Case Report
Étude de cas

Traumatic pseudoaneurysm of the hepatic artery after percutaneous liver biopsy and laparoscopic cholecystectomy in a patient with biliary cirrhosis: a case report

Sam T.M. Kwauk, MD, PhD;* Ron Cameron, MB, BCh;* Brent Burbridge, MD;† Roger G. Keith, MD*

Hemobilia is a rare but recognizable complication of percutaneous transhepatic diagnostic or therapeutic procedures. The diagnosis is sometimes difficult because of the time lag between the procedure and the first symptoms, which can be intermittent. A 35-year-old woman had hemobilia after percutaneous Trucut biopsy of the liver followed by laparoscopic cholecystectomy. The diagnosis of hemobilia was made on clinical grounds, and a pseudoaneurysm of the right hepatic artery was detected on selective angiography. The patient was successfully treated with arterial embolization during angiography.

L'hémobilie est une complication rare mais reconnaissable d'interventions diagnostiques ou thérapeutiques transhépatiques percutanées. Le diagnostic est parfois difficile à poser à cause du temps écoulé entre l'intervention et l'apparition des premiers symptômes, qui peuvent être intermittents. Une femme âgée de 35 ans avait de l'hémobilie après une biopsie percutanée du foie suivie d'une cholécystectomie par laparoscopie. On a diagnostiqué une hémobilie pour des raisons cliniques et détecté un pseudo-anévrisme de l'artère hépatique droite au moyen d'une angiographie sélective. La patiente a été traitée avec succès par embolisation artérielle au cours de l'angiographie.

The term hemobilia was introduced by Sandblom[1] to describe hemorrhage into the biliary tract. The most common cause of this abnormal communication between a blood vessel and the biliary tract is trauma, either accidental or iatrogenic. Diagnostic or therapeutic procedures such as liver biopsy, transhepatic cholangiography or drainage, and hepatic or biliary surgery may cause hemobilia.[2]

The treatment of hemobilia depends on the cause of the hemorrhage. For most of the iatrogenic causes, arterial embolization is the first choice.

Case report

A 35-year-old woman presented with a 6-month history of episodic nausea, vomiting and epigastric pain radiating to the back. The pain was aggravated by the intake of food. The patient was admitted on 4 previous occasions to a local hospital, and laboratory investigations gave the following results: a hemoglobin level of 144 g/L, a platelet count of 557×10^9/L, a total serum bilirubin level of 5 µmol/L (normal 3 to 22 µmol/L), an alkaline phosphatase level of 230 U/L (normal 30 to 120 U/L), an alanine aminotransferase level of 37 U/L (normal 0.5 to 43 U/L), an aspartate aminotransaminase level of 33 U/L (normal 10 to 40 U/L) and a γ-glutamyl transpeptidase level of 247 U/L (normal 1 to 50 U/L). The partial thromboplastin time and the international normalized ratio for coagulation were normal, and the antimitochondrial antibody titre was 1:320 (normal less than 1:20). Abdominal ultrasonography showed no gallstones; however, the gallbladder did not contract during cholecystokinin challenge. Findings on gastroscopy were normal.

Percutaneous liver biopsy was performed using 2 passes with a 14.9

*From the *Department of Surgery and †Department of Radiology, Royal University Hospital, University of Saskatchewan, Saskatoon, Sask.*

Accepted for publication Sept. 9, 1997

***Correspondence to**: Dr. Sam T.M. Kwauk, Department of Surgery, New York Downtown Hospital, 170 William St., New York NY 10003, USA*

Tru-cut needle (Travenol Laboratories, Norton Grove, NJ). The biopsies obtained revealed chronic inflammatory change, and an active process associated with granulomatous change supported a diagnosis of primary biliary cirrhosis.

The patient presented approximately 10 days after her liver biopsy with melena and a hemoglobin level of 129 g/L. Computed tomography shortly thereafter revealed a lesion, measuring 7 × 4 × 5 cm, in the right lobe of the liver suggestive of a hematoma. Endoscopic retrograde cholangiopancreatography (ERCP) showed normal pancreatic and common bile ducts.

Laparoscopic cholecystectomy was carried out for the presumed diagnosis of biliary dyskinesia. Preoperative blood work revealed the following levels: hemoglobin 95 g/L, total serum bilirubin 12 μmol/L, serum alkaline phosphatase 497 U/L, serum alanine aminotransferase 60 U/L, serum aspartate aminotransferase 41 U/L and serum amylase 27 U/L. Repeat CT before cholecystectomy (1 month after the liver biopsy), revealed complete resolution of the intrahepatic hematoma. At operation, the gallbladder was reported to be acutely inflamed, associated with dense vascular adhesions between it and the duodenum. The cystic duct was reportedly divided between 3 clips, and a small anterior and large posterior cystic artery each divided between 3 clips. The liver was reported as appearing normal, and essentially there was no blood loss. Pathological assessment of the gallbladder revealed chronic cholecystitis. The patient's immediate postoperative course was uncomplicated. However, approximately 2 weeks after laparoscopic cholecystectomy, the pain recurred with increased severity, and the pattern changed from episodic to constant. Two weeks later, the patient was admitted to Royal University Hospital for further investigation.

On admission, the patient appeared jaundiced and in mild distress. Her heart rate was 113 beats/min and the blood pressure 151/91 mm Hg. Findings on cardiac and chest examinations were within normal limits. Abdominal examination revealed localized tenderness in both the right upper quadrant and the epigastrium. There was no voluntary guarding. The findings of the remainder of the physical examination were within normal limits.

Laboratory investigation revealed the following values: leukocyte count 11 × 10^9/L, hemoglobin 109 g/L, total serum bilirubin 73 μmol/L (normal range 2 to 22 μmol/L), serum alkaline phosphatase 803 U/L (normal range 30 to 110 U/L), serum alanine aminotransferase 247 U/L (normal range 0 to 37 U/L), serum aspartate aminotransferase 239 U/L (normal range 10 to 35 U/L), serum γ-glutamyl transferase 498 U/L (normal range 7 to 33 U/L) and serum amylase 668 U/L (normal range 30 to 115 U/L). Hepatitis A and B serologic tests were negative and the antimitochondrial antibody titre was 1:320. Chest and abdominal x-ray films were normal. Abdominal ultrasonography revealed a 2 × 5-cm fluid collection in the gallbladder fossa with a normal intra- and extrahepatic biliary tree. A CT scan similarly showed a small collection in the gallbladder fossa with no evidence of biliary dilatation and a normal pancreas (Fig. 1). Hepatobiliary imaging showed mild intrahepatic cholestasis with minimal impairment of hepatocellular function. Liver/spleen scanning showed minimal inhomogeneity, consistent with primary biliary cirrhosis. Nothing abnormal was found on upper gastrointestinal endoscopy and ERCP showed normal pancreatic and common bile ducts.

Narcotic analgesics gradually relieved the patient's epigastric pain, and her jaundice began to resolve. She was

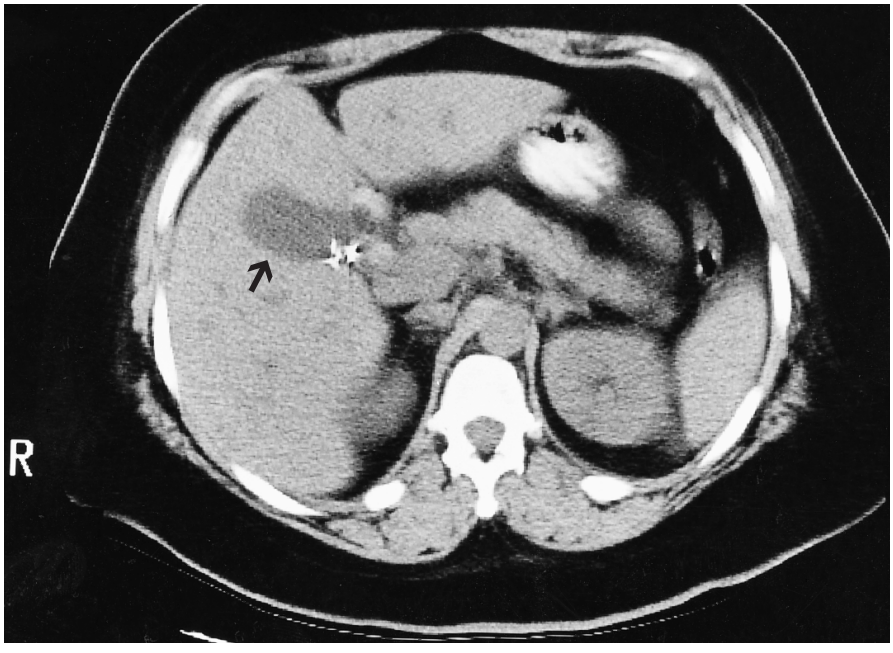

FIG. 1. Computed tomography scan shows fluid collection (arrow) in the gallbladder fossa with no evidence of biliary obstruction.

discharged 10 days after admission, only to be readmitted 10 days later with more severe epigastric pain. Laboratory investigations again showed abnormalities of all liver enzymes with a serum amylase level now within the normal range. Repeat CT without contrast showed intrahepatic biliary dilatation. The collection in the gallbladder fossa was drained percutaneously and continued to drain approximately 60 mL/d of bile. Radiography through the percutaneous drainage tube revealed contrast material in the common bile duct and later on in the duodenum, which suggested a communication between the collection and the biliary tree. A large filling defect suggestive of blood clot was noted in the common bile duct (Fig. 2). The patient noted a change in the pain pattern from constant to intermittent, associated with bilious vomiting. Two weeks after the percutaneous drainage blood was seen draining from the tube in the gallbladder fossa and the patient's hemoglobin level decreased to 79 g/L, necessitating blood transfusion.

Clinically, the patient had hemobilia. Angiography showed a pseudoaneurysm of a small branch of the right hepatic artery in the cranial aspect of the right hepatic lobe (Fig. 3). Initial selective embolization of the feeding vessel to the pseudoaneurysm was not successful, but embolization of the major tributary of the right hepatic artery by a coil was successful (Fig. 4). Repeat contrast study through the percutaneous tube in the gallbladder fossa revealed no further communication between the gallbladder fossa and the biliary tree (Fig. 5). Clinically, the patient's pain also resolved, and she was discharged on postoperative day 11.

At the last follow-up, 4 months after discharge, she had no abdominal pain or jaundice and her hemoglobin level was normal.

Discussion

Hemobilia is defined as hemorrhage into the biliary tract. In an initial report by Sandblom,[1] all patients in the series had traumatic accidents to the liver, resulting in either contusion or rupture of the hepatic parenchyma. However, more recently there has been a shift in the cause of hemobilia from accidental to iatrogenic.[3] Yoshida, Donahue and Nyhus[4] reviewed the world literature from 1982 to 1985 and found that 42% of the 103 patients had iatrogenically induced hemobilia whereas accidents accounted for only 20%. This shift is likely to continue because of the increase in the number of percutaneous liver biopsies, transhepatic cholangiography and drainage procedures[5]

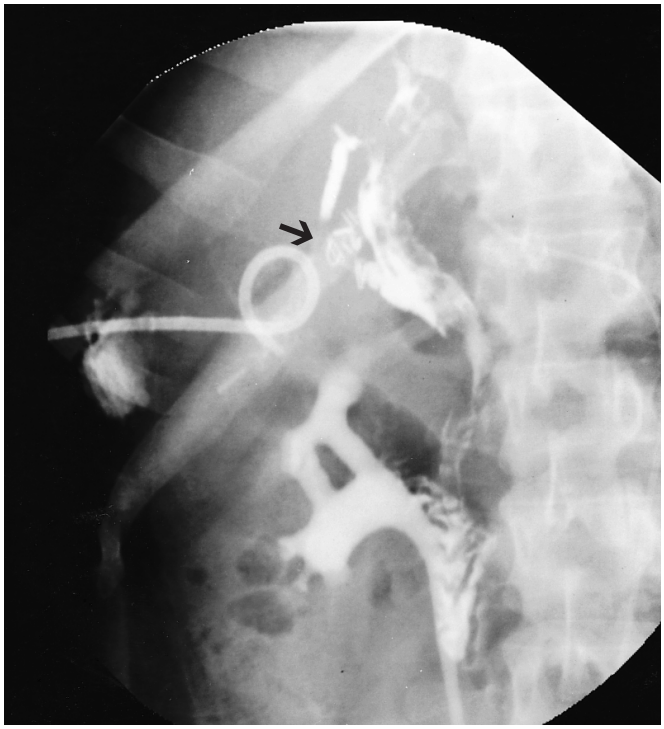

FIG. 2. Contrast study through the percutaneous tube demonstrating communication of the biliary tract with the fluid collection. The common bile duct was filled with clots.

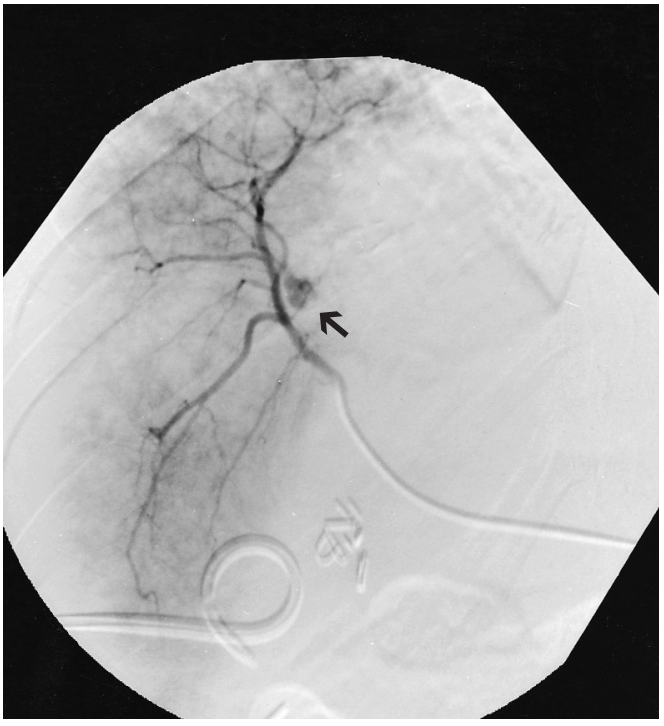

FIG. 3. Selective angiogram showing pseudoaneurysm of a branch of the right hepatic artery.

and placement of the hepatic artery catheter for regional chemotherapy.

The incidence of communication between a blood vessel and the biliary tract after percutaneous liver biopsy ranges from 0 to 10%.[2] Most of the affected patients were asymptomatic and the condition resolved spontaneously.[2] In 1992, Lichtenstein, Kim and Chopra[6] reviewed the literature and found only 32 cases of hemobilia related to percutaneous liver biopsy, and the mean interval from the time of biopsy to the onset of symptoms was about 5 days. Furthermore, the authors proposed that possible risk factors for hemobilia were the presence of thrombocytopenia, coagulopathy, the type of needle used and the number of passes made.

Hemobilia has been reported after cholecystectomy, performed open[7] or laparoscopically.[8] The site of the pseudoaneurysm is most likely the right hepatic artery, and bleeding may occur several months after the surgery. Most occurred in association with cholecystectomies where the anatomy was difficult to define, and in the presence of excessive bleeding. Deziel and associates[9] surveyed 77 604 cases of laparoscopic cholecystectomy and found only 1 case of hemobilia. Our patient had both percutaneous hepatic biopsy and laparoscopic cholecystectomy. An intrahepatic hematoma with associated melena was evident on CT after the liver biopsy. Therefore this represents the most likely etiology for the hemobilia. The laparoscopic cholecystectomy was reported to have been associated with essentially no blood loss yet still must be considered as a possible contributing factor.

The standard presentation of hemobilia is gastrointestinal bleeding, transient right upper quadrant pain and jaundice. This is noted in most patients with hemobilia secondary to percutaneous hepatic biopsy but in less than 40% of patients with hemobilia from other causes.[4] The condition of our patient was complicated by several factors: primary biliary cirrhosis, acalculous cholecystitis and laparoscopic cholecystectomy. In retrospect, the right upper quadrant pain initially may have been secondary to biliary dyskinesia, with worsening of her symptoms after percutaneous biopsy and associated intrahepatic hematoma. Laparoscopic cholecystectomy relieved her pain for 2 weeks, but the pain returned with increased severity and associated jaundice and recurrence of her hemobilia.

The diagnosis of hemobilia must be based on acute awareness of the condition. Gastroscopy can reveal other causes of upper gastrointestinal bleeding. Occasionally blood can be seen flowing from the ampulla of Vater, but usually the bleeding is intermittent. ERCP may demonstrate the presence of clots in the common bile duct.[10] Selective angiography is the investigation of choice. The test localizes the aneurysm, and occasionally delayed films will demonstrate contrast in the common bile duct outlining the clots.

In our patient, a contrast study through a drain placed percutaneously in the gallbladder fossa demonstrated a communication between the collection

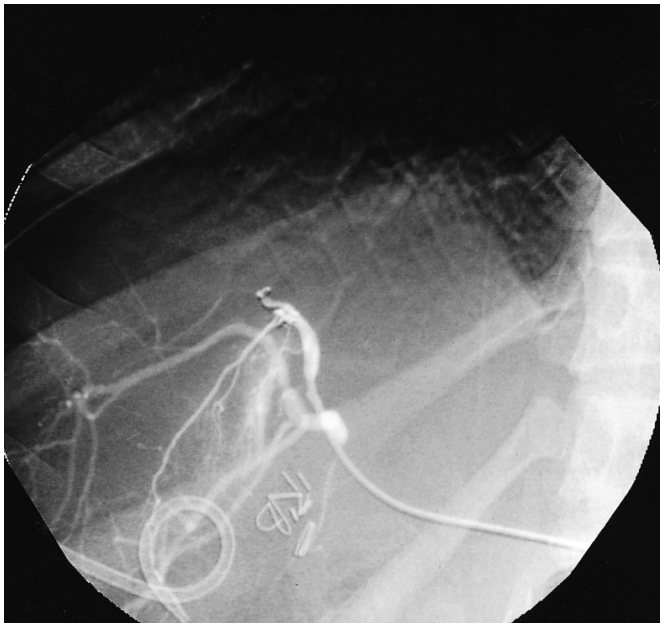

FIG. 4. Selective angiogram of the right hepatic artery after embolization with 2 coils.

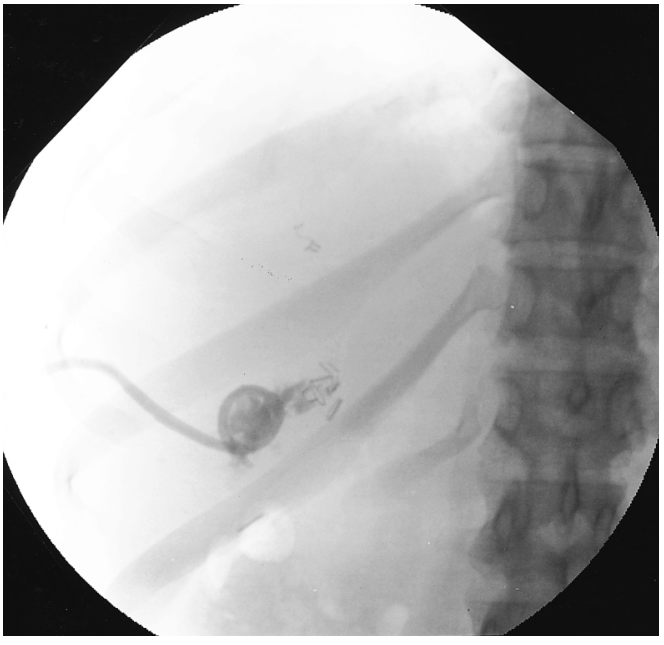

FIG. 5. Contrast study through the percutaneous tube demonstrating no communication between the biliary tract and the drainage tube.

in the fossa and the biliary tract, and the clots in the common bile duct were outlined during the delayed film. The leakage of bile into the gallbladder fossa was probably a combination of increased pressure in the biliary tract secondary to the clots intermittently blocking the common bile duct and disruption of the liver parenchyma at the time of laparoscopic cholecystectomy.

The treatment of hemobilia depends on the etiology. In an initial series of 9 patients with hemobilia secondary to trauma, treatment ranged from observation with spontaneous resolution to operative evacuation of the hepatic hematoma.[1] Spontaneous resolution of hemobilia secondary to percutaneous liver biopsy has been documented by Ball and associates.[10] Baker and associates[11] believed that the placement of a nasobiliary tube would relieve the jaundice, promote lysis of the clot and bring about cessation of bleeding. Angiography plus embolization has the advantage of being both diagnostic and therapeutic. Material used for embolization includes Gelfoam pledgets, stored human dura mater[12] and balloons.[5] Lee, Tasman-Jones and Wattie[13] described the intra-arterial infusion of epinephrine and propranolol[12] to reduce the flow into the aneurysm. Potential complications of angiographic embolization are low but may be serious. They include liver ischemia, infarction and embolization of nontargeted tissue. Surgery is usually reserved for failed angiographic embolization or recurrent aneurysm. The procedure involves either ligation of the hepatic artery or partial hepatectomy.[6]

References

1. Sandblom P. Hemorrhage into the biliary tract following trauma: "traumatic hemobilia." *Surgery* 1948;24:571-86.
2. Schiff L, Schiff ER. *Diseases of the liver*. 7th ed. Philadelphia: JB Lippincott; 1993. p. 1489-98.
3. Sandblom P. Iatrogenic hemobilia [review]. *Am J Surg* 1986;151(6):754-8.
4. Yoshida J, Donahue PE, Nyhus LM. Hemobilia: review of recent experiences with a worldwide problem. *Am J Gastroenterol* 1987;82(5):448-53.
5. Mitchell SE, Shuman LS, Kaufman SL, Chang R, Kadir S, Kinnison ML, et al. Biliary catheter drainage complicated by hemobilia: treatment by balloon embolotherapy. *Radiology* 1985;157(3):645-52.
6. Lichtenstein DR, Kim D, Chopra S. Delayed massive hemobilia following percutaneous liver biopsy: treatment by embolotherapy. *Am J Gastroenterol* 1992;87(12):1833-8.
7. Jeans PL. Hepatic artery aneurysms and biliary surgery: two cases and a literature review [review]. *Aust N Z J Surg* 1988;58(11):889-94.
8. Stewart BT, Abraham RJ, Thomson KR, Collier NA. Post-cholecystectomy haemobilia: Enjoying a renaissance in the laparoscopic era? *Aust N Z J Surg* 1995;65(3):185-8.
9. Deziel DJ, Millikan KW, Economou SG, Doolas A, Ko S-T, Airan MC. Complications of laparoscopic cholecystectomy: a national survey of 4292 hospitals and analysis of 77,604 cases. *Am J Surg* 1993;164:9-14.
10. Ball TJ, Mutchnik MG, Cohn GM, Burrell M. Hemobilia following percutaneous liver biopsy. *Gastroenterology* 1975;68(5 Pt 1):1297-9.
11. Baker AR, Corlett SK, Cookson JB, Carr-Locke DL. Hemobilia treated by nasobiliary catheterization. *Am J Gastroenterol* 1987;82(8):783-5.
12. Fagan EA, Allison DJ, Chadwick VS, Hodgson HJ. Treatment of haemobilia by selective arterial embolisation. *Gut* 1980;21(6):541-4.
13. Lee SP, Tasman-Jones C, Wattie WJ. Traumatic hemobilia: a complication of percutaneous liver biopsy. *Gastroenterology* 1977;72(5 Pt 1):941-4.

Books and Other Media Received
Livres et autres documents reçus

This list is an acknowledgement of books and other media received. It does not preclude review at a later date.

Cette liste énumère les livres et autres documents reçus. Elle n'en exclut pas la critique à une date ultérieure.

Minimal Access Surgery in Oncology. Edited by James G. Geraghty, Howard L. Young and Jonathan M. Sackier. Associate editors: H. Stephan Stoldt and Riccardo A. Audisio. 173 pp. Illust. Greenwich Medical Media Ltd., London, UK; Oxford University Press Canada, Toronto. 1998. Can$187.95. ISBN 1-900151-022

Wounds: Biology and Management. Edited by D.J. Leaper and K.G. Harding. 198 pp. Illust. Oxford University Press, New York; Oxford University Press Canada, Toronto. 1998. Can$175.50. ISBN 0-19-262332-X