

# Iatrogenic Post Biopsy Hemobilia with Pseudoaneurysm

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## ABSTRACT

Hemobilia is defined as bleeding into the biliary tree from an abnormal communication between a blood vessel and bile duct. It is an uncommon cause of upper gastrointestinal hemorrhage and iatrogenic most of the times. We report a case of hemobilia secondary to percutaneous liver biopsy presenting with classical Quincke's triad in a young lady which was treated with combined biliary balloon sweep thrombectomy and transarterial embolization for complete resolution of symptoms.

**Key Words:** Hemobilia. Pseudoaneurysm. ERCP. Embolization. Liver biopsy. Quincke's triad.

## INTRODUCTION

Hemobilia is one of the rarest causes of upper GI (Gastrointestinal) bleeding and results from an abnormal communication between the biliary tract and hepatic vessels. The etiology is variable and includes infections, cholelithiasis, hepatic tumors, ruptured hepatic cysts, arteriovenous malformations, aneurysms and pseudoaneurysm. More often the cause is an iatrogenic injury secondary to cholecystectomy or liver biopsy.<sup>1-3</sup> Clinical presentations include upper gastrointestinal bleeding, biliary colic and/ or jaundice. The diagnosis is suggested in those presenting with history of hepatic surgery, trauma or invasive biliary intervention. The key investigation is a selective angiogram which is used to diagnose, locate and in most cases treat the culprit vascular lesion.<sup>2-4</sup>

We report the case of a young lady with obstructive jaundice secondary to iatrogenic hemobilia who partially recovered after ERCP (Endoscopic Retrograde Cholangiopancreatography) and sphincterotomy with clot extraction until embolization of pseudoaneurysm had to be done for complete resolution of symptoms.

## CASE REPORT

A 27 years old lady presented with acute onset of biliary colic associated with pain in the right shoulder, nausea, vomiting, pruritus and melena following a routine liver biopsy 5 days earlier. Her general physical examination was unremarkable apart from moderately severe jaundice. There was no tenderness at biopsy site. Abdomen was soft with mild epigastric tenderness without guarding. Rest of the examination was unremark-

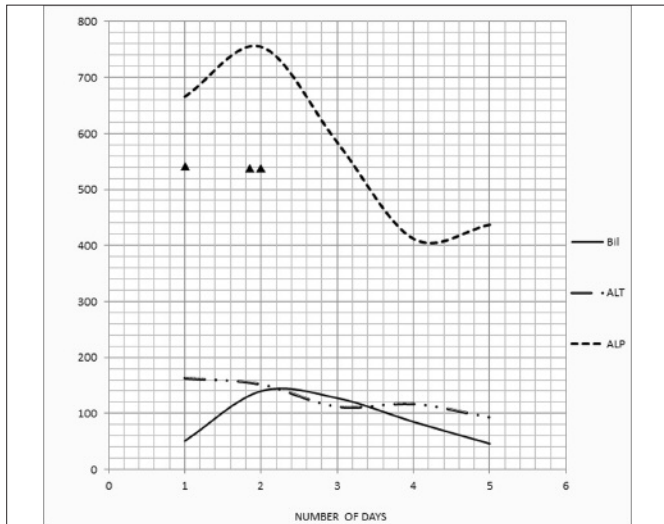
able. Her baseline LFT's (Liver Function Tests) showed a cholestatic pattern with total bilirubin of 51.3  $\mu\text{mol/L}$  and conjugated bilirubin of 25.65  $\mu\text{mol/L}$ . Her ALT (alanine aminotransferase) was 163 U/L, alkaline phosphatase 666 U/L, hemoglobin (Hb) 107 g/L, leukocyte count  $10.5 \times 10^9/\text{L}$  and platelet count  $272 \times 10^9/\text{L}$ . She had normal urea, creatinine and serum electrolyte levels with a normal coagulation profile. Her ultrasound abdomen revealed a distended gallbladder with sludge and intrahepatic cholestasis with mild dilatation of CBD (Common Bile Duct). An admitting diagnosis of biliary colic secondary to sludge was made and patient was managed with intravenous antibiotics, fluids, antispasmodics and analgesics.

She continued to have intermittent biliary colic over the next 3 - 4 days with a serial worsening in the severity of jaundice and rising bilirubin levels. A repeat ultrasound delineated an organized sludge ball in gallbladder with thick walls and filling defects in the common bile duct with suspicion of hemobilia. This was confirmed on abdominal Computed Tomography (CT) scan. Subsequent upper GI endoscopy showed a clot at the ampulla of Vater. Sphincterotomy was done and multiple clots were removed from the CBD using balloon sweep thrombectomy technique. A post-ERCP occlusion cholangiogram evinced normal CBD and intrahepatic ducts. Serial LFT's showed declining enzyme levels and serial ultrasounds depicted significant interval improvement with decreasing distension of gallbladder and absence of cholestasis. On the 5th post-procedure day, she had a bilirubin of 30.78  $\mu\text{mol/L}$ , ALT of 49 U/L, alkaline phosphatase of 411 U/L and Hb of 74 g/L (Figure 1). The same day, she developed hematemesis of more than 0.15 L. She had mild tachycardia with a pulse of 102/minute and B.P. of 90/50 mmHg (in SI units = 11999/ 6666 Pascal; conversion factor = 133.322). The patient was given rapid Ringer's lactate infusion for resuscitation. A dynamic CT scan was planned to evaluate the cause of hemobilia. CT revealed presence of an approximately 5 mm (in SI units = 0.005 m;

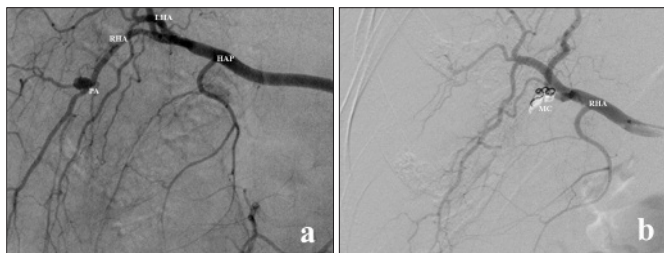
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**Figure 1:** Serial liver function tests - timeline. Serial Liver Function Tests of patient during the course of ailment represented in number of days. The graph clearly shows a worsening profile following liver biopsy. After ERCP, enzyme levels returned towards baseline. Legend: Bil = Bilirubin (normal = 3.4 - 17.1 μmol/ L), ALT = Alanine aminotransferase (normal = 65-306 U/ L), ALP = Alkaline phosphatase (normal = F: < 36 U/ L), ▲ = Point in time specifying liver biopsy, ▲▲ = Point in time specifying ERCP and sphincterotomy. Day 1 represents 5th day post-biopsy with already elevated enzyme levels at presentation.



**Figure 2 (a,b):** Pre and post-embolization CT angiogram of hepatobiliary vessels. (a) Pre-embolization CT angiogram of hepatobiliary vessels showing the presence of a pseudo aneurysm in the branch of RHA. The size of this aneurysm was approximately 5 mm along its long axis. (b) Post-embolization CT angiogram showing the site of placement of Guglielmi detachable metallic coil (MC) and lack of post-contrast enhancement of pseudoaneurysm. Legend: HAP = Hepatic Artery Proper, LHA = Left Hepatic Artery, RHA = Right Hepatic Artery, PA = Pseudo aneurysm, MC = Metallic coil, CT = Computed tomography.

conversion factor = 1000) nodular lesion in the branch of right hepatic artery. The nodular lesion showed no obvious extravasation of contrast into surrounding structures suggesting the presence of newly developed pseudoaneurysm in branch of right hepatic artery (Figure 2a).

After adequate resuscitation, the patient was scheduled for Transarterial Embolization (TAE). A celiac angiogram was performed and the affected branch was identified in segment V of liver. The branch was selectively embolized with polyvinyl alcohol instilled distally and a 3 mm (in SI units = 0.003 m; conversion factor = 1000) metallic Guglielmi detachable coil placed in its proximal part. A subsequent angiogram revealed loss of filling and complete occlusion of the pseudoaneurysm (Figure 2b).

She was transfused with 4 UK pints (in SI units = 0.00002272 m<sup>3</sup>; conversion factor: 1 UK pint = 568 ml; 1ml = 1/1000000000 m<sup>3</sup>) of red cell concentrate at Hb of 79 g/ L. The patient improved clinically and subsequently made an uneventful recovery with normal LFT's, Hb and good health on discharge.

**DISCUSSION**

Serious complications of liver biopsy are rare but potentially lethal. A thorough understanding of early diagnosis and prompt treatment is, therefore, imperative for better patient outcomes. Hemobilia is one of these rare presentations.<sup>1-3</sup> Among the various causes of hemobilia, iatrogenic liver biopsy stands on the top and is observed in one in a thousand cases (hemorrhage: intraperitoneal = 0.03 - 0.7%, intrahepatic and/or subcapsular = 0.05 - 23%, hemobilia = 0.05 - 0.2%).<sup>2-4</sup>

Hemorrhage in biliary tract was first identified by Francis Glisson in 1654, describing a young man dying from a massive upper GI bleed caused by a sword injury to the liver. Vascular lesions such as aneurysms of hepatic and cystic artery, vasculitis of cystic artery or portal vein tend to cause hemobilia that can be life-threatening.<sup>2-5</sup> The term "hemobilia" was first used by Sandblom in 1948.<sup>3,4,6,7</sup> The classic "hemobilia triad" described by Quincke in 1871 includes upper GI bleeding (hematemesis in 60% of cases and/or melena in 90%), biliary colic in 70% of cases and obstructive jaundice in 60%. Only 22% of patients present with all three symptoms.<sup>3-5,7</sup> Acute biliary symptoms and signs combined with acute upper gastrointestinal bleeding should strongly suggest the possibility of hemobilia. Obstructive jaundice can be caused by an obstructive biliary lesion or by blood clot. The latter is formed because bile and blood do not mix together.<sup>3,4,7</sup> A prompt upper GI endoscopy can be used to observe an active bleed or blood clot at the ampulla of Vater in such cases.<sup>4,5,7,8</sup>

The following features on abdominal ultrasound may be suggestive of hemobilia:<sup>3-5,8</sup> a blood-containing material in the bile ducts that is echogenic, non-shadowing and can merge with liver parenchyma, transitions from hyper- to hypo-echoic material in the bile ducts and blood at the walls of the gallbladder that can mimic acute cholecystitis.

A CT scan allows imaging of individual blood vessels but it is less sensitive in delineating dilatation of the bile ducts.<sup>3,4,8</sup> Hemobilia due to pseudoaneurysm can be detected on endoscopic ultrasound.<sup>4,5,7,8</sup> However, abdominal angiography is the gold standard for diagnosing hemobilia with an approximate 90% likelihood of finding the bleeding site.<sup>5,7,8</sup>

The main treatment goals are to first achieve hemostasis followed by removal of the biliary obstruction. ERCP with sphincterotomy can be done to relieve biliary obstruction.<sup>5-8</sup> If a persistent bleeding vessel is identified, the

success rate of angiography with selective embolization is almost 90%. Other management options include coagulation of the bleeding vessel, surgery or observation.<sup>4-8</sup>

Hemobilia from a hepatic artery source frequently requires TAE via a right femoral artery approach or even hepatic artery ligation. Arterial embolization can be a simple, safe and effective treatment for hemobilia with less morbidity and mortality than surgery.<sup>6-8</sup>

Surgery is considered if the cause of hemobilia is surgical, i.e., cholelithiasis, hemorrhagic cholecystitis, or neoplasms, angiography with embolization fails, or ERCP fails to relieve obstruction. Irrespective of cause, the success rate of therapy depends upon a timely diagnosis and prompt treatment.<sup>4-8</sup>

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