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CASE REPORT

Liver failure after long-limb gastric bypass

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Summary Liver failure is reported with increasing frequency in patients who underwent bariatric surgery for morbid obesity. In most cases, liver transplantation is the only possible treatment to avoid fatal outcome. Although most cases are observed after older bariatric surgery techniques characterized by high malabsorption rates, we report on a 38-year-old woman who developed liver failure seven months after long-limb gastric bypass.

Case presentation

We present a 38-year-old woman admitted to our service of hepatology for jaundice. Seven months before, she underwent long-limb gastric bypass surgery (alimentary limb 150 cm, biliopancreatic limb 60 cm and common limb 160 cm) for morbid obesity with a body mass index (BMI) of 39.5 kg/m^2 and multiple obesity-related comorbidities, including debilitating osteoarthritis, nonalcoholic fatty liver disease (NAFLD), gastroesophageal reflux disease and impaired fasting glucose. Liver function tests at that moment were not significantly abnormal (Table 1) and liver biopsy showed a moderate grade of microvesicular steatosis without significant fibrosis. In the postoperative period, she suffered from chronic diarrhea and experienced a total weight loss of 36 kg, corresponding to a reduction in BMI to 22.9 kg/m^2. Compliance to supplementation of vitamins, trace elements and essential amino acids was inadequate and the patient did not meet regular follow-up visits.

On admission, clinical examination revealed jaundice without ascites or signs of hepatic encephalopathy. Laboratory results showed an acute cholestatic hepatitis with signs of impaired hepatic synthetic capacity (Table 1). Serum markers for infectious causes (hepatitis A virus, hepatitis B virus, hepatitis C virus, hepatitis E virus, Epstein-Barr virus, cytomegalovirus and herpes simplex virus) and non-infectious causes of liver injury (antinuclear antibody, antineutrophil cytoplasmic antibody, anti-mitochondrial antibody, anti-liver kidney microsome antibody, anti-smooth muscle antibody, copper, ceruloplasmin, α1-antitrypsin and ferritin) were all normal. No use of alcohol, drugs or other hepatotoxic substances was reported. Abdominal ultrasound, computed tomography (CT) and magnetic resonance imaging (MRI) showed a normal liver parenchyma without signs of biliary obstruction, portal thrombosis, venous

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Table 1 Evolution of liver function tests and vitamin status.

	Unit	Reference range	−7 months Baseline	D1 Admission	D25 Start TPN	D34	D37 Transfer	D43 Liver Tx	Tx + 30d	Tx + 90d
INR			NA	1.5	2.0	2.0	3.4	3.2	1.2	1.1
Albumin	g/L	35–52	NA	NA	28	24	27	25	44	43
Total bilirubin	mg/dL	≤1.18	0.30	7.8	12.7	11.4	20.2	21.4	1.57	0.36
ALP	U/L	≤105	59	118	105	159	195	163	213	55
GGT	U/L	≤40	46	162	166	157	171	75	144	16
AST	U/L	≤31	14	767	553	431	519	266	38	21
ALT	U/L	≤31	16	1438	714	434	576	311	119	50
HDL	mg/dL	≥45	52	NA	9	NA	9	NA	57	48
25OH vitamin D	ng/mL	11–60	NA	NA	9.8	NA	NA	NA	NA	23.7
Vitamin A	μg/L	300–650	NA	NA	21	NA	24	NA	NA	583
Vitamin E	mg/L	5–20	NA	NA	13.2	NA	9.9	NA	NA	8.0
Zinc	mg/L	0.5–1.2	NA	NA	0.4	NA	2.7	NA	NA	6.4

INR: international normalized ratio; ALP: alkaline phosphatase; GGT: γ-glutamyltransferase; AST: aspartate transaminase; ALT: alanine transaminase; Tx: transplantation; TPN: total parental nutrition; D: day; NA: not available.

outflow tract obstruction or chronic liver disease. Revision of peroperatively performed liver biopsy confirmed the initial finding of microvesicular steatosis without significant fibrosis and showed no morphological or immunohistochemical evidence of familial intrahepatic cholestasis. In addition, diagnostic exploration of the patient's chronic diarrhea was performed: faecal analysis showed no enteropathogens, CT and MRI of the abdomen showed no signs of bowel inflammation or intestinal subobstruction and colonoscopic examination was normal.

Since no cause for the patient's liver dysfunction was identified after extensive work-up, our diagnostic hypothesis was liver injury due to severe nutritional deficiencies caused by malabsorption and inadequate compliance to nutritional supplements after bariatric surgery. This hypothesis was supported by the patient's chronic diarrhea and the presence of multiple nutritional deficiencies, in particular a severe deficiency of vitamin A (Table 1).

Total parenteral nutrition with supplementation of vitamins and trace elements was started. Although this led to initial stabilization of liver function tests, rapid liver decompensation with overt hepatic encephalopathy occurred 12 days after total parenteral nutrition was started (Table 1). The patient was transferred to a transplantation unit where she underwent urgent liver transplantation, six weeks after admission to our hospital. During this liver transplantation, a gastrostomy tube was inserted into the bypassed stomach as a way of feeding the patient enterally in the postoperative period; however, there has not been a need to use it. Pathologic examination of the explanted liver revealed extensive panlobular necrosis with underlying steatosis, but no signs of fibrosis or cirrhosis. The course of her recovery was characterized by satisfactory clinical improvement and normalization of liver function tests. Six months after liver transplantation, the patient is still doing well.

Discussion

The success of bariatric surgery in the treatment of morbid obesity has grown in the last decades since it has shown to produce durable weight loss and favorable effects on obesity-related comorbidities [1]. Our patient was judged to be a good candidate for bariatric surgery because of persistent obesity (BMI 39.5 kg/m^2) despite lifestyle changes and the presence of multiple obesity-related comorbidities [2]. Gastric bypass was the preferred bariatric technique based on the patient's food pattern that consisted mainly of volume eating and sweet eating, the presence of impaired fasting glucose, patient's preference and surgeon's experience [3]. Biliopancreatic limb length was 60 cm (of which 30 cm of jejunum) and related to operative selection of a favorable vascular arcade for transection of the jejunum; alimentary limb length was 150 cm and chosen by the operating surgeon because of less bile reflux, possible better glycemic control and tendency to faster weight loss when compared to shorter limbs; common limb constituted the remaining 160 cm of small intestine. This bariatric operation is indeed similar to the original long-limb gastric bypass described by Brolin et al. in 1992 [4], who demonstrated superior weight loss without additional metabolic complications in a superobese population (BMI > 50 kg/m^2). Multiple recent randomized controlled trials confirm these findings in the short term in the superobese, but fail to demonstrate superiority of longer alimentary limbs in non-superobese patients (BMI < 50 kg/m^2) like our patient [5,6].

The effects of bariatric surgery on liver function remain controversial. Although multiple studies show improvement of liver function tests and histology in patients with pre-existing NAFLD following bariatric surgery [7,8], multiple cases of liver failure after bariatric surgery have been reported as outlined in Table 2 [9–16]. While the timing of liver failure after bariatric surgery is highly variable, almost all reported cases are characterized by the need of urgent liver transplantation to avoid fatal outcome. Liver failure has been recorded mainly after jejunoileal bypass (JIB), Scopinaro and, to a lesser extent, after distal gastric bypass surgery. All these procedures are characterized by significant malabsorption due to a relatively short common limb length. Interestingly, our patient underwent a long-limb gastric bypass with a common limb length of 160 cm, which is significantly longer than Scopinaro (50 cm) or

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