

Hemobilia - a sporadic cause of hematemesis

Nagesh Kamath¹, Venkatakrishnan Leelakrishnan², Rajendiran Gopalan², Shiran Shetty^{1*}

¹ Department of Gastroenterology and Hepatology, Kasturba Medical College, Manipal University, Manipal, India - 576104.

² Department of Medical Gastroenterology, PSG Institute of Medical Sciences and Research, Coimbatore, Tamil Nadu, India - 641004.

ABSTRACT

A multitude of reasons contribute to gastrointestinal hemorrhage, hemobilia being less frequent. Here we present a case of hematemesis who was later found to have bleeding from the biliary tract that was due to pseudo-aneurysm arising from the hepatic artery. The diagnosis was supported by angiogram. He underwent coil embolization post which bleeding stopped. We present this case since hemobilia is a rare event and classical finding of blood clot on the ampulla on endoscopy is rare as seen in our case.

Key words: Hemobilia, pseudoaneurysm, right hepatic artery, coil embolization.

Citation: Kamath N, Leelakrishnan V, Gopalan R, Shetty S. Hemobilia – a sporadic cause of hematemesis. *Int J Pharmacol and Clin Sci* 2014;3:79-82.

INTRODUCTION

Gastrointestinal bleeding is among the most common emergency dealt by gastroenterologists needing quick initial triage assessment and appropriate resuscitation. Its incidence is about 50 to 150 per 1 lakh patients each year, with highest among areas of lower socio-economic status.^[1] Upper gastrointestinal bleeding (UGB) is associated with morbidity and mortality in India. It can be attributed to NSAID use viz. Ibuprofen, Diclofenac and Ketorolac. Peptic ulcer bleed is associated with whopping 60% of cases of UGB, while esophageal varices contribute to about 6%.^[2] Less common causes include Mallory-Weiss tear, gastritis, duodenitis, arteriovenous malformations, malignancy,^[3] and use of anti-platelets like clopidogrel, aspirin, anticoagulants viz. warfarin, low molecular weight heparin and unfractionated heparin. Upper endoscopy needs to be performed to exclude other causes of gastrointestinal bleed.

Hemobilia (bleeding in the biliary tree) occurs when conditions produce an abnormal communication between blood vessels and bile ducts.^[4] It can present many weeks after the initial injury.^[5] Bleeding can lead to biliary obstruction. The most common cause is liver

biopsy. Other more common causes include trauma, malignancy, arterio-biliary or arterio-portal fistula and pseudoaneurysm of the hepatic arteries.^[6] Hemobilia may be due to trauma - injury may be blunt (e.g. a fall, road traffic accident) or penetrating (e.g. stab or gunshot injuries); iatrogenic causes include percutaneous biliary drainage procedures, percutaneous liver biopsy, bleeding disorders, e.g. haemophilia.^[7] Inflammatory conditions, e.g. polyarteritis nodosa. Endoscopy is diagnostic in only 12% of cases.^[8] The causes and underlying mechanisms are many, the diagnostic approach employed and treatment modalities are discussed.

CASE REPORT

We present a case who was managed at Department of Medical Gastroenterology, PSG Institute of Medical Sciences and Research, Coimbatore, India. A 25 year old male presented with hematemesis of 2 days duration along with melena. There was history of trivial blunt bicycle related abdominal trauma 3 weeks back,

Received : 08 - 08 - 2014

Revised : 29 - 12 - 2014

Accepted : 30 - 12 - 2014

* Correspondence : drshiran@gmail.com

Conflict of interest: Nil

Source of support : Nil

Copyright: © 2014 Journal. All rights reserved.

which was ignored. He denied any significant past medical history. No history of fever and liver disease. No history of NSAID or other herbal supplements intake. He was non-diabetic, non-hypertensive, non-smoker, nonalcoholic. No history of fever, sweats or chills. His bowel habits were regular. He complained of mild right upper quadrant pain, non-radiating, not related to meal or postural change.

The vital signs were BP: 100/64 mmHg, PR: 86 bpm, RR: 18 cpm, BT: 37.3°C. Physical examination was unremarkable except for mild pallor. Cardiac and respiratory examination were normal. Per abdomen examination showed tenderness in the right hypochondrium. Laboratory test showed microcytic hypochromic anemia. His hemoglobin was low 6.5 g/dl. LFT showed normal bilirubin with elevated liver enzymes. There was no A: G reversal. Coagulation profile

was normal. Serum amylase, lipase, calcium and triglycerides levels were normal. AFP was normal. Viral markers were negative. Ultrasound abdomen was not contributory.

Esophagogastroduodenoscopy showed normal stomach and duodenum. Side view scopy showed prominent ampulla with adherent clot in the tip as seen in figure 1. CT angiography abdomen was done which showed thrombosed pseudoaneurysm in the right segmental branch of hepatic artery as shown in figure 2. Under local anesthesia 4 cm x 3 mm coil was placed at the distal segment of the right hepatic artery as seen in figure 3. Patient was transfused 2 pint of packed RBC before the procedure and received intravenous fluids along with intravenous pantoprazole. Post embolization there was no drop in hematocrit, pain abdomen had subsided and laboratory values improved so was discharged after one week.

Figure 1: Endoscopic images of the duodenal ampulla showing a large blood clot exiting the orifice confirming the diagnosis of hemobilia

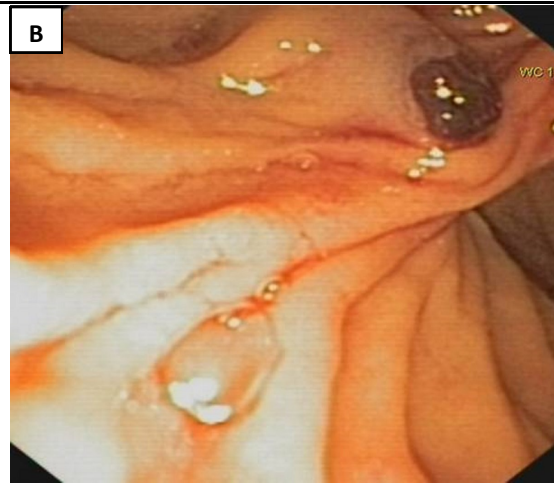
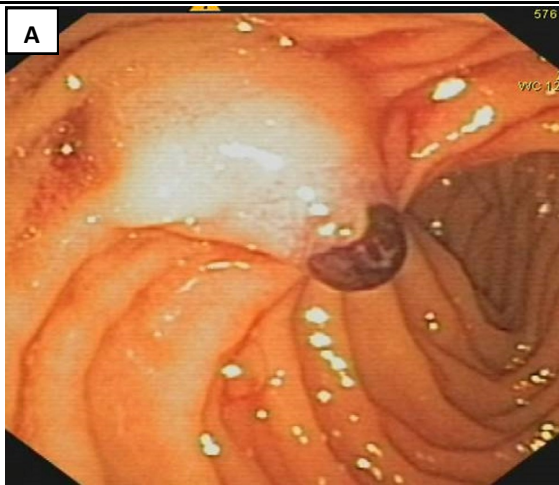
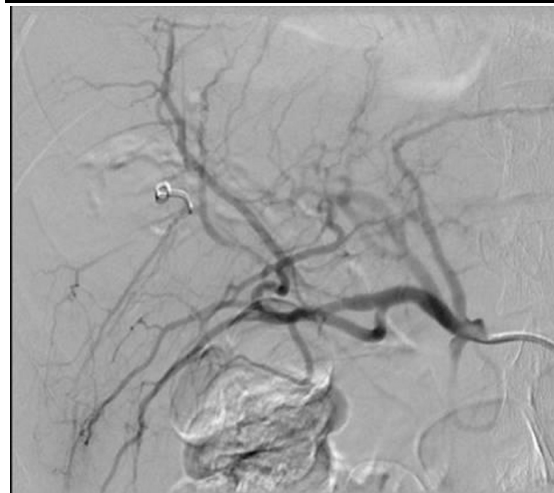


Figure 2: Angiogram showing leak from pseudo aneurysm located on the right hepatic artery



Figure 3: Post Coil embolization, Arteriogram showing 3 mm coils obstructing the replaced right hepatic artery with disappearance of pseudo aneurysm



DISCUSSION

Hemobilia is a kind of UGB caused by the connection between the biliary tract and blood vessels that results in bleeding through the duodenal papilla.^[9] Its incidence is increasing due to newer invasive diagnostic procedures resulting in better recognition.^[10] Hemobilia is a rare condition. Diagnosis is most usually done by angiography or percutaneous trans hepatic cholangiography. Both CT and ERCP are also useful in the diagnosis of hemobilia.

UGB occurs as a result of damage (ulceration or erosions) to the mucosa superjacent the blood vessels in the duodenum, stomach or esophagus. The mucosal layer integrity is affected by drugs, infection, excessive gastric acid production or alcohol. It can also occur due to bleeding esophageal varices often seen in chronic liver disease patients. The consequences of bleed could be hypotension and reduced tissue perfusion increasing the risks of damage to vital organs. Aim of therapy is to stop bleeding and restoration of bile flow past blood clots. Treatment options include electrocoagulation, angiography with embolization or surgical intervention. Angiography is considered the best among others with success rates above 95%.^[11-13]

The common features of UGB are hematemesis and melena. Initial evaluation should include hemodynamic assessment with orthostatic vital signs, and to correct fluid loss with crystalloid intravenous fluids and restore hemodynamic stability. Supplemental oxygen and plasma expanders with the use of packed red blood cells need to be considered, especially in frail or elderly patients, with tachycardia, hypotension, or Hb < 10 g/dl. Coagulopathy should be corrected. Hemobilia may cause acute pancreatitis. Hemosuccus pancreaticus can also present as gastrointestinal bleeding secondary to rupture of a pseudoaneurysm into the pancreatic duct.

A case of lethal hemobilia secondary to penetrating abdominal trauma was first

described by Glisson in 1654. Sandblom has shown that trauma is the cause of hemobilia in half of the cases, with 1/3rd of the cases being percutaneous transhepatic cholangiography, iatrogenic liver biopsy, with a period to onset of bleeding post accidental hepatic trauma of 4 weeks.

Hemobilia in our patient was due to abdominal trauma leading to posttraumatic pseudoaneurysm and it was diagnosed promptly by combination of EGD, angiogram, pathology and patient's disease state. Hepatocellular carcinoma was ruled out since ultrasound abdomen was unremarkable and AFP levels were within normal limits. Pancreatitis in hemobilia is seen due to obstruction of pancreatic duct but serum amylase, lipase, calcium and triglycerides levels were normal, hence pancreatitis was ruled out. In our patient 4 cms x 3 mm coil was placed at the distal segment of the right hepatic artery and hemostasis was achieved. There is need to monitor BP/pulse every 30 minutes until bleeding stops, then hourly for next 12 hours followed by 4 hourly. Proper diagnosis of hemobilia is needed, when lesser common causes of UGB have been ruled out. It is sometimes difficult to differentiate hemobilia from bile sludge or tumor. Hence routine tests need to be done to find the exact cause of hemobilia and give proper treatment. Laboratory tests and imaging techniques have limited accuracy for the diagnosis of hemobilia. Hence diagnosis remains a challenge for physicians, and high degree of suspicion is needed.

ACKNOWLEDGEMENT

Nil.

REFERENCES

1. Palmer K. Non-variceal upper gastrointestinal hemorrhage: guidelines. *Gut* 2002; 51 (Suppl 4): iv1–iv6.
2. Longstreth GF. Epidemiology of hospitalization for acute upper gastrointestinal hemorrhage a population-based study. *Am J Gastroenterol* 1995;90:206-10.

3. Massó González EL, Patrignani P, Tacconelli S, García Rodríguez LA. Variability among nonsteroidal antiinflammatory drugs in risk of upper gastrointestinal bleeding. *Arthritis Rheum* 2010;62:1592-601.
4. Sandbolm P. Hemorrhage into the biliary tract following trauma; traumatic hemobilia. *Surgery*. 1948; 24(3):571–586.
5. Wani NA, Gojwari TA, Khan NA, Kosar TL. Haemobilia in child due to right hepatic artery pseudoaneurysm: multidetector row computed tomography demonstration. *Saudi J Gastroenterol* 2011;17:152-4.
6. Demyttenaere SV, Hassanain M, Halwani Y, Valenti D, Barkun JS. Massive hemobilia. *Can J Surg* 2009;52:109-10.
7. Manolakis AC, Kapsoritakis AN, Tsikouras AD, Tsiopoulos FD, Psychos AK, Potamianos SP. Hemobilia as the initial manifestation of cholangiocarcinoma in a hemophilia B patient. *World J Gastroenterol* 2008; 14: 4241-4.
8. Yosida J, Donahue PE, Nyhus LM. Hemobilia: review of recent experience with a worldwide problem. *Am J Gastroenterol* 1987;82:448-53.
9. Jason K. Sicklick, D'Angelica M, Fong Y. The Liver. In: Courtney MT Jr, Daniel, Mark E, Kenneth L editors. *Sabiston Textbook of Surgery*. 19th ed. Philadelphia: PA Saunders; 2012. p. 1467-8.
10. Dousset B, Sauvanet A, Bardou M, LegMan P, Vilgrain V, Belghiti J. Selective surgical indications for iatrogenic hemobilia. *Surgery* 1997;121:37-41.
11. Franklin RH, Bloom WF, Schoffstall RO. Angiographic embolization as the definitive treatment of post-traumatic hemobilia. *J Trauma* 1980;20:702-5.
12. Wagner WH, Lundell CJ, Donovan AJ. Percutaneous angiographic embolization for hepatic arterial hemorrhage. *Arch Surg* 1985;120:1241-9.
13. Hirsch M, Avinoach I, Keynan A, Khodadi J. Angiographic diagnosis and treatment of hemobilia. *Radiology* 1982; 144:771-2.
