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Acute liver decompensation following bariatric surgery in patients without cirrhosis: clinical presentation, histological findings and management

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Introduction

Obesity is a growing problem with multiple complications, such as metabolic dysfunction-associated fatty liver disease (MAFLD). Bariatric surgery (BS) has shown efficacy in long-term weight loss achievement. Numerous reports have described MAFLD related cirrhosis at the time of BS, complicated by further liver decompensation, in particular in patients with ancient BS techniques. The exact mechanism is unknown. Sometimes, cirrhosis may be absent. Fatal outcome, liver transplantation and reversal surgery have been reported in those patients.

Aim

We wanted to describe the clinical characteristics, histological findings and clinical management of patients without cirrhosis who developed acute hepatic decompensation after BS for severe obesity.

Methods

We collected clinical, biological, histological and follow-up data from patients without cirrhosis with acute hepatic decompensation after BS.

Results

From December 2014 to October 2019, 6 patients (5 females and 1 male) underwent a transvenous liver biopsy for acute liver decompensation after BS. Four patients had a Roux-en-Y gastric bypass, one patient had a biliopancreatic diversion according to Scopinaro and one patient had a distal gastric bypass. At the time of BS, the mean age was 36 years (31-56), all patients were severely obese (mean body mass index 45 kg/m²) and there was no argument for cirrhosis (FIB-4 score < 1.3). The time between surgery and the onset of acute liver decompensation varied widely (min. 8 months, max. 17 years). Three patients consumed alcohol occasionally and only one patient had a chronic alcohol abuse. All patients described limited oral intake. The mean weight loss at the time of acute liver decompensation was 54.5 kg (31-76). The clinical presentation was as follows: fatigue and jaundice in 3 patients, edema of the lower limbs in 3 patients, ascites in one patient and altered coagulation in all patients. Blood tests showed an acute increase in transaminases (mean ALAT 47 UI/L, mean ASAT 81 UI/L), bilirubin (mean 3.75 mg/dL) and INR (mean 1.43) with a low albumin level (mean 24 mg/dL). The hepatic venous pressure gradient was high (mean 9.5 mmHg). Histology revealed severe steatosis (predominantly macrovesicular) in 5 patients and moderate steatosis in 1 patient. Hepatocyte ballooning was present in 4 patients. Mean fibrosis score was 2 (no patient with a F4 score). Interestingly, histological analysis also revealed cholangitis and bile duct alterations in all patients. There was no other cause of acute liver injury. All patients were treated with aggressive nutritional therapy (intravenous albumin supplements, parenteral and/or enteral nutrition, vitamins) as well as diuretics. The clinical course was favorable in all cases (mean time of hospitalization was 29 days), without the need for surgery or transplantation. All patients are still alive with a mean follow-up of 36 months.

Conclusions

Acute liver decompensation in the absence of cirrhosis can occur after bariatric surgery (with a highly variable delay). Hepatic injury is characterized histologically by a unique feature of steatoheptitis with bile duct alterations. Severe protein malnutrition and bacterial overgrowth are possible candidates for the development of this alarming complication. Substantial clinical improvement with appropriate refeeding seems to be effective.