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Original Article

Major hemobilia – experience from a specialist unit in a developing country

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ABSTRACT

Background and Aim: Hemobilia is a rare but potentially life threatening problem, which can be difficult to diagnose and treat. In the last few decades there has been a change in the etiologic spectrum and management of this problem in the West. The aim of this study was to analyze the etiology, clinical features, management and outcome of major hemobilia in a tertiary referral centre from western India.

Methods: A retrospective analysis was undertaken on 22 patients (16 males, 6 females; mean age 39 years, range 13 to 74) who presented with major hemobilia over a 5-year period.

Results: The etiology was iatrogenic in 13 patients (percutaneous transhepatic biliary drainage 8, post laparoscopic cholecystectomy 3, endoscopic retrograde cholangiopancreatography 1, and liver biopsy 1), liver trauma in 6 and liver tumors in 3 patients. Twenty patients presented with gastrointestinal bleeding (melena 20 patients, hemetemesis with melena 8 patients), 5 with jaundice and 8 had fever. Abdominal angiography was performed in 20 patients. Angiography revealed pseudoaneurysm of the right hepatic artery or its branches in 14 patients, left hepatic artery in 2, an arterio-biliary fistula in 1, tumor blush in 1 and the source could not be located in 2 patients. Seventeen of the 22 patients were treated with radiological intervention, 3 required surgery (liver resection for tumors 2, laparotomy for venous collateral bleeding of portal cavernoma 1) and two were managed conservatively. Radiological intervention involved embolisation with coils and/or glue in 16, and chemoembolisation in 1 patient. Sixteen of 17 patients responded to embolisation. Overall there were two deaths.

Conclusion: The spectrum of hemobilia seen in India is now similar to that in the developed world with iatrogenic causes being the commonest. Interventional radiology can treat a majority of patients reducing the need and morbidity associated with surgery.

KEYWORDS: Hemobilia, interventional radiology

Introduction

Hemobilia is a complex clinical problem and occurs when there is an abnormal communication between the vascular and biliary system. Although it was first described in 1654 by Francis Glisson, it was not until 1948[1] that Sandblom gave a detailed description of hemobilia. Profuse haemorrhage into the biliary tract i.e. major hemobilia is rare but can lead to morbidity and mortality. Although there is significant data available from the west, there is little published information on this condition from India. With the increasing number of invasive hepatobiliary interventions being performed, the incidence of hemobilia is

likely to rise. The aim of this study was to analyze the spectrum, clinical presentation and management of major hemobilia in a tertiary referral centre from western India.

Methods

A retrospective analysis of 22 patients with major hemobilia was undertaken over a 5-year period. There were 16 males and 6 females with a mean age of 39 years (range: 13-74 years). Major hemobilia was defined as bleeding in the biliary tree causing overt gastrointestinal bleeding in the form of haematemesis/melena associated with a fall in haemoglobin of >3 gm/dl. Cholangitis was diagnosed when there was associated fever and leukocytosis. All patients were resuscitated with intravenous fluids and covered with broad spectrum antibiotics. Upper gastrointestinal (UGI) endoscopy was performed in case of doubt about the source of bleed.

Results

The main presenting symptoms were melena in 20 patients and hemetemesis (with melena) in 8 patients. Seventeen patients needed blood transfusions (mean: 3 units, range: 1-5 units). The mean drop in hemoglobin was 5 gm/dl (range: 3–7 gm/dl). Eight patients presented with associated cholangitis and 3 of these developed septic shock.Thirteen patients (59%) developed hemobilia secondary to iatrogenic causes (percutaneous transhepatic biliary drainage 8, post laparoscopic cholecystectomy 3, endoscopic retrograde cholangiopancreatography 1, and liver biopsy 1). There was history of trauma in 6 patients and 3 patients bled from liver tumors. The hemobilia was seen immediately in the endoscopic papillotomy patient, but developed after a mean of 6 days in the post percutaneous transhepatic biliary drainage (PTBD) group and 11 days in the trauma group. Four out of the six patients with trauma had undergone laparotomy elsewhere, with 3 requiring suturing of liver lacerations.

Eight patients had an UGI endoscopy and fresh blood was seen in the second part of duodenum in five. Ultrasound examination with Doppler was performed in 10 patients, which revealed filling defects in the biliary tree suggestive of clots (3 patients), pseudoaneurysm (6 patients) and was normal in one patient. Doppler was performed in some cases elsewhere before referral.

Abdominal angiography (celiac & SMA) was performed in 20 out of the 22 patients. Angiography was not done in two patients (one patient had venous bled from portal biliopathy after stone extraction and the other underwent surgery directly for liver tumor, since the bleeding had stopped). Angiography revealed pseudoaneurysm of the right hepatic artery or its branches in 14 patients, left hepatic artery in 2, an arterio-biliary fistula in 1, tumor blush in 1 and the source could not be located in 2 patients. **Figure 1 (a, b, c)** shows traumatic pseudoaneurysm of a branch of right hepatic artery along with its embolisation. Seventeen patients were treated with radiological intervention in the form of embolisation (coils and/or glue-16, and chemoembolisation with doxorubicin –1). **Figure 2 (a, b, c)** shows a PTBD induced psuedoaneurysm with angioembolisation. Sixteen patients responded immediately to embolisation. One patient required two sessions, as he had an

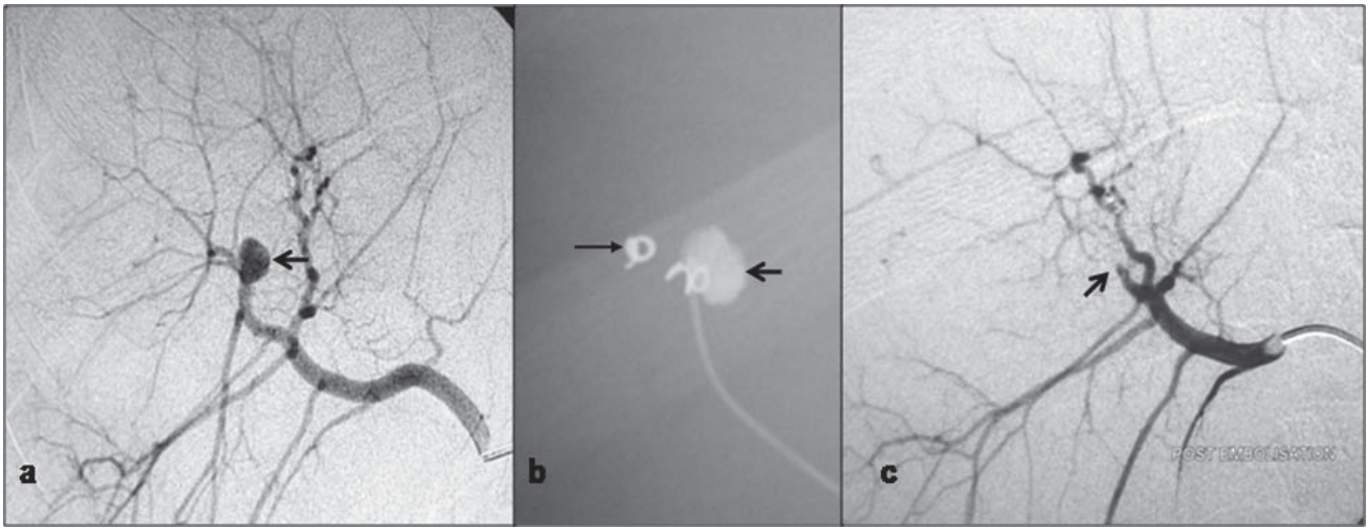

Figure 1: (a) Right hepatic artery angiogram showing pseudoaneurysm (arrow) of its branch (b) Embolisation using distal placement of coils (long arrow) followed by injection of glue (short arrow) into pseudoaneurysm through microcatheter (c) Post embolisation angiogram showing complete occlusion of the pseudoaneurysm (arrow)

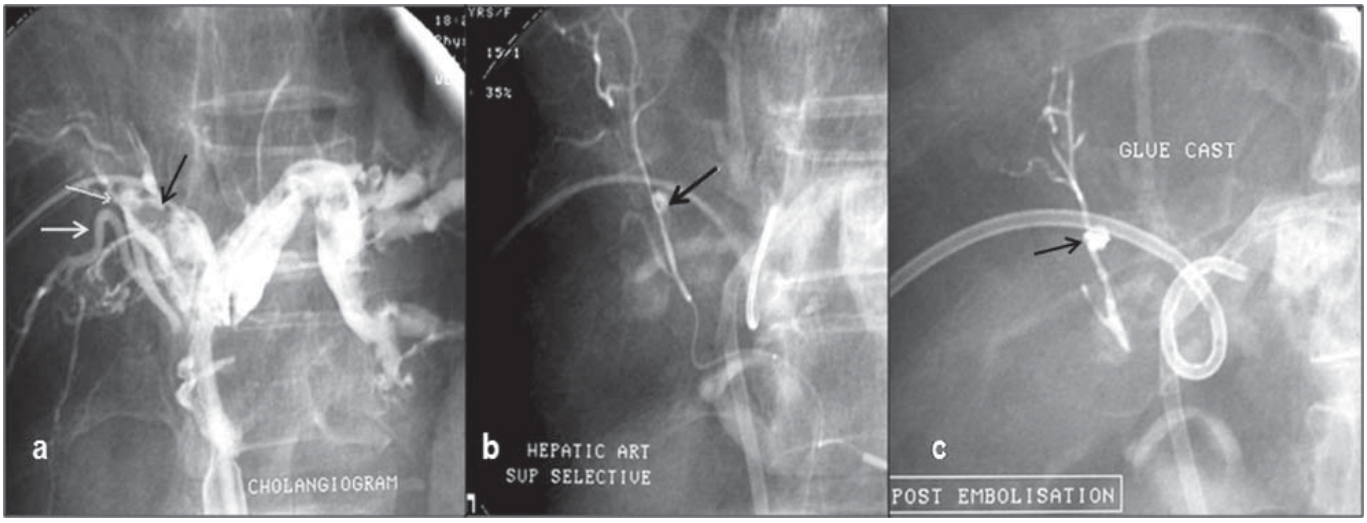

Figure 2: (a) Cholangiogram through the percutaneous transhepatic biliary drainage tube showing a filling defects (black arrow) in the biliary tree suggestive of clots. There is retrograde filling of the anterior division of right hepatic artery (white arrow) (b) Superselective right hepatic anterior branch angiogram showing a tiny pseudoaneurysm (arrow) (c) Embolisation with dilute glue (arrow)

accessory right hepatic artery arising from the superior mesenteric artery which was the cause of bleed and was missed on the initial angiogram. Three patients required surgery (liver resection for hepatocellular carcinoma – 2, laparotomy for bleeding from a portal biliopathy – 1) and two were managed conservatively. The cholangitis did not need separate drainage but settled with control of the hemobilia.

There were two early deaths (mortality: 9%). One patient with portal cavernoma died of massive venous bleed, which could not be controlled at laparotomy and the other due to liver failure 1 week after chemoembolisation. The other two patients with liver tumors died during the follow up due to extensive disease. Out of the eight patients who had undergone PTBD for hilar cholangiocarcinoma (4 preoperative biliary drainage, 4 palliative drainage and stenting), 4 were lost to follow up and 4 underwent definitive surgery once the bilirubin was < 3 mg/dl. Two patients in the post cholecystectomy group underwent definitive biliary repair after 3 months. Table 1 summarizes the etiology, angiography findings, management and outcome of our patients.

Discussion

In the first large review of hemobilia consisting of 545 patients published by Sandblom in 1972,[2] trauma was noted as the most common causative factor. In developed countries the increasing use of interventional procedures has now resulted in iatrogenic injury in being the most common cause.[3]

The incidence of iatrogenic hemobilia following liver biopsy, percutaneous transhepatic cholangiography and PTBD is 1%[4] and 2-10%[5,6] respectively. The commonest iatrogenic cause of hemobilia in our series was secondary to PTBD procedures, which was seen after mean of 6 days. A combination of pressure from a stiff catheter and local sepsis causes erosion of the blood vessel wall, leading to formation of a pseudoaneurysm in close proximity to the bile duct, causing hemobilia on rupture.

The incidence of blunt liver injury causing hemobilia is about 2.5 - 3%.[7] Out of the 6 patients with liver trauma in our series, 3 had undergone hepatorrhaphy elsewhere. The frequent reason is bleeding within the liver substance with intact capsule or superficial suturing of the injury, with resultant hematoma rupturing into the biliary tree.[8] The hematoma may also get infected leading to a mycotic pseudoaneurysm, which may later erode into the biliary tree. Therefore, while exploring a liver laceration, precise identification and suturing of vessels and ducts is important. The trend towards conservative management of liver injury may be expected to result in an increase in the incidence of hemobilia.[9] Therefore we believe that there should be a low threshold for follow up imaging in this group of patients.

Tumors account for about 6% of all causes of major hemobilia and malignant tumors outnumber the benign ones.[10,11] Hepatocellular carcinoma secondary to cirrhosis is the commonest tumor causing hemobilia.[12] The three tumor patients in our series represent an interesting group and all of them had small lesions.

The classic triad of upper abdominal pain, GI bleed and jaundice,[13] was seen only in a minority of our patients. We believe that for any patient undergoing hepatobiliary intervention and subsequently presenting with persistent

abdominal pain with pallor, there should be a low thereshold in suspecting hemobilia. Side viewing endoscopy can identify bleeding from the papilla in only 30% of patients.[14] We could demonstrate fresh blood in the second part of duodenum on UGI scopy in 62% (5/8 patients). Pseudoaneurysms are noted as well circumscribed anechoic lesions on ultrasound and doppler shows a turbulent flow within,[15] as was seen in 60% (6/10 patients) of our patients. CECT can demonstrate smaller hematomas, anatomical variations, pseudoaneurysms and cavitating lesions.[16]

Once hemobilia is strongly suspected, the most useful study is angiography, which may reveal the precise source of bleed.[17] It can also be combined with definitive therapy by radiological intervention. Celiac axis angiography should always be accompanied with superior mesenteric arteriography because anomalous/accessory right hepatic artery may originate from the SMA in almost 20 % of the patients, and was observed in one of our patients. Selective right and left hepatic angiogram may be performed especially in cases of hemobilia secondary to PTBD, as nonselective angiograms may miss small pseudoaneurysms. The angiogram may sometimes appear normal in the absence of any active bleeding or when there is no demonstrable lesion. Hence we feel that if bleeding continues or recurs it may be worth repeating an angiogram.

Angioembolisation has now become the first line of treatment and involves selective occlusion with permanent embolic agents like microcoils and cyanoacrylate glue.[18] Coils induce thrombosis, hence with gross coagulopathy, the vessel may remain patent. Glue can be used to treat smaller pseudoaneurysm where coil placement may be difficult. Also it conforms to the shape of pseudoaneurysm and forms a cast instantly, even in the presence of coagulopathy, and is much cheaper as compared to coils. Combination of coil and glue also can be used.[19] Ideally embolisation distal and proximal to the pseudoaneurysm is necessary to prevent collateral filling of the pseudoaneurysm. Alternatively, complete occlusion of the pseudoaneurysm with coils or glue may be done followed by proximal occlusion with coils.

Previous reviews and retrospective series have shown the success rate of transarterial embolisation (TAE) to be in the range of 80 – 100%.[4] We had a success rate of 90%. Failure may be due to technical reasons or extensive collaterals. Antibiotic prophylaxis is recommended.[20] Selective embolisation as close to the pseudoaneurysm or fistula possible is desirable to reduce the likelihood of both recurrence and hepatic necrosis.

Emergency surgery to control major hemobilia is difficult and should be avoided, as the results are poor. It may be better to transfer the patient to a center with angiography facilities rather than operate. Operative intervention may be required if radiological expertise is not available, there is failure of TAE, or manifestation of hepatic sepsis.[21] It involves ligation of the bleeding vessel and hepatic resections. If bleeding is not controlled by ligation or there is severe trauma an urgent liver resection may have to be performed. Our mortality of 9% was slightly higher than that the 5% reported in literature.[3]

There is paucity of literature regarding the etiology, management and outcome of hemobilia from India. The published case reports from India mainly describe hemobilia following trauma or cholecystectomy.[22,23] In the only published series by Srivastava et al,[24] the predominant etiology of hemobilia was liver injury following road traffic accidents. It highlighted the role of TAE in the management of hemobilia.[24] Our series highlights the changing spectrum of hemobilia from liver injuries to hepatobiliary interventions in our country, mirroring the spectrum of the developed world. This could be due to a referral bias. It also emphasizes the role of angiography in its diagnosis and management. A high index of suspicion and timely intervention is important. In India the increasing incidence of biliary stone disease, as well as increasing interventions on the hepatobiliary system are likely to result in clinicians encountering this problem more frequently.

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