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Hemobilia: A Rare Cause of Upper Gasrointestinal Bleeding

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Abstract

Hemobilia, the presence of blood within the biliary tract, is a rare but significant cause of upper Gastrointestinal (GI) bleeding. It can result from various etiologies, including trauma, hepatic procedures, bile duct malignancies, or spontaneous rupture of blood vessels in the biliary system. The clinical presentation often includes hematemesis, melena, or signs of shock, which can mimic other causes of upper GI bleeding, making diagnosis challenging. This condition may be suspected in patients with a history of liver disease, recent abdominal trauma, or invasive biliary procedures. Diagnosis is typically confirmed through imaging techniques such as Computed Tomography (CT) scan, Magnetic Resonance Cholangiopancreatography (MRCP), or Endoscopic Retrograde Cholangiopancreatography (ERCP). Treatment options vary depending on the underlying cause and severity of bleeding and may include supportive care, endoscopic complications. This review discusses the pathophysiology, diagnostic approach, and therapeutic strategies for hemobilia, emphasizing the importance of considering it in the differential diagnosis of unexplained upper GI bleeding. By raising awareness of this rare condition, we aim to improve timely diagnosis and clinical outcomes for affected patients.

Keywords: Hemobilia • Gastrointestinal bleeding • Biliary tract

Introduction

Hemobilia is one of the rarest causes of upper gastrointestinal bleeding. Hemobilia is defined as bleeding from the hepatobiliary tract due to communication between the splanchnic circulation and the biliary system. The classic triad of symptom of hemobilia right upper quadrant pain, jaundice and hematemesis. More than 50% of cases would have experienced liver trauma, undergone a liver biopsy or manipulation of the hepatobiliary system (Endoscopic retrograde cholangiopancreatography, percutaneous transhepatic cholangiography or TIPSS), or have hepatocellular carcinoma or a biliary parasitic infection. Diagnosis is usually confirmed by duodenoscope to identify bleeding from the ampulla. Treatment is usually arterial embolization *via* arteriography.

Case Presentation

We present a case of 43 years old male admitted at Rajiv Gandhi Government General Hospital, Chennai who is already a known case of ethanol related decompensated chronic liver disease with portal hypertension. The patient was admitted with abdominal pain, abdominal distension, yellowish discoloration of eyes and urine present over a period of 10 days and passing dark colored stools for 3 days

Patient had abdominal pain present for over a period of 10 days in the right upper abdominal region, mild to moderate in intensity, dull aching, not related to food intake and not radiating. Abdominal distension was present for over a period of 10 days, generalized distension insidious in onset and progressive in nature. Yellowish discoloration of eyes presents for over a period of 10 days, progressive and associated with itching. He was passing dark colored stools over a period of 3 days before admission. There was no fever.

Patient was a known case of type 2 diabetes mellitus for the past 10 years. Before four months he was diagnosed with ethanol related decompensated chronic liver disease and he was on conservative management. During that admission he was diagnosed with calculous cholecystitis with choledocholithiasis and obstructive Jaundice. He also developed acute biliary pancreatitis and was

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managed conservatively. Later he underwent ERCP and CBD stenting for choledocholithiasis on the 3rd day.

Patient is on mixed diet. His sleep pattern was normal. He was a chronic smoker and chronic alcoholic for the past 15 years which he stopped 5 months ago.

On admission patient was conscious, oriented, afebrile, pallor and Icterus was present. Vitals was stable on admission. Abdomen was soft, mildy distended, mild tenderness was present in the right hypochondrial region. Grade 2 ascites was present. Bowel sounds was present.

His hemoglobin was 6.5, total WBC count-7200, platelet count-99,000 and his liver function test showed, total bilirubin-4.2, direct bilirubin-2.9, SGOT-29, SGPT-13, alkaline phosphatase-165, total Protein-6.9, serum albumin-3.3, serum globulin-3.6, INR-2.14.

Upper GI scopy showed Small esophageal varices with severe portal hypertensive gastropathy. CBD stent was visualized *in situ* and mild oozing of blood from stent was seen.

CT abdominal angiogram was done and no significant abnormality was detected. Our patient was given three units of packed red blood cells. Patient improved symptomatically and was feeling better. He didn't have melena and was planned for ERCP and stent exchange.

Uppper GI scopy was done three days later, ampulla was seen and stent in CBD was visualized. Oozing of blood from ampulla had stopped spontaneously. No further oozing of blood was seen.

After 6 days patient again had melena and a drastic drop in Hemoglobin from 8.1 to 5.5 in 24 hours. Emergency upper GI scopy was done which showed small oesophageal varices and portal hypertensive gastropathy. Blood clots were seen in the ampulla and CBD stent was seen *in situ* (Figure 1).



Figure 1. The above pic (Digital subtraction angiography) arrow points the hepatic artery pseudoaneurysm which is next to CBD stent.

Further 2 units of packed red blood a cell was given and with the help of intervention radiology, patient was taken up for digital subtraction angiography. Celiac angiography showed normal splenic artery and aneurysm in the proper hepatic artery which measured 5×6.1 mm. On identifying this aneurysm was super selectively catheterized

with mini catheter. Two coils were deployed into the aneurysm. Post procedure check angiogram showed occlusion of the aneurysm.

Post procedure patient improved symptomatically and was discharged after correction of anemia, with further plans of ERCP, CBD stent exchange and cholecystectomy.

Discussion

The classical triad of hemobilia symptoms, as described by Sandblom, are upper GI bleeding, right upper quadrant pain and obstructive jaundice [1]. It is very difficult to see patients with all three classical features. In our patient, he presented with Right upper quadrant pain and melena at initial presentation and went on to have obstructive jaundice in the later course.

Bismuth published a review with 55 well-documented cases of hemobilia, 53% originated in the liver, 23% in the gallbladder (almost all related to gallstones), 22% in the bile ducts, and 2% in the pancreas [2]. Our patient had rupture of Hepatic artery pseudoaneurysm into the common bile duct.

Based on severity hemobilia is classified into mild, moderate and severe. In mild and moderate hemobilia usually the blood loss is <10% and thus, it does not necessitate blood transfusion. Mild to moderate hemobilia usually resolve within 48 hours without any interventions. Severe hemobilia is diagnosed when hemorrhage constituting blood loss is greater than 10% of the total blood volume and results in hemodynamic instability that necessitate transfusion. Our patient had melena and drastic drop in haemoglobin and fits into severe hemobilia [3].

Upper GI scopy helps in identifying hemobilia, only in about less than 10% of patients but the sensitivity is improved if the patient has active bleeding. Our patient was having active bleeding at the time of presentation and we were able to appreciate it in the upper GI scopy with ooze from the ampulla and blood clots in the duodenum. Digital subtraction angiography forms the diagnostic modality of choice in severe hemobilia patients which helps in identifying the bleeding source and also helps in arterial embolization which can be done as a single procedure to the patient [4,5].

Mild to moderate hemobilia can be managed conservatively without the need of trans-arterial embolization. But, in patients with severe hemobilia adequate blood products need to be transfused and coagulopathy if present need to be corrected. Angiography with transarterial embolization forms the gold standard of management in severe hemobilia achieving cure rate in about 80 to 100 percent of patients. Surgery is rarely indicated. Surgery is indicated in patients with recurrent bleeding after arterial embolization.

More than half of the patients with hemobilia would have undergone liver biopsy or some form of hepatobiliary manipulation like ERCP, which rarely develop pseudoaneurysm and later rupture and causes upper gastrointestinal bleeding [6,7].

Conclusion

In our patient, he had ERCP and CBD stenting done for choledocholithiasis which would be the probable cause of Hepatic artery pseudoaneurysm, and later he presented with upper gastrointestinal bleeding four months later. Since he had severe blood loss and active oozing of blood was seen from the ampulla, he was taken up for digital subtraction angiography by which diagnosis and treatment was given to the patient in the single sitting. Though hemobilia is rare, it should be kept in mind if any patients with hepatobiliary system manipulation in the recent past presents with upper Gl bleeding.

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