Two rare cases of acute liver failure after gastric bypass

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Mini review

The prevalence of obesity in the world has increased drastically in the past decades. Currently, physicians manage obesity by lifestyle and dietary changes. If these treatment options fail, bariatric surgery becomes a possibility. The gastric bypass is currently one of the favored techniques, because of its positive long-term effects. Severe complications, including serious impaired liver function, were seen in the past after performing jejunoileal bypass (JIB) and biliopancreatic diversion (BDP). This led to the abandonment of these techniques. We would like to present two similar and interesting cases of acute liver failure associated with the gastric bypass procedure.

A 49-year-old woman was referred to our hepatology department in October 2017 for painless jaundice and a gradually increasing fatigue. The patient underwent gastric bypass surgery in January 2017 for severe obesity, with a body mass index (BMI) of 41.5 kg/m². The pre-operative evaluation of this patient revealed no signs of liver impairment and no post-operative complications were noted. In addition, she experienced a rapid weight reduction of 30 kg. Upon admission, 9 months after surgery, physical examination revealed jaundice, flapping tremor and an increasing fatigue. Laboratory results revealed: AST 1860 U/l (normal<35 U/l); ALT 693 U/l (normal<35 U/l); LDH 667 U/l (normal<247 U/l); GGT 492 U/l (normal<38 U/l); total bilirubin 21.54 mg/dl (normal 0.30-1.20 mg/dl); direct bilirubin 5.68 (normal<0.20 mg/dl); albumin 28.0 g/l (normal 35.0-52.0 g/l); INR 1.92 (normal 1-1.16); creatine 1.04 mg/dl (normal 0.51-0.95 mg/dl); platelets 202 x10³/μl (normal 150-400 x10³/μl). Serum markers for viral hepatitis and autoimmune related parameters were negative. No acetaminophen abuse or recent alcohol abuse was present. The patient was placed on the transplantation list after progressive coagulopathy and limited steatosis (Figure 3). Signs of cirrhosis were not seen. This pathological signatures of multilobular necrotising hepatocytes leave the differential diagnosis: (endo-)toxic process, autoimmune hepatitis and a viral induced hepatitis.

A second and similar case was presented two years ago. A 38-year-old woman was referred to the department in December 2015. This patient also underwent gastric bypass surgery in April 2015 (BMI: 46.5 kg/m²), which induced a weight loss of 62 kg without any post-operative complications throughout the first months. Mid-November, the patient started to develop a subacute cholestatic hepatitis. Laboratory results revealed: AST 449 U/l; ALT 505 U/l; GGT 118 U/l; total bilirubin 21.13 mg/dl; direct bilirubin 16.22; albumin 20.0 g/l; INR 3.9; platelet 155 x10³/μl. Extensive clinical and biochemical evaluations were not able to suggest a clear origin of this liver failure. The patient deteriorated during the observation period. This led to an increase of INR and the development of an overt encephalopathy in addition to an already long-standing jaundice, which caused the necessity of a lifesaving liver transplantation.

Interestingly, both young women showed a similar progression towards (sub)acute liver failure several months after their gastric bypass. They both received a liver transplantation after their acute onset of liver impairment. We examined both explanted livers macro- and microscopically and found similar histopathological findings (Figure 1). An extensive multilobular necrosis was present, which destroyed completely the liver architecture. Furthermore, a mild inflammatory infiltrate consisting of mainly lymphocytes, some plasma cells and some eosinophils was notable. Parenchymal and ductular bilirubinostasis were present, these are clear signs of parenchymal decompensation and sepsis (Figure 2). The few surviving areas of hepatocytes showed ballooning in presence of Mallory-Denk bodies and limited steatosis (Figure 3). Signs of cirrhosis were not seen. This pathological signatures of multilobular necrotising hepatocytes leave the differential diagnosis: (endo-)toxic process, autoimmune hepatitis and a viral induced hepatitis.

Figure 1 Macroscopic evaluation of the explanted liver shows diffuse red zones represents the massive multilobular necrosis in the presence of some tan nodular zones which contain the remaining hepatocytes.

Remarkable about these two cases is the onset of acute liver failure several months after gastric bypass with a drastic decrease in weight. A definitive cause of the liver failure remains unclear until today. Nevertheless, since both women experienced a very similar clinical course after bariatric surgery and both explanted livers had similar histological features, we can propose a narrowed differential diagnosis. At first, we have to consider an (endo-)toxic process, which could be induced by bacterial overgrowth and/or bile acid metabolites, as seen in previous JIB. The increased gut permeability and the release of

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Toxic components lead up to an increased release of proinflammatory cytokines into the circulatory system and consequently into the liver. All these changes could have the capability to induce liver failure in these patients. Certainly in patients who have other comorbidities, as is often seen in obese patients. Another potential mechanism could be explained by the oxidative stress in the liver due to rapid weight loss/malnutrition.\(^6,7\) It is possible to propose that the liver impairment is triggered by a combinatory effect of the two mechanisms since both are related to the surgical disruption of the normal gastrointestinal tract. Strictly speaking, the differential diagnosis of autoimmune hepatitis and viral hepatitis should be made, however, negative serology and the relatively mild inflammatory infiltrate make these possibilities unlikely. These cases underline the importance of post-operative follow-up of patients after gastric bypass surgery. Therefore, we stress to be attentive for liver impairment, since early detection and referral of these cases can be lifesaving.

**Figure 2** The explanted livers show multilobular necrosis, associated with an extensive ductular reaction with bilirubinostasis inside some bile ductules (ductular bilirubinostasis: arrowheads). (Haematoxylin and Eosin, x50 and x200).

**Figure 3** Both cases show few remaining hepatocytic nodules, which are surrounded by multilobular necrosis and a mild inflammatory infiltrate dominated by lymphocytes (arrows: portal tract; arrowheads: central veins). (Sirius red, x50).

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**Conflict of interest**

The authors declare no conflict of interest.

**References**