INTRODUCTION

Haemobilia, bleeding into the biliary tree, is an unusual cause of obscure upper gastrointestinal bleeding after laparoscopic cholecystectomy (LC) with reported incidence of 0.3-1.0%.1 Hepatic artery pseudoaneurysm is a rare complication of LC resulting in haemobilia. Hepatic artery variants are quite common and are present in approximately 42% of individuals.2 Michel et al. first described a basic classification system for hepatic artery variants, the most common being accessory right hepatic artery arising from the superior mesenteric artery (SMA) and accessory left hepatic artery arising from the left gastric artery.2

Pseudoaneurysm of the accessory/aberrant hepatic artery is extremely rare. It can rupture into the biliary tract, and most of the patients present in emergency with severe upper abdominal pain, jaundice, gastrointestinal bleeding and shock. In few patients, presentation might be delayed for months after LC.3 The diagnosis can be made via contrast-enhanced CT, ultrasound, magnetic resonance imaging, arteriography and endoscopic retrograde cholangiopancreatography (ERCP).4 The last also helps in relieving biliary obstruction caused by blood clots. In recent years trans-arterial embolization (TAE) has been recognized as first line treatment with higher success rate of around 80-100%.5 Ligation of bleeding vessels or excision of aneurysms surgically can be opted, if embolization fails.5

To our knowledge we report first case of accessory hepatic artery pseudoaneurysm following LC presenting as haemobilia.

CASE REPORT

A 60 years old male presented with history of haematemesis, haematochezia and severe epigastric pain. His blood count showed low haemoglobin of 4.5 g/dl with normal total leukocyte, platelet count and renal function test. Liver function tests found serum total bilirubin of 2.2 mg/dl, ALT at 60 (normal 0-55) IU/L and alkaline phosphatase (ALP) at 155 (normal 30-117) IU/L. No other co-morbidity was identified. He had previous history of LC for gallbladder stones 4 months earlier in another hospital after which he recovered. After resuscitation, gastroscopy was done which revealed haemobilia as blood trickling from swollen ampulla (Figure 1); no other abnormality was seen on endoscopy. He was shifted to radiology suite immediately for CT scan of upper abdomen which revealed a soft tissue enhancing structure just adjacent to common bile duct (CBD) (Figure 2). It was diagnosed as pseudoaneurysm arising from accessory hepatic artery. As the patient was bleeding profusely without clinical evidence of cholangitis or any biliary obstruction evident on CT scan, so it was decided to proceed immediately for TAE.

Celiac and mesenteric angiogram were carried out which revealed accessory hepatic artery arising from SMA with a pseudoaneurysm measuring 3.5 x 1.5 cm (Figure 3). There was an associated arteriovenous fistula (Figure 4). The accessory branch was successfully embolized by placing 4 fibered platinum coils proximal and distal to the pseudoaneurysm. Bleeding stopped and the patient recovered uneventfully. He was followed in clinic for 6 months and remained well.
DISCUSSION

Hepatic artery pseudoaneurysm is a rare complication of LC, but haemobilia with accessory hepatic artery has not been reported in literature previously. Haemorrhage arising from hepatic artery injury can be life-threatening with mortality rate as high as 50%, and necessitates immediate haemostatic intervention. Hepatic artery pseudoaneurysms may be asymptomatic or may present with intermittent upper abdominal pain, acute pain due to spontaneous rupture, jaundice due to biliary obstruction, haematemesis or melena.

Blunt abdominal trauma is the leading cause of haemobilia; other less common causes are liver and biliary interventions and T-tube choledochostomy. Non-traumatic or spontaneous hepatic artery pseudoaneurysm although rare, but are reported. Exact pathogenesis of post LC hepatic artery pseudoaneurysm is unclear. Various hypotheses are direct vascular injury, erosion due to clip encroachment, bile leak and secondary infection. As this patient was operated elsewhere and was without availability of operative notes, got discharged from hospital in 3 days and remained well till this episode of gastrointestinal bleeding. Exact cause of pseudoaneurysm in this patient is difficult to explain but in the absence of any other etiology LC was presumed as the likely cause.

Therapeutic options include TAE as a first line minimally invasive option, for massive haemobilia but operative ligation of the hepatic artery and excision of the aneurysm with revascularization or bypass can be considered if TAE is not available or fails to achieve haemostasis. Percutaneous injection of Histoacryl directly into aneurysm has also been reported, if TAE fails. ERCP can play a key role in diagnosis of hemobilia and management of cholangitis and biliary obstruction due to blood clots. This patient was bleeding profusely and did not have any evidence of cholangitis or biliary obstruction, so we decided to proceed immediately for TAE to achieve haemostasis.

For prevention of recurrent haemorrhage due to refilling of the residual lumen of vascular lesion, we embolized the accessory hepatic artery first distally then proximal to the pseudoaneurysm by placing four pushable fibered coils.
REFERENCES


