Hemobilia: Historical overview, clinical update, and current practices

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Abstract
Hemobilia refers to macroscopic blood in the lumen of the biliary tree. It represents an uncommon, but important, cause of gastrointestinal bleeding and can have potentially lethal sequelae if not promptly recognized and treated. The earliest known reports of hemobilia date to the 17th century, but due to the relative rarity and challenges in diagnosis of hemobilia, it has historically not been well-studied. Until recently, most cases of hemobilia were due to trauma, but the majority now occur as a sequela of invasive procedures involving the hepatopancreatobiliary system. A triad (Quincke’s) of right upper quadrant pain, jaundice and overt gastrointestinal bleeding has been classically described in hemobilia, but it is present in only a minority of patients. Therefore, prompt diagnosis depends critically on a high index of suspicion based on a patient’s clinical presentation and a history of recently undergoing hepatopancreatobiliary intervention or having other predisposing factors. Treatment of hemobilia depends on the suspected source and clinical severity and thus ranges from supportive medical care to urgent advanced endoscopic, interventional radiologic, or surgical intervention. In the present review, we provide a historical perspective, clinical update and overview of current trends and practices pertaining to hemobilia.

KEYWORDS
abdominal pain, biliary flow, haemostasis, hepatopancreatobiliary interventions, upper gastrointestinal haemorrhage

1 | INTRODUCTION

1.1 | Overview

Hemobilia refers to the extravasation of blood into the biliary tract. The most common causes of hemobilia are iatrogenic, trauma related and neoplastic. While hemobilia represents only a small minority of digestive tract bleeding cases, its incidence has increased as the repertoire of minimally invasive hepatopancreatobiliary procedures has expanded. These same procedures, including advanced endoscopic and interventional radiologic techniques, have also opened doors to novel approaches for diagnosing and treating hemobilia. Despite these innovations, the diagnosis of hemobilia can be clinically challenging, and the most appropriate therapeutic approach may not be apparent or available.

This review provides a comprehensive yet concise summary of the aetiology, diagnosis and treatment of hemobilia. We present historical perspectives, clinical updates and current practices relevant for both specialists and subspecialists.
Historic background

The first reported case of hemobilia dates to 1654, when Francis Glisson wrote of a nobleman whom, while dueling, suffered a blow to the right upper abdomen leading to massive gastrointestinal bleeding and ultimately death. On autopsy, the source of bleeding was noted to be from a laceration of the liver which led to bleeding into the biliary system prompting the initial report of hemobilia. Antonie Portal was the first to report an incidence of hemobilia identified antemortem, describing a suspected hemobilia that was later confirmed on autopsy in 1777. Portal brought attention to the difficulty in identifying the specific location of bleeding, a problem still encountered today. Nearly a century later, Quincke described the clinical triad of right upper quadrant pain, jaundice and gastrointestinal bleeding, now referred to eponymously as ‘Quincke’s triad’. Literature from the early 1900s began to contain various case reports of biliary tract bleeding, but it was not until 1948 that the specific term ‘hemobilia’ was coined.

Epidemiology

Data on the epidemiology of hemobilia have been primarily presented as case reports and three large case series. In the first of these, Sandblom reported in 1973 a series reviewing 355 cases, including 59 iatrogenic cases (16.6%) and 137 (38.6%) traumatogenic cases. In 1987, Yoshida et al published a series of 103 patients with hemobilia, of whom 41% were iatrogenic and 19% were traumatogenic. In 2001, Green et al showed that the trend towards more iatrogenic causes of hemobilia continued, finding that among 222 patients, 65% had an iatrogenic cause and only 6% had a traumatogenic cause (Figure 1).

Clinical Presentation

The classically described presentation of hemobilia, that is, Quincke’s triad (Table 1), only occurs in only 22%-35% of cases. Hemobilia can also present with haematemesis, melena or haematochezia, with or without choluria, depending on the rate of bleeding, anatomical factors (eg post-bilioenteric surgical anatomy) and the cause.

The timing of symptoms can vary and may help in identifying the underlying cause of hemobilia. Endoscopic retrograde cholangiopancreatography (ERCP)-related hemobilia is usually seen immediately after or within days of the initial biliary injury (eg biliary stricturoplasty or sphincterotomy). Hemobilia can come from either an arterial or venous source; the latter is lower volume or self-limited bleeding (except in cases of portal hypertension) compared to the former.

Whatever the location of bleeding may be, the variability in the biochemical properties and density between blood and bile creates a distinct separation of the two within the biliary tree. Intraductal clot formation may ensue, functioning as a physical barrier to biliary outflow. Clots can also lead to biliary stasis and predispose to the development of acute cholangitis and right upper quadrant or...
epigastric pain. It should be noted that intraductal blood clots can be mistaken as biliary stones on imaging (because of their similar echogenicity) and require a high index of suspicion congruent with the clinical scenario.

On laboratory studies, hemobilia can present with anaemia and/or abnormal serum liver tests. The degree and rate of anaemia and liver test abnormalities depend on the etiology, severity and time point in the clinical course of hemobilia. Hyperbilirubinaemia and elevated alkaline phosphatase are the most frequent changes, though a mixed cholestatic-hepatocellular pattern has also been reported. In a series of 37 patients diagnosed with hemobilia, the mean total bilirubin was 10.5 mg/dL, alkaline phosphatase 834 IU/L, aspartate aminotransferase 353 IU/L and alanine aminotransferase 243 IU/L.

### 4.1  Iatrogenic causes

#### 4.1.1  Percutaneous interventions

Diagnostic percutaneous transhepatic cholangiography (PTC), percutaneous liver biopsy and percutaneous transhepatic cholangiography and biliary drainage (PTCD) placement have all been associated with developing hemobilia. Specifically for percutaneous liver biopsy, there have been inconsistent findings in the literature. For example, while a recent study by Zhou et al noted that hemobilia accounts for 3% of all major complications of percutaneous liver biopsy, a larger retrospective study of over 68,000 liver biopsies reported only a 0.005% risk (four cases of hemobilia of 68,276 liver biopsies). This inconsistency in the literature may be attributable to an increasing number of higher risk biopsies being performed or the improved ability to diagnose and greater reporting of hemobilia over time.

Percutaneous interventions can also cause hemobilia by inadvertently disrupting vascular structures while accessing the desired target. The potential for this is higher in cases where there is a chronic portal vein thrombus (especially in the presence of collateral vessels/cavernous transformation) or a non-dilated biliary tree (i.e. a smaller target to access). Comparing the risk of hemobilia associated with PTCD to that of PTC, it was noted in one study that PTCD had a higher rate of hemobilia, 2.2% vs 0.7% respectively. This over threefold increase in hemobilia risk may be secondary to both the larger size of the opening made in the tissues (e.g. the bile duct wall) with PTCD as well as there being a foreign material remaining in the duct which may incite local inflammation or erosion into the bile duct wall. A subsequent study reported similar findings, noting a 2.6% risk of hepatic artery injury with PTCD as compared to 0.7% with PTC.

Other percutaneous interventions which can result in hemobilia include transjugular intrahepatic portosystemic shunt (TIPS).

### 4.1.2  Endoscopic hepatopancreatobiliary interventions

The interventional endoscopic procedure most associated with hemobilia is ERCP, especially when accompanied by sphincterotomy. Bleeding related to sphincterotomy primarily occurs at the site of the cut papillary sphincter. Ultimately the risk of hemobilia depends on

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**TABLE 1  Spectrum of presenting features of hemobilia**

| Presenting features          |
|------------------------------|
| Jaundice^                   |
| Gastrointestinal bleeding^   |
| Haematemesis^                |
| Melena                      |
| Haematochezia                |
| Abdominal pain               |
| Right upper quadrant pain^   |
| Epigastric pain              |
| Laboratory test abnormalities|
| Anaemia                     |
| Leucocytosis                 |
| Abnormal serum liver tests   |

^Quincke’s Triad consists of right upper quadrant pain, jaundice and gastrointestinal hemorrhage. 

^Seen primarily in patients with surgically altered anatomy or absent/incompetent pyloric sphincter.

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**FIGURE 2** The three most common causes of hemobilia are (from left to right) iatrogenesis (from invasive procedures), trauma and malignancy.
the degree of invasiveness of the manoeuvres performed during ERCP (eg extraction of large stones or stricturoplasty) as well as patient-dependent variables such as coagulopathy and presence of underlying diseased tissue (eg a hypervascular or friable tumour). Further risk factors to be considered include variant anatomy (especially if not recognized up front), aggressive biliary balloon dilation, intraductal biopsies and vascular anomalies or disorders (eg Osler-Weber-Rendu syndrome, i.e. hereditary haemorrhagic telangiectasia).

Procedures such as endoscopic ultrasound (EUS)-guided fine-needle aspiration or biopsy of pancreaticobiliary lesions and trans-biliary ductal drainage procedures (eg EUS-guided choledochoduodenostomy and hepaticogastrostomy) are also known to be associated with a risk of hemobilia. Similar to ERCP, these procedures carry a higher risk for hemobilia due to their complexity and because patients needing them often have significant comorbidities.

### 4.1.4 Treatment of malignancy

The treatment of malignancy can itself be a potential cause of hemobilia. Radiofrequency ablation (RFA) used in the treatment of early-stage hepatocellular carcinoma (HCC) has been reported to cause hemobilia. While the cause is unclear, a fistula is ultimately formed between the biliary system and vasculature. The reported incidence of hemobilia following ultrasound-guided percutaneous liver RFA ranges from 0% to 0.5%. Interestingly, with CT-guided RFA, a study of 195 patients found the incidence of post-RFA hemobilia to be as high as 8.2%. There have been also reports of bleeding occurring with non-procedural treatment of malignancies, for example, pharmacotherapy with sorafenib, which has led to hemobilia in patients with HCC invading the biliary tract.

### 4.2 Non-iatrogenic causes

#### 4.2.1 Malignancy

The most common cause of spontaneous hemobilia is primary or metastatic hepatobiliary malignancy. Cholangiocarcinoma, pancreatic adenocarcinoma, gall bladder carcinoma, HCC and metastatic lesions to the liver have all been associated with hemobilia. Malignancies account for approximately 10% of all hemobilia cases. The pathophysiology is believed to be related to the fact that there is an enriched vascular supply yet more friable tissue in malignancy, thereby leading to an increased risk of spontaneous haemorrhage.

Manolakis et al reported a case of hemobilia presenting with Quincke’s triad as the initial manifestation of cholangiocarcinoma but in whom all initial imaging studies, including ultrasound, MRI/ MRCP, MRA and ERCP did not visualize a tumour. Given the high clinical suspicion, a repeat ERCP was performed, which revealed that the bile ducts had been infiltrated with tumour and allowed for visualization of peripapillary blood clots. This particular case highlights the importance of considering hemobilia as a possible indicator of underlying biliary tract malignancy (primary or metastatic).

Similar to cholangiocarcinoma, HCC is a highly vascular tumour and can be responsible for hemobilia with biliary ductal invasion. In a study of 140 patients with HCC by Carella et al, tumour invasion of the bile duct was appreciated in 2.1% on necropsy. There are also numerous case reports of spontaneous rupture of HCC leading to haemorrhagic shock and end-organ dysfunction, and while spontaneous rupture is very rare (<3% reported), this scenario has high reported mortality (>50%). Similarly, metastasis of other malignancies to the hepatobiliary system can cause hemobilia and present with other manifestations such as unexplained biliary obstruction. Cases of hemobilia caused by malignancy can be treated with procedures such as transarterial embolization (TAE). Additionally, when clinically appropriate and technically feasible, emergent hepatic resection has been reported to be a treatment option for patients with tumour bleeding from HCC.

#### 4.2.2 Portal biliopathy

Infrequently, hemobilia may be seen in the setting of portal biliopathy, either with or without preceding biliary tract intervention. Portal biliopathy is caused by hypertension of the periportal (ie choledochal) venous plexus, and often seen in patients with portal vein thrombosis and ensuing portal cavernomas; it manifests radiographically and physiologically as multifocal biliary stenosis caused by enlarged, tortuous, venous structures found encircling the bile duct. Hemobilia in this setting generally needs advanced intervention (eg interventional radiology) as the bleeding is not caused by an abnormality of the biliary epithelium.

#### 4.2.3 Chronic ductal obstruction

Prolonged obstruction of the pancreaticobiliary tract can lead to inflammation, erosion and fistulization with adjacent vascular structures and resultant hemobilia. Intrabiliary clots, which form due to hemobilia, can be mistaken as stones on various imaging studies. However, even when gallstones are present, there can still be concurrent hemobilia, particularly in cases wherein the gallstone
erodes through the cystic artery or other vascular structure or fistulously into the duodenum leading to duodenal outlet obstruction (eg Bouveret’s syndrome).27

Haemosuccus pancreaticus (also known as Wirsungorrhagia), though not technically classified as hemobilia, can occur via a similar mechanism, eg pancreatic inflammation eroding into the splenic artery and causing bleeding into the pancreatic ductal system.48,49 The clinical presentation may also be similar, though jaundice is typically absent, and hyperamylasaemia is seen instead of hyperbilirubinaemia.50

### 4.2.4 Intraductal infection

The most clinically significant cause of infectious hemobilia is ‘tropical hemobilia’, which is due to parasitic infestation of the biliary system. Commonly implicated organisms are the Chinese liver fluke (*Clonorchis sinensis*), roundworms (eg *Ascaris lumbricoides*) and the sheep liver fluke (*Fasciola hepatica*). Echinococcal infections have also been noted to cause hemobilia indirectly via hydatid cysts leading to inflammation of the perivascular tissue, compromise of vessel wall integrity and possible pseudoaneurysm formation. China, Korea and Vietnam have the highest incidence of ascariasis and unsurprisingly have relatively high rates of hemobilia due to infection.27

### 5 | DIAGNOSIS

In patients with a history of recent blunt force or penetrating trauma to the upper abdomen, biliary instrumentation or manipulation (particularly in the setting of clinical and biochemical signs or symptoms of biliary obstruction) or simply an unknown source of bleeding, hemobilia should be considered on the differential diagnosis. Diagnosing hemobilia can be challenging as it is often not suspected due to its uncommon occurrence, particularly in cases with no risk factors such as recent instrumentation or trauma. Imaging can be helpful for making the diagnosis and guiding the choice of therapeutic options, though findings are often nonspecific. Direct visualization via upper endoscopy of either clots or blood exiting the biliary tract essentially confirms the diagnosis of hemobilia (Figure 3). This, along with other imaging modalities, is discussed in further detail below.

#### 5.1 | Computed tomography

Computed tomographic angiography (CTA) of the abdomen can be a useful modality for evaluation of suspected hemobilia (Figure 4). When CTA of the abdomen may be needed will depend on each individual case and presentation and should not be determined by

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![Figure 3](image-url) *Vascular complications include hepatic artery aneurysms, pseudoaneurysms and cholangiovenous or arterio-ductal fistulae. The initial diagnostic study for hemobilia is frequently a CT angiogram. Endoscopic therapy is most often performed as the initial therapy, though if vascular complications or haemodynamical instability is present, interventional radiology (IR) techniques are preferred. If endoscopic therapy does not successfully achieve haemostasis, IR techniques should be attempted, or vice versa, depending on the clinical scenario. Surgical intervention is indicated for biliary obstruction and/ or haemostasis when endoscopic and IR techniques fail or are not available or technically feasible.*
strictly following algorithms of care. The benefits of CTA include its lack of invasiveness, quick results, robust diagnostic performance characteristics and the ability to assess the abdomen intra- and extraluminally.

5.2 | Upper endoscopy and ERCP

Upper endoscopy has also reportedly been used to identify hemobilia cases. A duodenoscope (ie side-viewing scope) can be used to visualize the major papilla as well as assess for clots or other evidence of hemobilia (Figure 5A,B). ERCP can be used to further visualize the biliary tree while also offering possible therapeutic options in patients with hemobilia. Characteristic ERCP findings suggestive of hemobilia include tubular, amorphous or cast-like filling defects with unexplained dilation of the common bile duct or perihilar ductal dilation.

When ERCP findings are equivocal, EUS may be used as an ancillary, non-invasive method for evaluation of vascular abnormalities and blood clots within the biliary tree. EUS can also be used to detect portal biliopathy-related bleeding (eg in the context of portal hypertension with intra- or para-choledochal varices).

5.3 | Angiography

While conventional angiography remains the gold standard for diagnosing and treating hemobilia, it is generally no longer used as a first-line modality due to its invasive nature. When the bleeding vessel has not yet been located despite having undergone non-invasive imaging, the first angiographic study should be a celiac arteriogram with delayed phase imaging to help visualize the hepatic arteries and the portal vein. Patency of the portal vein should be confirmed prior to any planned hepatic artery embolization, as the liver is supplied by both vessels, and performing hepatic artery embolization in the setting of portal vein occlusion can lead to significant hepatic ischaemia. Among patients who are liver transplant recipients, this is of particular importance as allograft survival is directly related to both portal vein and hepatic artery blood flow. Patients with cirrhosis and hereditary haemorrhagic telangiectasia involving the liver are similarly at risk for hepatic ischaemia.

Angiographic evaluation typically is performed in a stepwise fashion in the sense that if coeliac arteriography cannot reveal a clear source of bleeding, then the catheter should be further advanced.
5.4 | Other diagnostic modalities

Less frequently used imaging to help in identifying hemobilia include magnetic resonance cholangiopancreatography (MRCP), abdominal US and surgical exploration. MRCP is a non-invasive alternative to ERCP; however, it lacks the therapeutic options that ERCP can offer and is also more time-consuming for obtaining imaging (compared to other studies such as CT or fluoroscopy). Abdominal US has been used to assess for blood within the gall bladder or enlarged biliary ducts; however, its diagnostic effectiveness is limited, especially in the distal common bile duct/periampullary region and in patients with truncal obesity. As a final option, surgical exploration can be considered if other modalities cannot determine suspected hemobilia.48

6 | TREATMENT

The treatment of hemobilia consists of both achieving haemostasis and maintaining bile flow. The latter is important because the formation of blood clots within the biliary tree can lead to a multitude of complications such as obstructive jaundice, acute cholecystitis, acute cholangitis and acute pancreatitis.9

The approach to treating hemobilia depends on several factors, including the suspected source of bleeding (arterial vs venous), the cause of bleeding (Figure 3), degree of blood loss and haemodynamic stability. All patients should have a type and screen performed, complete blood count, comprehensive metabolic profile and coagulation parameters assessed and close clinical monitoring for signs of haemodynamic instability. Patients who present with minor hemobilia (ie small volume of blood loss without haemodynamic instability) can often be managed conservatively with intravenous fluids and correction of any underlying coagulopathy. Major hemobilia is defined as causing a significant haemoglobin drop or persistent haemorrhage despite supportive care and typically requires endoscopic, interventional radiologic or rarely surgical intervention. Patients with significant haemodynamic instability should generally be taken directly to interventional radiology for hepatic angiogram and embolization or to surgery. If there are signs or symptoms of acute cholangitis and/or biliary obstruction, intravenous antibiotics targeting biliary microflora should be administered. Vasopressors may also be required as part of resuscitative efforts as a bridge to more definitive therapeutic intervention.

6.1 | Conservative management

Minor hemobilia can present in various ways, eg blood-tinged output from a PTBCD or a small drop in haemoglobin with or without transient abnormalities in serum liver tests in the setting of a recent hepatobiliary intervention. Minor hemobilia often results from local tissue irritation and can typically be frequently managed conservatively. In the case of the former example, exchanging the PTBCD with a larger diameter catheter and adjusting its position such that its side holes are not adjacent to potential portal venous transgression sites can usually resolve minor hemobilia through a tamponading effect. When related to the presence of a PTBCD, minor hemobilia ultimately ceases upon maturation of the catheter tract and correction of coagulopathy (if present). A ‘tubogram’, an imaging study in which contrast is injected into the PTBCD tract to visualize its course and patency, may be performed if bleeding does not resolve or if there is impaired biliary drainage due to obstructing clots. If hemobilia continues to persist, embolization of the existing percutaneous tract and creation of a new tract may be considered.57

6.2 | Advanced endoscopic techniques

In the setting of haemodynamically stable hemobilia with no clear source of bleeding or significant vascular abnormalities on initial imaging, ERCP and upper endoscopy (with either a duodenoscope or a clear endcap-outfitted gastroscope) are usually the procedures of choice given their ability to detect and manage both bleeding and biliary obstruction in a minimally invasive fashion.58

There exists a wide variety of endoscopic techniques and accessories that can assist in achieving haemostasis, with the choice of which to implement depending on the cause (eg trauma), location (eg right hepatic duct) and vascular source (eg pericholedochal vein) of hemobilia. One example is post-sphincterotomy hemobilia, which can be caused by injury of the posterior branch of the superior pancreaticoduodenal artery. This scenario can be managed by a variety of methods such as catheter-directed dispersal of diluted epinephrine (1:10,000) over the area of haemorrhage, injecting epinephrine into the adjacent submucosa, electrocautery, fibrin or other sealant usage, endoscopic clipping, balloon tamponade and/or large diameter stent placement (Figure 3).59–65 These techniques are more successful when the site of bleeding is distal, such as at the level of the papilla or ampulla. If hemobilia is caused by a more proximal bleeding source, such as perihilar, other techniques and accessories are often required, such as devices to extract intraductal clots, for example, extraction balloon catheters and retrieval baskets, followed by stent placement, among other options (Figures 3 and 5). It has been reported that application of endobiliary radiofrequency ablation for haemorrhage secondary to malignant hemobilia can be effective in conjunction with an uncovered stent; however, this has been minimally studied.66

Biliary stents have also been to manage hemobilia. Placement of biliary stents has been shown to achieve immediate haemostasis by creating a tamponade effect on the bile duct wall while maintaining luminal patency of the duct, thereby allowing for continued bile flow. Stenting can serve as salvage therapy when other methods fail and also as a bridge to more definitive treatment, such as interventional radiologic or surgical67 Both plastic and metal stents have been used...
successfully in hemobilia. It should be noted, however, that fully covered self-expanding metallic stents (FCSEMS) (Figure 5A, B) appear to have a superior capacity to tamponade active bleeding while ensuring greater patency and have therefore largely supplanted the use of plastic stents in this setting.67-70

Stenting can be performed concurrently with other techniques such as balloon tamponade through the insertion of a dilation balloon catheter into the common bile duct (e.g., as a temporizing measure until blood flow has slowed enough to permit visualization and stent placement as a more definitive treatment).61 In addition, endoscopic nasobiliary drainage also offers unique benefits that can help treat hemobilia but is not routinely performed (mostly due to the associated discomfort, technically cumbersome procedure and ease of inadvertent catheter dislodgment).70

6.3 | Transarterial embolization

Over time the primary cause of hemobilia has transitioned from traumagenic to iatrogenic, with radiologic intervention becoming the gold standard for diagnosis and management of persistent and/or haemodynamically unstable hemobilia. Angiography with TAE should be the initial therapy of choice if initial non-invasive imaging shows prominent arterial extravasation, large arterial aneurysms or pseudoaneurysms, arterio-biliary fistulae and/or intrahepatic or extrahepatic vascular lesions. Reported TAE success rates range between 80% and 100%.71,72 However, as mentioned above, TAE should generally be avoided in patients with liver allografts and portal vein thrombosis given TAE in these scenarios can lead to severe ischaemic liver injury; such patients may preferentially benefit from arterial stenting in lieu of TAE.48

Once a bleeding site has been identified angiographically, super selection of the injured artery via threading of a microcatheter to the target area is performed, followed by TAE using coils. Coiling should be performed in a distal-to-proximal fashion to avoid back bleeding via intrahepatic arterial collaterals.55 Alternatives to coils include Gelfoam and liquid embolic agents such as n-BCA and Onyx copolymer. There have also been case reports of percutaneous injection of thrombin into pseudoaneurysms under ultrasound guidance.73 The method by which TAE is performed depends primarily on the anatomy of the involved vasculature, vessel tortuosity, presence of vasospasm and operator/centre experience. For instance, liquid embolic agents may be helpful in patients with tortuous vessels but require experienced interventionalists due to the risk of (remote) embolization of non-target vessels.74

If selective embolization of the bleeding artery cannot be performed, non-selective embolization of either the right or left hepatic artery can be pursued. Embolization of the main hepatic artery can be performed if the patient is a poor surgical candidate and haemodynamically unstable, but this carries a risk of causing hepatic necrosis.55 If a bleeding source is not identified, it is not recommended to empirically embolize any hepatic arteries due to this risk of necrosis even when portal veins are patent. Furthermore, because vascular supply to the bile ducts is predominantly via the hepatic arteries rather than the portal vein, there is a risk of biliary ischaemia and resultant multifocal ischaemic biliary strictures.8

Possible complications of TAE include hepatic ischaemia, infarction, abscess formation and rarely acute hepatic failure.51 In a study of patients who underwent TAE, 55 of 72 experienced hepatic ischaemia as evidenced by elevated serum liver enzymes, while three experienced focal hepatic infarcts in the distribution corresponding to the embolized arterial rami.72

6.4 | Vascular stenting

An alternative to embolization is the placement of a covered stent that crosses the site of identified vascular injury. Vascular stenting preserves flow through the artery, which is particularly important in liver transplant patients or compromised portal vein flow. The diameter of most hepatic vessels is similar to that of coronary vessels, making coronary stents ideal for this application. Stent diameter should be slightly oversized by about 10%-20% of the diameter of the target vessel and extend approximately 10 mm on either side/end of the site of injury to ensure proper tamponade and adequate coverage.75,76
6.5 | Surgery

Surgical intervention is infrequently required unless endoscopic, endovascular and/or percutaneous therapies have failed. However, it remains a first-line intervention if pseudoaneurysms are infected or compressing other vascular structures. Options for surgical intervention include hepatic artery ligation, pseudoaneurysm excision or hepatic segmentectomy with the potential for concurrent cholecystectomy if cholecystitis is present or the gall bladder is otherwise involved or diseased. While surgery has a high success rate of over 90%, it is also associated with a high mortality rate of up to 10%.8

6.6 | Managing complications of hemobilia and its therapies

Complications that develop due to hemobilia should be managed as they otherwise would be in any other scenario. For example, cholecystitis should be treated with early cholecystectomy.77 Acute pancreatitis, which can occur due to obstruction of the ampulla or main pancreatic duct by blood clots, can be managed medically and in some instances, via ERCP. Biliary strictures can form following hepatic artery embolization as the vascular supply to the biliary tree comes predominantly from the hepatic artery; such strictures generally require treatment with endoscopic and/or percutaneous techniques such as balloon dilation and stenting.8

7 | CONCLUSION

Hemobilia is an uncommon but important cause of GI bleeding to be familiar with. The diagnosis can be challenging due to its uncommon occurrence, especially in cases with no known history of recent biliary tract instrumentation, trauma or malignancy. CT angiography and endoscopy/ERCP have become the preferred initial diagnostic modalities used due to their versatility in excluding other causes of bleeding and their relative safety compared to angiography and surgery. While most cases of minor hemobilia can be treated conservatively or with minimally invasive endoscopic management, major hemobilia, characterized by haemodynamic instability, should be managed by interventional radiology in conjunction with endoscopy/ERCP. Interventional radiographic therapy is primarily via TAE; however, vascular stenting continues to gain traction as a possible alternative due to its ability to ensure preservation of hepatic arterial blood supply. Surgery is reserved as a last resort due to its high mortality rate and invasive nature relative to other approaches. Although the gold standard for initial management remains angiography, advanced endoscopic and radiologic procedures have become an attractive adjunct or alternative, for both the diagnosis and the treatment of hemobilia.

CONFLICT OF INTEREST

No potential conflicts of interest.

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AUTHOR CONTRIBUTIONS

Rani Berry, Alex Zhornitskiy, James Han and James H. Tabibian acquired and selected the figures; Rani Berry, James Han and Alex Zhornitskiy drafted the manuscript; James H. Tabibian provided supervision and critical revision of the manuscript.

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