to acute liver failure after bariatric surgery. If severe malnutrition persists in these patients, reversal surgery should be performed.

Keywords: Bariatric surgery; liver failure.

Introduction

Morbid obesity is increasingly seen in the industrial world. Approximately 630 million adults are estimated to be obese,^[1] which causes comorbid conditions, such as diabetes, cardiovascular diseases, sleep apnea syndrome, osteoarthritis and an increase in some types of cancer. Obesity may cause complications, such as non-alcoholic fatty liver disease, cholelithiasis and gastroesophageal reflux disease in the gastrointestinal tract. Bariatric surgery is applied in patients who do not respond to diet, exercise and medical treatment with a body mass index >40 kg/m². Laparoscopic gastric band, sleeve gastrectomy, Roux-en-Y gastric bypass and biliopancreatic diversion with duodenal switch are frequently applied bariatric procedures.

Weight loss is achieved by surgery in the majority of patients. On the other hand, it may lead to complications, such as a gastric leak, stenosis, marginal ulcer, choledocholithiasis, short bowel syndrome, dumping syndrome, hernia, electrolyte imbalance or nutritional deficiency. Another complication that rarely develops after bariatric surgery and causes mortality is acute liver failure.

Case Report

A 39-year-old female patient was admitted due to weakness, anorexia, nausea and diarrhea after bariatric surgery performed four months ago.

Sleeve gastrectomy was performed four years ago when she was 110 kilograms (Body Mass Index: 40.4 kg/m²). After the operation, the patient had regressed to 68 kg. In the last one year, patient gained weight of 30 kg again and SADI (Single anastomosis duodeno-ileal bypass) operation was performed four months before. She lost 25 kilograms after the second surgery (BMI: 26.8 kg/m²). Patient was using diphenoxylate + atropine sulfate due to watery diarrhea. She was hospitalized for malnutrition and hypoalbuminemia in the previous month. Physical examination revealed bilateral pretibial edema in the lower extremities. Laboratory examinations of the patient were as Table 1.

No proteinuria was detected in urinalysis. An enteral feeding solution was administered because of the patient's diarrhea and malnutrition findings. Parenteral nutrition support was given to the patient due to she could not tolerate enteral solutions. Albumin replacement and furosemide were administered for pretibial edema. Gastroscopy was planned due to nausea. Tube stomach and normal gastroenterectomy anastomosis were seen in gastroscopy. In abdominal tomography, liver parenchymal density decreased in accordance with steatosis (Fig. 1). Thyroid function tests were normal. Celiac antibodies were negative. No pathology was detected in stool examinations. During her follow-up within 20 days, the patient's total bilirubin value was progressed up to 11 mg/dL. INR value increased up to 2.9. Transaminases reached the level of AST: 585 U/L, ALT: 192 U/L. Ascites were developed in the patient. Patient's hepatitis serology, autoimmune markers and ANCA were detected as negative. Immunoglobulin G and Immunoglobulin M were at normal limits. Alpha-1 antitrypsin level was also detected within normal limits. Ceruloplasmin value was 0.105 g/L (0.2–0.6). Intrahepatic bile ducts were not differentiated in MRCP due to parenchymal compression. The extrahepatic biliary tract and choledoch were normal. Vascular structures were clearly observed on computed tomography angiography. Liver biopsy could not be performed due to coagulopathy. Reversal surgery was planned for the patient, but acute liver failure developed during this period. While the patient was being followed up due to acute liver failure and evaluated for liver transplantation, she died due to the development of pneumonia. The patient's consent was obtained for this study.

Discussion

Bariatric surgery is now routinely applied in increasing numbers in the treatment choice of obesity. Weight loss by bariatric surgery prevents non-alcoholic fatty liver disease (NAFLD) and cirrhosis after its progression. Decreased leptin, glucose and insulin levels with adipose tissue reduce inflammation in adipose tissue.^[2] In approximately 60% of patients after bariatric surgery, steatosis in the liver regresses, as confirmed by biopsy. On the other hand, 12% new NAFLD or worsening of fibrosis may occur.^[3] In particular, this situation occurs after jejunioileal bypass and biliopancreatic diversion. Changes in the intestinal mucosa

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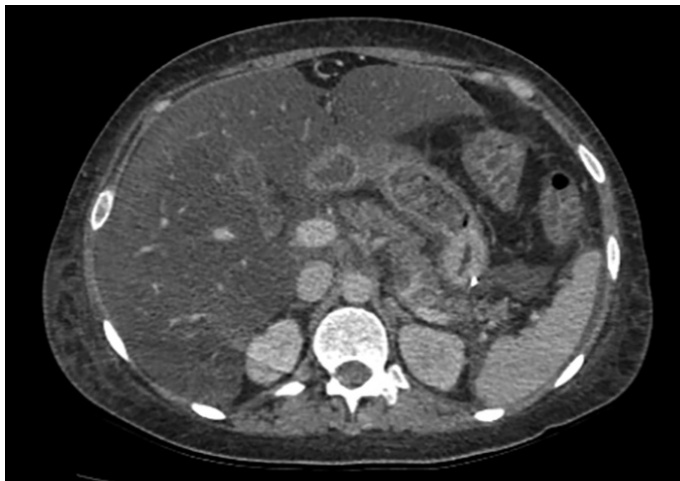
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Table 1. Laboratory findings of the patient

Es WBC: 8600	(4–10.300 / μ L)
Hb: 10.4	(12–16 g/dL)
MCV: 87	(80–95 fL)
PLT: 323000	(156–373 * 10^3 / μ L)
INR: 1.4	(0.8–1.2)
AST: 35	(0–35 U/L)
ALT: 36	(0–35 U/L)
ALP: 92	(30–120 U/L)
GGT: 66	(0–38 U/L)
Total bilirubin: 1.37	(0.3–1.2 mg/dL)
Direct bilirubin: 0.5	(0–0.2 mg/dL)
Total protein: 5.35	(6.6–8.3 g/dL)
Albumin: 1.89	(3.5–5.2 g/dL)
Prealbumin: 0.104	(0.2–0.4 g/L)
Serum iron: 103	(60–180 μ g/dL)
Iron binding capacity: <55	(155–355 μ g/dL)
Total iron binding capacity: 158	(250–450 μ g/dL)
Ferritin: 244	(11–306 ng/mL)
Transferrin saturation: 49	(12–45%)
Vitamin B12: 700	(126–505 pg/nL)
Folate:	7.49 (3.1–19.9 ng/mL)
Zinc: 55	(70–114 μ g/dL)

WBC: White blood cell; MCV: Mean corpuscular volume; PLT: platelet; INR: International normalized ratio; AST: Aspartate transaminase; ALT: Alanine transaminase; ALP: Alkaline phosphatase; GGT: Gamma-glutamyltransferase.

**Figure 1.** Fatty liver in tomography.

and bacterial flora that provide by-pass of intestinal content could be causes of increasing liver fibrosis in the postoperative period. It is believed that hepatotoxic molecules, such as inflammatory cytokines and intestinal toxins after the formation of the mucosal barriers and bacterial overgrowth, cause hepatocyte damage through the portal venous system.^[4,5] Many factors are effective in bacterial overgrowth formation: the absence of intestinal bile, short intestine, decreased gastric acidity, dysmotility, protein deficiency and undigested food reaching the colon. Another theory is that hepatic metabolism is affected by nutritional

changes.^[6] Hepatotrophic factors reaching the liver decrease after protein malnutrition. Also, most obese patients have NAFLD and may progress to acute liver failure after acute malnutrition. In a systemic review, the median time between the development of liver failure after bariatric surgery in 32 patients was 20 months.^[7] In a study conducted in Belgium, it was stated that liver failure started after postoperative periods ranging from 13 months to 21 years.^[8] Liver failure secondary to malnutrition is a rare condition and has also been described in non-bariatric patients. If malnutrition cannot be treated, reversal surgery should be performed before liver failure develops. If liver failure develops, it should be performed simultaneously with reversal surgical transplant or in the early post-transplant period.^[8]

Severe protein and nutritional deficiencies due to malnutrition developed in our patient approximately four months after the operation. In these patients, thiamine, cyanocobalamin, folic acid, iron, calcium, vitamin A and vitamin C levels should be replaced.^[5] No data were available on whether there was fibrosis in the liver since no liver biopsy was performed before or during the operation. In the post-op period, weight loss should be ensured by following these patients in a controlled diet. Patients should be monitored not only in the early post-op early period, but also lifelong concerning chronic liver disease, as well as weight tracking.

Informed Consent: Written informed consent was obtained from the patient for the publication of the case report and the accompanying images.

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Conflict of Interest: The authors have no conflict of interest to declare.

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