Hemobilia

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Abstract

Keywords

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Hemobilia is a rare source of upper gastrointestinal bleeding, though the incidence is increasing along with the rise in minimally invasive biliary interventions. Prompt diagnosis and treatment rests on having appropriate clinical suspicion which should be based on the patient's presenting signs and symptoms, as well as history including recent instrumentation. Endoscopy should be reserved for cases of upper gastrointestinal bleeding with low suspicion for hemobilia. Interventional radiology may be the first-line diagnostic and therapeutic option for patients with a high suspicion of hemobilia. While embolization is the mainstay of therapy, other options include thrombin injection, stent placement, and/or placement of a percutaneous biliary drain. Surgery should be reserved for failed treatment by interventional radiology.

Objectives: Upon completion of this article, the reader will be able to discuss the current diagnostic and treatment options for managing hemobilia.

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Hemobilia results from a fistulous communication between the hepatic vasculature and the biliary ductal system. It is a relatively rare cause of upper gastrointestinal (GI) bleeding, though it is increasing in frequency as hepatobiliary interventions become more common. The first description of hemobilia was in 1654 by Francis Glisson, Regius Professor of Physics at the University of Cambridge, who reported that a liver laceration sustained during a sword fight caused fatal biliary bleeding 1 week after the injury.¹ However, the term *hemobilia* was not coined until 1948 by Philip Sandblom.² The rarity of this condition is reflected by

the preponderance of case reports and paucity of series in the literature.

Presentation

Presenting symptoms of hemobilia include melena, abdominal pain, hematemesis, and jaundice. Slowly bleeding lesions may present as iron-deficiency anemia. The clinical triad of jaundice, upper abdominal pain, and obscure upper GI bleeding is known as Quincke triad and suggests a biliary tract bleeding source.³ However, only 22 to 35% of patients will have all these three symptoms.^{4,5} In patients with a percutaneous transhepatic biliary drain (PTBD) in place, bloody output from the biliary drain or along biliary drain tract may be the first sign of hemobilia.

The onset of bleeding after an interventional procedure or trauma is variable, ranging from a few days to months. Most commonly, hemobilia occurs within 4 weeks of bile duct injury.⁶ Iatrogenic endoscopic retrograde cholangiopancreatography (ERCP)-related hemobilia tends to occur immediately after or within several days of the procedure. Delayed onset can be due to bile stasis, hepatic necrosis, or a slowly expanding pseudoaneurysm. Chronic bile duct erosion and inflammation can also result in injury to adjacent vessels and delayed-onset hemobilia.⁷

Hemobilia is frequently accompanied by clot formation within the biliary system. This may be attributed to

differences in specific gravity between blood and bile which prevents the two from mixing. Clot formation may consequently result in biliary obstruction and jaundice.

Hemorrhage into the biliary tree is predominantly arterial in origin due to the high-pressure differential between the hepatic artery and bile ducts. Biliary-venous communications on the other hand are more likely to stop spontaneously owing to the low-pressure gradient, though venous hemobilia may still occur, particularly in the setting of portal hypertension.⁸ In rare instances, biliary fistulae to the portal or hepatic vein may result in flow of bile into the venous system with resultant hyperbilirubinemia and jaundice.⁹

Etiology

The etiology of hemobilia is broad and includes blunt liver trauma, iatrogenic injury, stones, inflammatory conditions, vascular pathology, and neoplasm. The first large published review of hemobilia by Sandblom in 1973 reported that the majority of cases were attributable to accidental trauma, with only 17% found to be iatrogenic in origin.¹⁰ The most recent review, based on literature published between 1996 and 1999, reported that approximately two-thirds of cases were the result of iatrogenic injury, with trauma accounting for only 6% of cases.⁵ Iatrogenic causes include both diagnostic and therapeutic hepatobiliary interventions such as percutaneous transhepatic cholangiography (PTC), PTBD, percutaneous and transjugular liver biopsy, transjugular intrahepatic portosystemic shunt, and portal venous interventions. Owing to the close relationship between the intrahepatic bile duct and the branches of the hepatic arteries and portal vein, needle punctures from percutaneous interventions can easily cause fistulous communications leading to hemobilia. Additionally, as bile is known to damage blood vessels, simultaneous injury to bile duct and artery can predispose to formation of pseudoaneurysm.¹¹

Though iatrogenic injury accounts for the most common cause of hemobilia, the rate of vascular complications/injury after PTBD is still quite low overall, reported at 2.3% in one series.¹² A more recent series published by the same group found the incidence of hemobilia from PTBD to be higher (2.2%) than the rate from PTC alone (0.7%), suggesting that the process of tract dilatation and drain placement may also cause inadvertent enlargement of the iatrogenic fistula.¹³ Others have suggested that a combination of pressure from a relatively stiff biliary drainage catheter and local infection may lead to erosion of the blood vessel wall, resulting in pseudoaneurysm formation and hemobilia upon rupture into an adjacent bile duct.¹⁴ Avoidance of major vessels during needle placement may be facilitated by ultrasound guidance of PTC/PTBD, though subsegmental branches can be difficult to avoid for even the most experienced operators.

Hemobilia accounts for 3% of all major percutaneous liver biopsy complications,¹⁵ though this figure may underestimate the true incidence of iatrogenic injury. In one study, transient subsegmental hepatic parenchymal enhancement was found on CT in 38% of patients following percutaneous biopsy. This finding suggests that the majority of traumatic

fistulae resulting from percutaneous biopsy resolve spontaneously. Transjugular biopsy can also cause massive, even life-threatening, hemobilia.¹⁶

Other iatrogenic causes of hemobilia include ERCP and hepatobiliary surgery. ERCP-related injury includes bile duct perforation, as can occur during removal of stones. Cases of hemobilia have also been reported after endoscopic stent insertion—possibly due to erosion by the stent itself. Post-cholecystectomy hepatic artery pseudoaneurysms present with hemobilia in approximately 20% of cases.¹⁷ It is worth mentioning that asymptomatic hepatic artery pseudoaneurysms should be treated to avoid potential complications of compression of or rupture into adjacent bile ducts.

Hepatobiliary tumors are the predominant cause of non-iatrogenic hemobilia.¹⁸ It has been postulated that in patients with biliary obstruction, as often results from tumoral mass effect, there is an increase in hepatic arterial blood flow along with hyperplasia and distention of the arterial peribiliary vascular plexus. This is thought to predispose this patient population to bile duct fistula hemorrhage.¹⁵

Diagnosis

The diagnosis of hemobilia as the source of upper GI bleeding can be easily overlooked given its infrequency. The intermittent nature of bleeding adds to the diagnostic challenge. Clinical suspicion should hinge on the presenting signs and symptoms, as well as medical history, including recent instrumentation. In some instances, the diagnosis may be straightforward such as with bloody output from biliary drainage catheters or cholecystostomy tubes. These patients should proceed directly to fluoroscopic interrogation of the biliary tract with provisions made to convert to angiography if needed. In cases without a high index of clinical suspicion, management should follow the standard protocol for upper GI bleeding beginning with endoscopy. Endoscopy allows for the evaluation of other potential sources of upper GI bleeding and is the first-line diagnostic and therapeutic modality in such nonspecific cases¹⁹ (→ Fig. 1).

At endoscopy, visualization of blood or clot arising from the papilla or the present of fresh blood in the second portion of the duodenum can help localize bleeding to the biliary system or pancreas (hemosuccus pancreaticus). In one small series of patients with suspected hemobilia, endoscopy demonstrated direct or indirect evidence in only 60%.⁴ Once a hepatobiliary source of hemorrhage is identified, there are no endoscopic procedures to achieve hemostasis. Unlike with gastric and small bowel sources of bleeding, there is no role for thermocoagulation, sclerosant injection, or clips/banding.

If endoscopy is nondiagnostic, CT imaging should be considered next. We prefer CT angiography (CTA), as it permits the evaluation of the arterial anatomy in addition to the hepatobiliary parenchyma. CTA is also helpful in planning endovascular interventions, and can be particularly useful when dealing with surgically altered anatomy such as with transplanted livers. Direct findings include contrast extravasation into the biliary tract or liver parenchyma. Indirect findings include biliary ductal dilatation, thrombus

Fig. 1 Flowchart of diagnostic evaluation of hemobilia.

within bile ducts or the gallbladder, pseudoaneurysms/aneurysms, arteriovenous fistulas, or vascular malformations. Other indirect findings such as hepatic tumors, calculi, or signs of blunt liver trauma may point to the etiology of the bleed.

Digital subtraction angiography (DSA) is indicated when the bleeding site is identified by endoscopy or imaging, or when there is hemodynamic instability. Alternatively, if the patient presents with bleeding from an existing biliary drain or along the drain tract, fluoroscopic interrogation in the angiography suite should be undertaken with plans to convert to angiography as needed. The advantage of angiography is that therapy may be initiated immediately should the source of hemobilia be identified. Findings at DSA include extravasation into the biliary ductal system, arteriovenous fistula, or pseudoaneurysm. If a biliary drain is present, it should be removed over a wire prior to angiography, as the drain may induce a tamponade-like effect and obscure a bleed. Subselection and angiography of specific branches along the course of a biliary drain may be necessary if nonselective angiography of the liver is negative. Angiographic yield is reported between 88 and 100%.²⁰ In one study, catheter arteriography was successful in detecting 100% of hepatic artery pseudoaneurysms compared with only 67% by CT and 33% by Doppler ultrasound.²¹

Ultrasound may be helpful in some cases, though findings are nonspecific. For example, ductal dilatation can be seen as a result of partial or complete obstruction by thrombus secondary to hemobilia. Blood clots initially appear hyperechoic or isoechoic. As the thrombus organizes, it becomes more hypoechoic and, consequently, more easily missed. Adding to the difficulty is the fact that clot takes on the shape of the ducts. Clot within the gallbladder, on the other hand, is more easily recognized.⁴ Pseudoaneurysms may appear as well-circumscribed anechoic lesions with turbulent flow on

color Doppler imaging. Magnetic resonance cholangiopancreatography is more costly, takes more time, and provides suboptimal interrogation of vasculature, though it does well in demonstrating blood or clots within the biliary system. There is essentially no role for tagged-RBC studies in the diagnostic algorithm for hemobilia due to the lack of spatial resolution.

Treatment

Minor hemobilia may be managed with conservative therapy, whereas massive hemobilia, defined as potentially life-threatening hemorrhage, requires invasive treatment. Appropriate therapy requires not only resolution of hemorrhage but also restoration of bile flow if there is associated biliary obstruction.

Minor hemobilia typically presents as blood-tinged output from an internal-external or external biliary drainage catheter. Bleeding is attributable to injury to a vein during needle placement or tract dilatation. Conservative management in such cases should begin with correcting any underlying coagulopathies. A trial of capping of the drain may also facilitate hemostasis, but eliminating an outlet for hemorrhage. However, it is important to recognize that if an internal-external drain has been placed, bleeding may continue into the upper GI tract through the transpapillary route. If bloody output persists, the patient should be brought to IR for fluoroscopic interrogation of the drain and the biliary system. Cholangiography rarely reveals a vascular communication, as the direction of bleeding is into the bile duct, but it is helpful in identifying blood clots within the biliary tract or worsening ductal dilatation. Other maneuvers to manage minor hemobilia include slightly retracting or advancing the tube so that side holes do not engage the injured vessel or upsizing tube to create a tamponade effect.²² Upsizing of the drain is also

helpful in managing worsening biliary obstruction secondary to clots. Mild venous hemobilia typically resolves once the tract is mature.

Embolization

Arterial embolization is the primary treatment option for patients with unabating minor hemobilia, or with major hemobilia requiring transfusion.⁵ The dual vascular supply of the liver (75% via the portal vein and 25% via the hepatic artery) permits embolization of hepatic artery branches without significant concern for ischemia or infarction.

A celiac arteriogram is performed to delineate the hepatic vasculature, along with delayed imaging to confirm patency of the portal vein. Portal vein patency is important to establish prior to potential hepatic arterial embolization, especially in liver transplant patients. If hemobilia is related to recent PTBD, the drain should be removed over a guidewire prior to angiography to relieve the relative tamponade effect of the drain and to allow for better visualization of the vasculature. Superselection of the right and left hepatic arteries, or of any vessels along the path of the biliary drain, should be performed if no active bleeding or lesion is seen on celiac

Fig. 2 Hepatic artery pseudoaneurysm. (a) Percutaneous transhepatic cholangiogram with relatively central puncture site (arrow). (b and c) Axial and coronal CT images show the internal–external biliary drain crossing a segmental hepatic artery (dashed circle). A small pseudoaneurysm can be seen on the axial series (arrow). (d) Selective segmental hepatic arteriogram clearly shows a 3- to 4-mm pseudoaneurysm (white arrow) where the biliary catheter (black arrow) crosses the hepatic artery. (e) Microcoil embolization (arrow) of segmental hepatic artery pseudoaneurysm from distal to proximal.

arteriography. Superior mesenteric arteriography is also recommended to exclude accessory right hepatic arterial sources of hemobilia.

The primary angiographic findings of bleeding are visualization of active contrast extravasation and pooling into the biliary system. Other indirect signs of bleeding on angiography include pseudoaneurysm or vessel spasm. Identification of an arteriovenous fistula can also guide the operator to the injured artery. Just as with other causes of upper GI bleeding, hemobilia can be intermittent in nature and consequently missed on angiography.

Once the site of the bleeding is identified, superselective catheterization with a microcatheter and microcoil embolization is recommended. Detachable coils may be a prudent choice in cases where nontarget embolization will likely be poorly tolerated (transplant liver) and in patients with tortuous arterial anatomy. Pseudoaneurysms should be embolized across the neck of the aneurysm from distal to proximal. Because they lack a true wall, pseudoaneurysms may continue to expand if they are simply packed (►Fig. 2).

Liquid embolic agents (n-BCA or Onyx) can be useful in cases with extremely tortuous arteries or when multiple vessels feed a pseudoaneurysm. Additionally, coil embolization of smaller hepatic artery branches can be technically challenging and limited by vasospasm. Liquid embolic agents, however, do require greater operator experience, as overfilling can cause spillage of the agent into nontarget vessels or into the biliary ductal system.²³ Placement of a balloon across the bile duct at the site of arterial-biliary fistula before injecting the liquid embolic is one technique to avoid spillage.

Empiric embolization of the hepatic artery branches supplying the area of concern is not advised. Even with normal portal hepatic perfusion, arterial embolization can be complicated by hepatic necrosis, abscess formation, or biliary stenosis. The latter can occur because the bile ducts draw their supply primarily from the arterial rather than the portal system. If selective embolization cannot be performed, non-selective embolization of the right or left hepatic artery may be an option before resorting to surgery. In cases of massive life-threatening hemobilia, embolization of the common or proper hepatic artery can be justified, though it risks impairment of liver function (►Fig. 3).

Endovascular therapy is technically successful in 75 to 100% of cases.⁴ Failed treatment is usually due to inability to identify the bleeding vessel or missed collaterals.

Stent Placement

Placement of a covered stent across the site of vascular injury is an alternative option to embolization. It allows preservation of distal flow which can be a particularly beneficial in liver transplant patients. Most segmental hepatic arteries are small and require a low-profile delivery system and a small diameter covered stent. As such, balloon-expandable coronary stents can be useful in stenting an injured hepatic artery, as hepatic vessels are similar in size to coronary vessels. Proper sizing is critical to ensure sealing of the vascular injury. The stent diameter should be oversized by 10 to 20%

Fig. 3 (a and b) Large 5-mm pseudoaneurysm (arrow) resulting as a complication of laparoscopic cholecystectomy performed for chole-docholithiasis. (c) After unsuccessful attempt at placement of covered stent in hepatic artery due to angulation of celiac artery and proper hepatic artery, coil embolization of the proper hepatic artery with extension into the common hepatic artery was performed.

and the stent should ideally extend 10 mm on either side of the injury.²⁴

Deployment of stent grafts into small and/or tortuous vessels can be technically challenging. Newer flow-directing stents, with their relatively lower profile delivery system, may

be an alternative option. These stents are designed to increase laminar flow through the artery and reduce flow into the pseudoaneurysm, thereby allowing it to slowly occlude over time.²⁵

Stent placement within the biliary system, rather than within the vessel, may be considered in cases of portal-biliary fistula. In these cases, placement of a covered stent within a biliary duct can be performed through the existing PTBD tract and obviates the risk and challenge of accessing the portal venous system (► Fig. 4).

Percutaneous Thrombin Injection

Percutaneous thrombin injection (PTI) is an option for cases of hemobilia secondary to hepatic artery pseudoaneurysm.

Targeted pseudoaneurysms should have a narrow neck to avoid distal embolization. PTI of peripheral pseudoaneurysms has been proven to be a safe and effective treatment, and there are numerous case reports of PTI treatment of hepatic artery pseudoaneurysms.²⁶ While embolization of the dominant feeding branch to a pseudoaneurysm is usually feasible, should there be any distal intrahepatic collaterals, these may not be amenable to coil embolization. PTI can be quite useful in such cases, and may even avoid embolization of larger arterial distributions. In some patients, a combination of both coil embolization and thrombin injection is necessary to treat hemobilia.^{27,28}

As with peripheral pseudoaneurysm applications, this is an off-label use of thrombin. Dry powdered bovine thrombin

Fig. 4 (a) Coronal imaging in portal venous phase suggests possible portal-biliary communication. (b) Contrast injection through sheath opacified the biliary system (arrow) and remained static with no drainage into the small bowel. (c, d) Retraction of sheath uncovered portal-biliary fistula and allowed portal flow to briskly “wash out” contrast in the biliary tree as noted by the lower contrast density in the more delayed images. (e) Viabahn covered stent (arrow) placed within segmental biliary duct to exclude portal-biliary fistula. A new 8.5F biliary drain placed to facilitate clearance of clot within the biliary system.

(Thrombin–JMI, Gentrac, Middleton, WI) can be prepared with saline to a concentration of 1,000 IU/mL. The agent should be administered using a tuberculin syringe, as only a few hundred units is needed to achieve thrombosis.²⁹ Under ultrasound guidance, a 21-gauge needle is advanced into the pseudoaneurysm and thrombin is slowly injected using real-time color Doppler interrogation to assess for cessation of flow. While there are no published postprocedure surveillance recommendations, some authors suggest sonography at 1 week and 1 month following treatment.²⁷

Compared with coil embolization, PTI is less expensive, quicker, avoids contrast and radiation exposure, and is relatively less invasive. Potential risks include nontarget embolization with ischemia, bleeding resulting from needle placement, and anaphylaxis. The complication rate for peripheral PTI is less than 1%.³⁰

Surgery

Surgical intervention is rarely necessary, as the majority of hemobilia cases may be managed by interventional radiology. Surgery is reserved for failed endovascular and percutaneous therapies. Additionally, it remains the first-line treatment for pseudoaneurysms complicated by infection or compression of adjacent structures. Surgery may also be indicated in cases of hemobilia associated with tumor, cholelithiasis, cholecystitis, or intrahepatic sepsis.³¹ Surgical treatment options for hemobilia include hepatic artery ligation or excision of hepatic artery pseudoaneurysm. Hepatic segmentectomy or lobectomy is performed as last options. Surgery entails direct exploration of the liver with possible resection, ligation, aneurysm excision, cholecystectomy, and “release of bile duct obstruction.”²⁰ Reported surgical success rates approach 90%, though they are associated with a relatively high mortality (10%).⁴

Bile Clearance

Clot that forms within bile ducts can cause obstructive jaundice. Depending on the location of clot formation, the resultant obstruction can also lead to acute cholangitis, acute cholecystitis, or even acute pancreatitis.^{15,30,32} Consequently, biliary drainage is an important adjunct in the management of hemobilia.

Placement of a PTBD with regular drain irrigation can help manage biliary obstruction due to thrombus. Alternatively, ERCP with biliary sphincterotomy, nasobiliary drainage, and/or biliary stenting may be performed, although endoscopic placement of a (plastic) biliary stent may not be helpful as the stent can become occluded with clot. Percutaneous or endoscopic decompression of the biliary system is also an option, should inflammation of the pancreas or biliary system occur following treatment of hemobilia. There are also anecdotal reports of endoscopic or percutaneous infusion of thrombolytic agents into the biliary tree to dissolve clot.

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