Hemobilia: Evaluation and Management

CASE VIGNETTE

A 68 year old man with chronic pancreatitis developed splenic vein thrombosis with extensive peripancreatic collateral vessels, including around the pancreatic head and duodenal wall. The patient presented with new jaundice and melena and was referred for ERCP. ERCP was performed and revealed ampullary varices with active hemobilia, consistent with variceal erosion and hemorrhage into the distal common bile duct. (Figures 1 and 2) A fully covered metal stent was placed across the distal CBD to produce tamponade on the bile duct, which stopped the bleeding temporarily. (Figure 3) The patient was referred to surgery for splenectomy, which was performed to good effect. The stent was subsequently removed.

Etiology

As a clinical entity, hemobilia can develop from a wide variety of conditions and has been reported in patients with hereditary architectural vascular disease, auto-immune or inflammatory conditions, and malignancy. However, historically the vast majority of cases have arisen in patients who suffered from abdominal trauma. In recent years, with the advent of percutaneous and endoscopic procedures, there has been a shift over time towards iatrogenic cases, and most cases of hemobilia in the modern era are secondary to interventional procedures.

Hemobilia has been reported as a complication of many procedures including, but not limited to, percutaneous and transjugular liver biopsy, percutaneous transhepatic biliary drainage and cholangiography, radiofrequency ablation of hepatocellular carcinoma, endoscopic retrograde cholangiopancreatography (ERCP) as well as surgical procedures including laproscopic cholecystectomy, liver resection, and liver transplantation.

Mechanism of Injury

Typically, hemobilia occurs when there is direct trauma to the biliary tree and the hepatic blood supply. This then leads to the formation of a fistula followed by a bleeding event, which is often immediate. Delayed bleeding has also been reported in some patients. In cases of delayed hemobilia, the initial trauma of the procedure and subsequent inflammation can lead to the formation (continued on page 32)
of a pseudoaneurysm in an adjacent blood vessel. The pseudoaneurysm can then, due to poor structural integrity, rupture or fistulize at a later date, which effectively expands the time between the initial injury and subsequent presentation of hemobilia. Furthermore, both arterial and venous vessels can be affected and have been implicated as sources of hemobilia.

**Incidence and Presentation**

Hemobilia can present on a spectrum ranging from an immediate bleed with hemodynamic compromise to minor upper gastrointestinal bleeding that may not even be noticeable clinically. Furthermore, symptoms may not be temporally related to the inciting event, i.e., the initial trauma may have occurred months prior to symptom onset. To complicate this further, hemobilia can present as an intermittent gastrointestinal bleed, which may further impede accurate diagnosis. Quincke’s triad of abdominal pain, upper gastrointestinal bleeding and jaundice has been described as the classical presenting symptoms but may only be present in 20-30% of patients. For these reasons, the incidence of hemobilia by procedure type or technique is not well understood.

In a retrospective review of 333 patients who underwent percutaneous biliary drainage, Savader et al. (1992) found that 13 patients developed hemobilia with symptom onset ranging from 1 day to 1.8 years following catheter placement. (Figure 4) However, the incidence of hemobilia in percutaneous procedures has been estimated to range from 2.3 - 3.9% in two moderate sized retrospective reviews of percutaneous biliary drainage and 0.005% in those who underwent percutaneous liver biopsy in a large multicenter retrospective study of 68,276 patients. Hemobilia following transjugular liver biopsy is relatively rare and has been reported at 0.006% in a retrospective review of 601 patients.

Post endoscopic hemobilia has not been well described in the literature. To our knowledge, there are no large retrospective studies that have examined hemobilia following endoscopy. Of the reported cases, hemobilia appears to most commonly follow biliary stenting and may be more prevalent among patients who received metal stents as compared to plastic.
to those who received plastic stents.22,23,24,25,26
(Figure 5) Hepatic artery pseudoaneurysm from traumatic stent placement has been reported following biliary stenting and is thought to be the mechanism that promotes hemobilia.15 Otherwise, hemobilia has been reported in patients with malignancy or underlying architectural vascular disease who undergo ERCP.27,28 To complicate matters further, there are no known risk factors that may aid in the identification of those with an increased likelihood of developing hemobilia as a complication of endoscopy.

Management
Currently, there are three main methods that can be utilized in the definitive treatment of hemobilia: surgical intervention, biliary stenting, and angiography followed by selective embolization. The approach to management may change depending upon hemodynamic stability, the suspected source of bleeding, and the inciting event.

Historically, surgical intervention with segmental liver resection, pseudoaneurysm repair, and/or nonselective arterial ligation was the mainstay of treatment. However, more recently surgery has been reserved to patients with extensive liver trauma, hemodynamically instability, and those who develop hemocholecystitis or have failed alternative therapies.29

Endoscopic biliary stenting has been reported to be successful in the treatment of hemobilia but is often a poor choice for definitive therapy as the location of the bleed must be readily identifiable, extrahepatic, and easily accessible.30 Covered and non-covered self-expandable metal stents have seen some success in the treatment of massive hemobilia that occurs from bile duct varices or primary tumor bleeding due to extrahepatic malignancy.31,32 In these patients, the stent will generally be left in place for a prolonged period of time (or forever) unless the patient develops another complication such as stent obstruction or cholangitis. It is believed that expandable stents are able to distribute pressure over the communicating venous vessel and thereby provide a tamponade effect to achieve hemostasis.

Angiography with selective embolization was first described in 1976 and now represents the first line therapy to achieve hemostasis as it is noninvasive and has a high success rate with an associated low morbidity.33,34 Furthermore, angiography with selective embolization is the only intervention that can both delineate the vascular anatomy and the location of the pseudoaneurysm or fistula and guide therapeutic intervention. Although one drawback is that false negative studies may occur if there is intermittent bleeding.15 Embolization is typically achieved through the

(continued on page 36)
injection of Gel-foam, polyvinyl alcohol particles or steel coils at the site of the lesion. Inadvertent embolization of adjacent arteries leading to fatal ischemic disease has been reported but occurs infrequently. A caveat to these treatment modalities may exist in patients who develop hemobilia secondary to percutaneous biliary drainage. In this scenario, success with repeated drain flushing followed by an upsizing of the percutaneous catheter may provide a tamponade effect. Regardless of which treatment modality is performed, initial evaluation should be directed at hemodynamic stabilization and resuscitation.

After hemostasis is achieved and the patient is hemodynamically stable, attention is often shifted to the clearance of blood from the biliary tree if clinically indicated. However, in minor hemobilia, patients can often be managed conservatively with the correction of any underlying coagulopathy and volume resuscitation with intravenous fluids or transfusions. In those who experience persistent minor hemobilia without a recognizable vascular lesion on imaging, it is reasonable that ERCP be performed, as there may be an identifiable lesion of the distal biliary tree, which could be amendable to biliary stenting. In patients with minor hemobilia, most intrabiliary blood will dissolve under the influence of flowing bile. However, if intrabiliary blood becomes immiscible it is prone to forming a pure clot, which may lead to biliary obstruction and would warrant nasobiliary drainage or ERCP with sphincterotomy and ductal clearance.

Hemocholecystitis is a feared complication of biliary obstruction as it may necessitate the need for surgical intervention, which carries an increased risk of mortality. Furthermore, it has been hypothesized that even transient obstruction may favor the reflux of blood clots into the cystic duct. Thus, if biliary ductal dilatation is demonstrated on imaging then decompression through either a nasobiliary drain or endoscopic sphincterotomy and clot extraction should be pursued.

In instances of major hemobilia, surgical intervention is warranted as this approach allows for the identification and selective ligation of the bleeding vessel. If a bleeding vessel cannot be visualized then nonselective ligation of the hepatic vasculature with intraoperative bleed surveillance may be pursued. Nonselective ligation allows for gross localization of the source of hemobilia, which can then guide more precise interventions. In rare instances, both ligation and selective embolization can be insufficient in controlling the bleed and partial heptectomy may be required.

Discussion
Hemobilia should be suspected in patients with evidence of upper GI bleeding in the setting of a history of prior hepatobiliary intervention or malignancy. The management of hemobilia will depend upon the presenting symptoms and the acuity of the bleeding itself but should initially focus on hemodynamic stabilization. Hemobilia can often be managed conservatively with transfusions and the correction of any underlying coagulopathy. However, in patients with persistent bleeding, angiography with selective embolization is the first line therapy to achieve hemostasis but endoscopic biliary stenting and surgical interventions may be warranted. Biliary dilatation on imaging may represent obstruction and should warrant biliary decompression with either nasobiliary drainage or ERCP with sphincterotomy and clot extraction. Hemocholecystitis carries an increased risk of mortality and is a feared complication of hemobilia with biliary obstruction. The presence of hemocholecystitis or bleeding unresponsive to less invasive therapies should warrant urgent surgical intervention.

References


Answers to this month’s crossword puzzle:

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P I P S  R I B O S O M I L L U R
A A B N  W A I U
N U R S E  N O N T U M O R
C A D Y E F N G I
R M S  S E R O L O G I C C
E I T I C U H A
A N T A C I D V I L L O U S
S E R I P L L E
A S T O D U O D E N A L
A H S H O T N S
C H R O N I C C G U T
Y U E C A L I A
C O L O R E C T A L S T A T
L E N U R M I E
E S P A S M B I O S Y
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