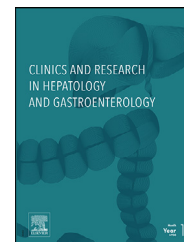




Available online at
ScienceDirect
 www.sciencedirect.com

Elsevier Masson France
EM|consulte
 www.em-consulte.com



LETTER TO THE EDITOR

Emergency reversal of a Roux-en-Y Gastric bypass to rescue liver graft from recurrent steatohepatitis



Introduction

Metabolic associated liver disease (MALD) constitutes a rapidly growing indication for liver transplantation. Bariatric surgery has been proven as an effective treatment of MALD by resolving in most of the patient steatohepatitis and fibrosis [1]. Nevertheless, the performance of bariatric surgery in some obese patients has been associated with impairment of steatohepatitis with progression toward liver failure [2,3]. Extreme protein malnutrition and bacterial overgrowth of the small bowel have been claimed as causes of aggravation of preexisting steatohepatitis following bariatric surgery [2]. While some patients might recover from liver failure under appropriate medical treatment (enteral nutrition, oral antibiotics, and albumin infusion), others progress toward liver failure that might require liver transplantation [2]. Herein we describe a recurrent steatohepatitis after LT in a patient with Roux-en-Y gastric-bypass (RYGBP) requiring emergency bariatric surgery reversal to rescue the transplanted liver.

Case observation

A 58-year-old woman was referred for LT evaluation for an acute liver failure on chronic metabolic and alcoholic liver disease. Her past medical history revealed a laparoscopic Roux-en-Y gastric bypass (RYGBP) four years previously with an alimentary limb of 100 cm. She had moderate alcohol consumption since then. Postoperatively she lost 50 kg with a body mass index reduction from 56.6 to 37.1 kg/m². Two years earlier, she had small intestinal bacterial overgrowth (SIBO) symptoms that resolved on oral antibiotics. Preoperative work-up revealed no contraindications for liver transplantation. She was listed with a model for End-Stage liver disease score of 40 and was transplanted. Following transplantation, she was treated with corticosteroids, tacrolimus, and mycophenolate mofetil. The explanted liver showed marked pre-cirrhotic fibrosis with intense steatosis. She was discharged in an outpatient clinic at postoperative day 25 with quasi-normal liver function test. She was readmitted

urgently ten days later because of diarrhea treated with oral antibiotics due to high suspicion of small intestinal bacterial overgrowth (SIBO). An abdominal CT-Scan revealed massive graft steatosis. A transjugular biopsy of the liver graft showed macrovacuolar steatosis (95%) with no fibrosis. Clinical conditions and biological test rapidly deteriorated despite enteral nutrition and oral antibiotics. Rapidly, she had bilirubin level of 350 μmol/L, an albumin of 26 g/L, an international normalized ration of 2.05. Reversal of RYGBP was then urgently performed by a side-to-side gastro-gastric anastomosis and Roux-en-Y limb dismantling with an end-to-end jejuno-jejunal anastomosis. A jejunostomy was placed for postoperative enteral nutrition. Liver biopsy showed macrovacuolar steatosis (95%) with inflammatory infiltrate and hepatocellular necrosis consistent with steatohepatitis. Postoperatively bilirubin decreased rapidly and normalized at postoperative day 42. Postoperative CT scan at day 10 showed marked regression of the steatosis (Fig. 1). The patient gave written consent form to publish this case.

Discussion

Acute steatohepatitis with liver failure has been reported as a cause of patient's death and an indication for liver transplant in patient undergoing bariatric surgery [2]. The mechanisms leading to liver failure after bariatric surgery in patients with hepatic steatosis remain poorly understood. However, in the two-hit theory for explaining steatohepatitis, it has been hypothesized that a fat rich diet could cause gut dysbiosis. The consequent changes in intestinal microbiome would be responsible of an increased production of lipopolysaccharides (second hit) which translocating into the portal system could activate Kupffer cells and hepatic stellate cells resulting in an inflammatory cascade activation [4]. The reasons for which only few patients with obesity will impair their liver steatosis while the majority would eventually heal their liver from MASH following bariatric surgery remains unclear. In a systematic review of the literature, we found that most cases of liver failure requiring LT followed highly malabsorptive procedures namely Scopinaro's biliopancreatic diversion and biliointestinal bypass [2]. Bacterial overgrowth with gut dysbiosis occurs probably more frequently in bariatric procedures with a long alimentary limb such as biliojejunal bypass, biliopancreatic diversion and one anastomosis gastric bypass. In the reported case, the

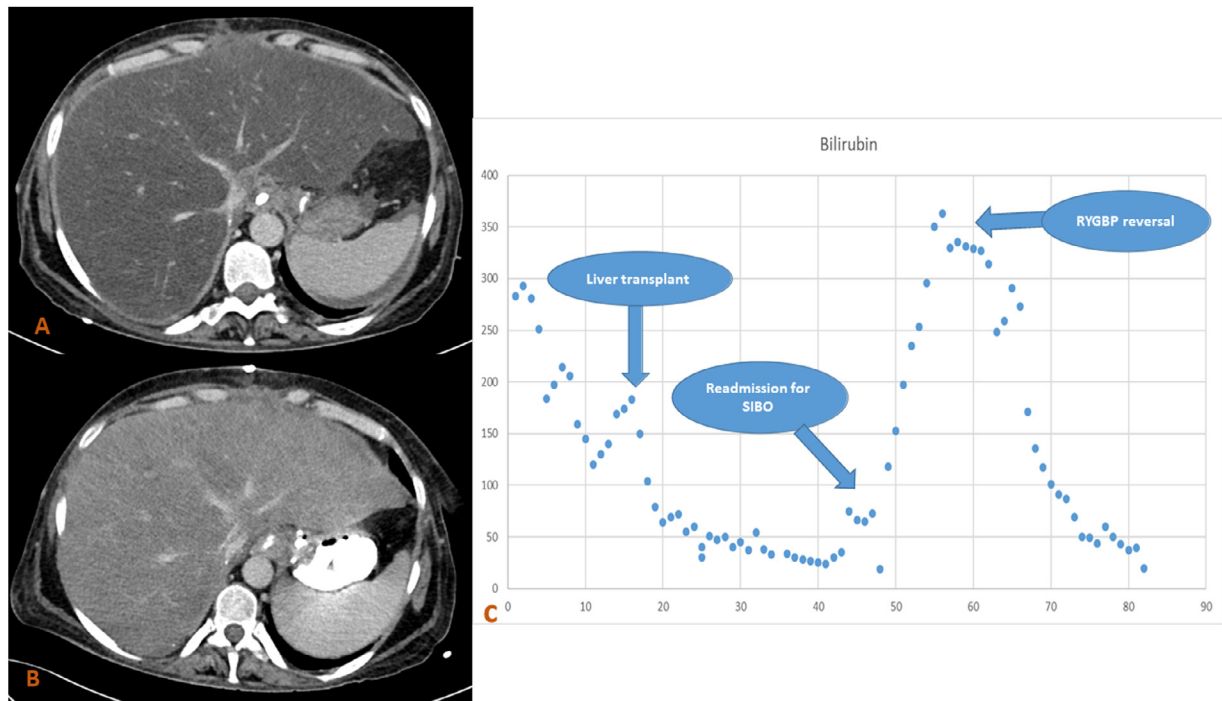


Fig. 1 Radiologic appearance of liver steatosis evolution before (A) and after RYGBP reversal (B). Bilirubin patient levels during the postoperative course (C), SIBO: small intestine bacterial overgrowth.

patient had a standard 100 cm alimentary limb, but she presented early recurrence of steatohepatitis after LT indicating that SIBO might occur independently from the length of the alimentary limb. Recurrent steatohepatitis has been reported also after LT especially in patients with not-reversed bariatric procedures as a potential cause of graft loss and death [3]. Reversal can be performed either during or earlier after LT [2]. We did not proceed to reverse RYGBP during LT because of the diagnosis of alcohol-related liver diseases and also because of not reverse BS either in our other patients having previous bariatric surgery.

As a final remark of this case observation and taking into account data from the literature [2] we could propose a close follow-up to patients undergoing LT who have a previous BS especially RYGBP. In these patients, the presence of bypassed bowel can be the origin of SIBO leading to recurrent steatohepatitis on the liver graft.

Conclusions

Small bowel bacterial overgrowth in patients with RYGBP might be the origin of recurrent steatohepatitis after liver transplantation. A close follow-up is indicated in patients with RYGBP after LT. Bariatric surgery reversal should be performed once diagnosis of recurrent steatohepatitis has been made to rescue the liver graft.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

References

- [1] Lassailly G, Caiazzo R, Ntandja-Wandji LC, et al. Bariatric surgery provides long-term resolution of nonalcoholic steatohepatitis and regression of fibrosis. *Gastroenterology* 2020;159:1290-301 e5.
- [2] Addeo P, Cesaretti M, Anty R, Iannelli A. Liver transplantation for bariatric surgery-related liver failure: a systematic review of a rare condition. *Surg Obes Relat Dis* 2019;15:1394-1401.
- [3] Lefere S, Hoorens A, Raevens S, et al. Refractory subacute steatohepatitis after biliopancreatic diversion. *Hepatology* 2017;66:289-91.
- [4] An L, Wirth U, Koch D, et al. The role of gut-derived lipopolysaccharides and the intestinal barrier in fatty liver diseases. *J Gastrointest Surg* 2021.

Pietro Addeo^{a,b,*}, Maxime Foguene^a,
Baptiste Michard^a, Thierry Artzner^a,
Mathilde Deridder^a, François Faitot^{a,b},
Lawrence Serfaty^c, Philippe Bachellier^a

^a Hepato-Pancreato-Biliary Surgery and
Liver transplantation, Pôle des Pathologies
Digestives, Hépato- et de la Transplantation,
Hôpital de Hautepierre-Hôpitaux
Universitaires de Strasbourg,
Université de Strasbourg, Strasbourg,
France

^b ICube, Université de Strasbourg,
CNRS UMR 7357, Illkirch, France

^c Hepatogastroenterology department,
Hôpital Hautepierre, Hôpitaux Universitaires de
Strasbourg, Strasbourg, France

* Corresponding author. Pietro Addeo,
Hepato-Pancreato-Biliary Surgery and Liver
transplantation, Pôle des Pathologies Digestives,
Hépatiques et de la Transplantation,
Hôpital de Hautepierre-Hôpitaux Universitaires de

Strasbourg, Université de Strasbourg, Strasbourg, France 1,
Avenue Moliere, 67098, Strasbourg.

E-mail address: pietrofrancesco.addeo@chru-strasbourg.fr
(P. Addeo).