Cirrhosis following single anastomosis duodeno-ileal switch: A case report

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A B S T R A C T

INTRODUCTION: Single anastomosis duodeno-ileal switch (SADI-S) involves a single, loop duodeno-ileal anastomosis with omission of the traditional Roux-en-Y distal ileo-ileal anastomosis. Not much has been published on the complications of SADI-S.

PRESENTATION OF CASE: The patient is a 40 year-old male who underwent robot-assisted SADI-S three years prior. At the time of surgery, his body mass index (BMI) was $69 \, kg/m^2$. His BMI was $31 \, kg/m^2$ at the time of presentation. Computed tomography of the abdomen and pelvis showed a liver with a nodular appearance and moderate ascites throughout the abdomen and above the liver. Liver function showed AST 73 U/L, ALT 63 U/L, alkaline phosphatase 128 U/L, bilirubin 1.0 mg/dL, and albumin 2.8 g/dL. He underwent diagnostic and therapeutic paracentesis with removal of 6L of fluid that was negative for infection or malignant cells. Esophagogastroduodenoscopy showed grade I esophageal varices and diffuse mild gastritis.

DISCUSSION: Mild hepatic dysfunction is common after biliopancreatic diversion and usually manifests as a temporary rise in liver enzymes that peak several months after surgery and normalize by one year. Cirrhosis and liver failure are rare sequelae that may develop at any time after surgery. Potential mechanisms include the absorption of hepatotoxic compounds from the bypassed small intestine in the context of bacterial overgrowth, protein malnutrition, and excessive free fatty acid mobilization causing steatosis and oxidative damage to hepatocytes.

CONCLUSION: Close laboratory monitoring is important after SADI-S in order to detect worsening hepatic dysfunction, which may occur many years after the surgery in the absence of other etiologies for liver failure.

1. Introduction

Biliopancreatic diversion for weight loss was developed in the 1970's by Scopinaro et al. [1] and originally consisted of a subtotal horizontal gastrectomy with anastomosis to a 250 cm Roux limb and a 50 cm common channel. A subsequent variant, described by Hess and Hess [2], involved sleeve gastrectomy with preservation of the pylorus, a duodenoileostomy and a common channel of 100 cm. A modern adaptation has been the single anastomosis duodeno-ileal switch (SADI-S), which involves a single, loop duodeno-ileal anastomosis with omission of the traditional Roux-en-Y distal ileo-ileal anastomosis. Biliopancreatic diversion results in the greatest magnitude of weight loss that is, moreover, the most sustained when compared to other contemporary bariatric surgical procedures [3–6]. The technical difficulty of the procedure as well as concerns over greater malabsorption [7–9], steatorrhea and cirrho-

sis or liver failure [10–16] have limited widespread adoption. We present a case of cirrhosis 3 years after SADI-S, in accordance with the SCARE criteria [17].

2. Case report

The patient is a 40 year-old Latino male who underwent robot-assisted biliopancreatic diversion with duodenal switch with a single loop anastomosis (single anastomosis duodeno-ileal switch) three years prior. At the time of surgery, his body mass index (BMI) was $69 \, kg/m^2$. He weighed 462 pounds and his other comorbidities included obstructive sleep apnea, generalized anxiety disorder and mild gastroesophageal reflux disease controlled with medication. He was a former smoker and he reported one alcoholic beverage per month. His preoperative liver function and kidney function were within normal limits and an ultrasound of his liver and gallbladder showed no gallstones with some fatty infiltration of the liver. During the operation, there was no ascites and his liver was noted to be large but not nodular or cirrhotic. His postoperative course was uneventful and he was discharged home four days after surgery.

The patient followed up in clinic after surgery for his one week visit, then at one month and three months. However, he was then lost to follow up until three years later. At his three-year clinic visit he reported that his primary physician had discovered elevated liver enzymes on a routine lab check and had ordered an ultrasound that revealed fatty liver.

Five months later, the patient presented to the emergency department with abdominal pain and bilateral lower extremity swelling. He reported that he did not drink any alcohol. He denied having any diarrhea. His weight was 209 pounds for a loss of 253 pounds from his preoperative weight (83% of excess weight loss), and his BMI was 31 kg/m^2. Computed tomography of the abdomen and pelvis showed a liver with a nodular appearance and moderate ascites throughout the abdomen and above the liver. Liver function showed AST 73 U/L (normal 13–39 U/L), ALT 63 U/L (normal 7–52 U/L), alkaline phosphatase 128 U/L (normal 34–104 U/L), bilirubin 1.0 mg/dL (normal 0.3–1.0 mg/dL), and albumin 2.8 g/dL (normal 3.5–5.7 g/dL). Hepatitis serology was negative. Coagulation labs and kidney function were normal.

Ultrasound of the lower extremities was negative for deep venous thrombosis. Echocardiography showed normal ejection fraction with no valvular pathology. He underwent diagnostic and therapeutic paracentesis with removal of 6 L of fluid that was negative for infection or malignant cells. Esophagogastroduodenoscopy showed grade I esophageal varices and diffuse mild gastritis that was negative for H. pylori on biopsy.

3. Discussion

Most patients exhibit an improvement in hepatic steatosis and steatohepatitis after biliopancreatic diversion. Aldoheyan et al. [18] performed liver biopsies at the time of bariatric surgery and at three months postoperatively and found statistically significant reductions in hepatic steatosis and fibrosis scores as well as non-alcoholic fatty liver disease activity scores. Similarly, Weiner et al. [19] reported complete regression of nonalcoholic fatty liver disease by liver biopsy in 83% of patients who underwent either gastric bypass, gastric banding or biliopancreatic diversion. In this series, of twelve patients who had fibrosis on their intraoperative biopsy, ten had complete resolution of the fibrosis and two exhibited improvement on follow up biopsy.

A mild degree of subclinical hepatic injury consisting of an asymptomatic elevation of liver enzymes was reported in a series of 697 patients who underwent open biliopancreatic diversion [20]. In this study they noted a mild increase in liver transaminases at 6 months postoperatively that returned to baseline by 12 months postoperatively. In the 78 patients who had repeat liver biopsy in addition to the routine biopsy performed at the time of the original biliopancreatic diversion, they reported a statistically significant reduction in the percentage of steatosis and the nonalcoholic steatohepatitis (NASH) grade. There were no reported cases of liver cirrhosis or failure. This pattern of transient elevation of liver function tests in the several months after surgery followed by normalization of lab values by one year was also reported in another series of 99 patients who underwent biliopancreatic diversion [21].

Progression to cirrhosis and liver failure is rare. The frequency of severe liver dysfunction and liver failure in a series of 929 patients who underwent biliopancreatic diversion was reported as 1.2% and 0.2%, respectively [10]. A Spanish study of 470 patients who underwent biliopancreatic diversion reported 10 cases of postoperative hepatic dysfunction that were not due to viral hepatitis or biliary ductal disease [11]. There are numerous case reports of liver failure after biliopancreatic diversion often requiring liver transplantation [12–16]. To our knowledge, this is the first report of cirrhosis after single anastomosis biliopancreatic diversion. Of note the time to

development of cirrhosis and liver failure ranges from a few months after surgery to up to 14 years postoperatively, so life-long monitoring of hepatic function after biliopancreatic diversion is important [14].

The development of hepatocyte damage after biliopancreatic diversion has been speculated to involve several possible mechanisms. The bypassed intestine may develop bacterial overgrowth, which leads to the absorption of toxic metabolites and endotoxin that cause damage to hepatocytes. This process was reversed by administration of the antibiotic metronidazole in patients after jejunoileal bypass [22]. Bacterial overgrowth has been reported after biliopancreatic diversion although the frequency is unclear [23].

Protein malnutrition may also contribute to hepatic steatosis and dysfunction. Severe steatosis and elevated liver enzymes after jejunoileal bypass were reversed after infusion of amino acids in one series [24].

Another proposed mechanism involves the mobilization of free fatty acids from adipose tissue, which, during periods of rapid weight loss may exceed the capacity of the hepatocytes to metabolize resulting in steatosis and oxidative damage [21,25]. This process is not unique to biliopancreatic diversion as it may occur in the context of rapid weight loss by diet or jejunoileal bypass [26].

4. Conclusion

Mild hepatic dysfunction is common after biliopancreatic diversion and usually manifests as a temporary rise in liver enzymes that peak several months after surgery and normalize by one year. Cirrhosis and liver failure are rare sequelae that may develop at any time from several weeks after surgery up to 14 years later. Potential mechanisms include the absorption of hepatotoxic compounds from the bypassed small intestine in the context of bacterial overgrowth, protein malnutrition, and excessive free fatty acid mobilization causing steatosis and oxidative damage to hepatocytes. Caution should be exercised in selecting biliopancreatic diversion as a bariatric procedure for patients with a known history of cirrhosis. The discovery of cirrhosis intraoperatively should prompt consideration for changing the surgical procedure, such as to a sleeve gastrectomy. Close laboratory monitoring is important after biliopancreatic diversion in order to detect worsening hepatic dysfunction, which may occur many years after the surgery in the absence of other etiologies for liver failure. In patients with cirrhosis and liver failure who do not respond to supportive care with amino acid infusions and nutritional supplementation consideration should be made for surgical intervention to ameliorate the malabsorption such as lengthening the common channel or creation of a side-to-side anastomosis between the proximal biliary limb and the alimentary limb.

Conflicts of interest

Drs. Jawad and Teixeira are consultants of Ethicon-Endosurgery and received consultant fees.

Dr. Kirkpatrick and Dr. Moon do not have any conflict of interest to report.

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Vincent Kirkpatrick – data collection, interpretation, writing the paper.

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