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Hepatic artery pseudoaneurysm and hemobilia following laser laparoscopic cholecystectomy

A case report

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Abstract. This report describes injury to the hepatic artery with pseudoaneurysm formation and hemobilia following the use of laser-assisted dissection to perform laparoscopic cholecystectomy.

A 57-year-old woman was referred emergently 2 weeks after laser laparoscopic cholecystectomy with upper abdominal pain, upper gastrointestinal bleeding, and jaundice. A selective hepatic arteriogram showed a right hepatic artery pseudoaneurysm which was embolized. Two weeks later the patient had recurrent hemobilia as the result of blood flow restoration in the pseudoaneurysm and a fistula to the cystic duct remnant. She was treated with two additional embolizations and direct injection of the aneurysm with thrombogenic material. Follow-up at 2 years showed no further recurrence.

Since the laser has never been shown to have advantages over electrocautery, its use during laparoscopic cholecystectomy is difficult to justify.

Key words: Laparoscopy – Laser – Hepatic artery pseudoaneurysm – Hemobilia

Laparoscopic cholecystectomy has been associated with various intraabdominal complications specifically related to biliary surgery, such as intraabdominal abscess, bile leak, bleeding, and injury to the common bile duct. In addition, laparoscopy-associated injuries, such as visceral and vascular injuries, pneumothorax, subcutaneous and mediastinal emphysema and "loss" of the gallbladder in the peritoneal cavity have occurred [8, 9]. This report describes a unique complication resulting from the use of laser-assisted dissection to perform cholecystectomy. Specifically, the videotape showed the laser beam applied in Calot's triangle. The complication likely represents a "past-pointing" injury to the hepatic artery.

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Case study

A 57-year-old woman underwent a seemingly uneventful laser-assisted laparoscopic cholecystectomy in a community hospital. On postoperative day 6 she began experiencing sharp epigastric pain radiating into the back. On physical examination she was pale, cool, clammy, and diaphoretic. She had tachypnea, bradycardia, and hypotension. Abdominal examination was unremarkable, but she had grossly bloody stool. Laboratory tests included Hct 27%, alkaline phosphatase 233 U/L, SGOT 605 U/L, and LDH 727 U/L. Total bilirubin was normal. Upper and lower gastrointestinal tract endoscopy, abdominal sonogram, CT scan, and 99m-technetium-labeled erythrocyte scan failed to provide a diagnosis. Also, endoscopic retrograde cholangiopancreatography was normal. One week later, the patient had another episode of bloody stool and elevation of liver enzymes and required blood transfusions. Abdominal CT scan at that time revealed a low-density mass surrounding the left liver lobe suggestive of a subcapsular fluid collection, possibly representing a subacute hematoma. An arteriogram revealed a replaced right hepatic artery arising from the superior mesenteric artery. There was a large localized extravasation of contrast at the region of the gallbladder fossa representing a 4.5-cm pseudoaneurysm of the right hepatic artery. On postoperative day 18 she was transferred to the Hospital of the University of Alabama at Birmingham.

Upon admission her serum bilirubin was 5.7 mg/dl with elevation of all liver enzymes. Angiography again demonstrated the pseudoaneurysm arising at the first bifurcation of the replaced right hepatic artery (Fig. 1). There was no detectable communication with bile ducts or portal vein. The right hepatic artery and both branches were successfully occluded with Gianturco coils soaked in thrombin.

On postoperative day 30 she again developed severe abdominal pain, hypotension, and hematochezia. Repeat arteriogram revealed filling of the pseudoaneurysm from the left hepatic artery through the

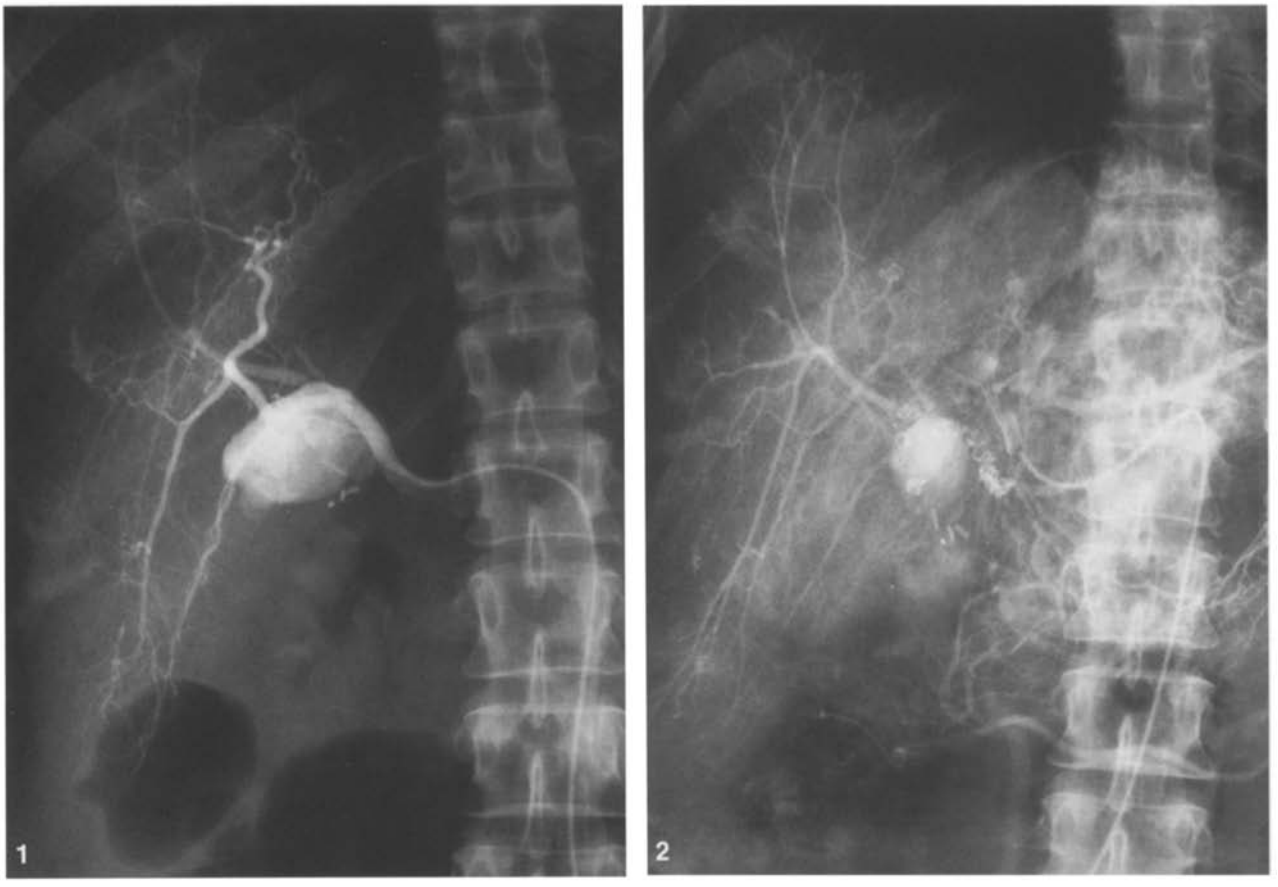

Fig. 1. Pseudoaneurysm of the replaced right hepatic artery

Fig. 2. After initial embolization, recurrence of the pseudoaneurysm with filling from the left hepatic artery

well-developed intrahepatic collateral vessels (Fig. 2). The replaced right hepatic artery remained occluded. The left hepatic artery was then embolized with Gelfoam pledgets and a prominent branch was selectively catheterized and occluded with a coil. Nevertheless, there was persistent filling of the pseudoaneurysm through the proximal branches of left hepatic artery which the embolization failed to occlude. The pseudoaneurysm was then punctured directly and injected with thrombin solution. A final hepatic arteriogram showed no filling of the pseudoaneurysm.

On postoperative day 44 she again developed epigastric pain and bloody stools. On arteriogram, the previously occluded pseudoaneurysm had reestablished patency and in addition the communication with the biliary tree through the cystic duct remnant was now detectable (Fig. 3). Six vascular coils soaked in thrombin were placed into the pseudoaneurysm, followed by injection of liquid polymer glue. The pseudoaneurysm appeared to be completely thrombosed on abdominal ultrasound 3 days later.

At 2 years' follow-up the patient was clinically well. Doppler ultrasound indicated persisting thrombosis of the pseudoaneurysm.

Discussion

Hepatic artery aneurysms are relatively rare. They can be either true or false and most are located extrahepatically. Atherosclerosis is the most prevalent etiology, followed by trauma, medial degeneration, and inflammatory lesions [10]. Iatrogenic injuries may account for up to one-third of cases of hepatic artery false aneurysms. Hepatobiliary trauma and operations on the hepatic artery are other significant contributors to their occurrence [10, 11]. The well-documented natural history of hepatic artery aneurysms is one of continuous enlargement and eventual rupture. Most aneurysms of the hepatic artery represent an incidental finding during the investigation of aortic vascular disease or hemobilia. Often, the first and the most dramatic clinical presentation is rupture [7, 11, 14]. Rupture can occur into the free peritoneal cavity with hemoperitoneum, into a hollow viscus with upper gastrointestinal bleeding, into a vein with formation of arteriovenous fistula, or into the biliary tree with hemobilia [15]. Following rupture, a mortality rate as high as 35% has been observed [10]. Rupture of an hepatic artery aneurysm into the biliary tree accounts for a significant number of cases with hemobilia [3].

Other etiologic factors playing an important role in causing hemobilia are iatrogenic injuries, accidental trauma, inflammation, gallstones, tumors, vascular disorders, and coagulopathies [12, 17]. The incidence of iatrogenic hemobilia is increasing due to more invasive diagnostic techniques and aggressive surgical interventions. Iatrogenic disease now accounts for 40.8% of instances of hemobilia. The most common causes are

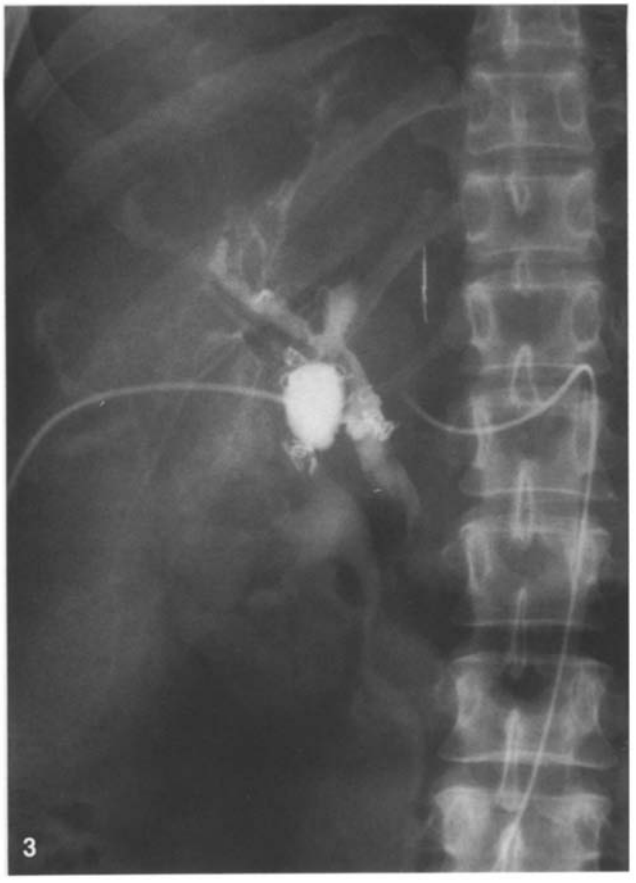

Fig. 3. Opacification of the biliary tree through the cystic duct remnant after injection of contrast medium directly into the pseudoaneurysm

transhepatic cholangiography, percutaneous transhepatic biliary drainage, needle liver biopsy, endobiliary prosthesis, and hepatobiliary surgery [2, 17]. Symptoms of hemobilia include gastrointestinal bleeding (with melena in 90% and hematemesis in 60%), biliary colic in 70%, and jaundice in 60%. These events constitute the pathognomonic triad of hemobilia [13]. Major hemobilia with profuse hemorrhage demanding emergency measures is uncommon. Minor hemobilia with clot formation in the ducts is more common and may cause diagnostic confusion [12]. The diagnosis of hemobilia in some cases may present certain difficulties due to the intermittent nature of hemobilia. The first diagnostic measure usually is gastrointestinal endoscopy, which is useful in ruling out other bleeding sources. It may be combined with ERCP, which sometimes reveals clots in the ducts [13]. Computerized tomographic scans and sonography can be helpful if the bleeding source is a sizable lesion, but they also can provide misleading information [5, 17]. Nuclear scanning may occasionally be an efficient way of establishing the diagnosis during actual bleeding events [13]. Selective hepatic arteriography remains the single most accurate and helpful diagnostic test in evaluation of patients with hemobilia [2, 3, 13]. Arteriography alone can define and localize the arterial abnormality causing hemobilia. Although passage of contrast medium into the biliary ducts during hepatic arteriography usually is not seen [2], the major sources of bleeding in most cases will be revealed by showing the displacement of vessels around a liver mass or by filling a hepatic artery aneurysm [13]. It is important to perform hepatic angiography by systematic filling of the celiac trunk and superior mesenteric artery since false-negative results of hepatic arteriography can be due to a replaced right hepatic artery [2].

Angiography with arterial embolization has also become the preferred treatment of hemobilia. It is often more reliable, easier, and safer than ligature of the hepatic artery or its branches or liver resection [3, 11]. Sometimes repeat embolization is required. Direct pseudoaneurysm injection with thrombogenic material may also be considered, especially in recurrent cases [3]. The success rate of embolization has been 81–96% [5, 11]. Hepatic artery ligation or resection of the aneurysm can also be successful, especially when the source of bleeding is located extrahepatically [14]. Serious complications can occur after either procedure. Liver infarction with necrosis and abscess formation, liver dysfunction, biliary strictures, and fistulas can occur in the postoperative period [1, 2].

Hepatic artery pseudoaneurysm and hemobilia as complications of cholecystectomy are very rare [3, 7], especially when compared to the more typical complications, such as: intraabdominal abscesses, wound infection, bile leak, intraperitoneal bleeding, and iatrogenic injury to the common bile duct [8, 9]. Today, laparoscopic cholecystectomy is one of the most commonly performed surgical procedures and has the potential to achieve a very low rate of morbidity and mortality [8].

The application of laser technology in endoscopy and laparoscopic surgery further attracted attention to those endeavors. However, it has sparked debate as to whether laser-assisted dissection should be used during laparoscopic cholecystectomy [4, 16]. After initial great enthusiasm for using lasers, there have been doubts about benefits of this technology. Laser-assisted laparoscopic cholecystectomy has increased the cost of medical care ($500–546 greater) as compared to conventional electrocautery [6, 16]. It has been also associated with increased operative time, blood loss, and incidence of perforated gallbladders [6]. Although there have been no major prospective, randomized, controlled trials, laser cholecystectomy seems to be associated with an increased incidence of infectious complications and injuries to the common bile duct [4]. Injuries to the portal vein and vena cava have also been observed [4]. The "past-pointing" injury is a documented disadvantage of laser and can contribute to increased morbidity [6].

This experience has demonstrated that misuse of new technology may lead to new complications. Since laser-assisted dissection has never been shown to have advantages over electrocautery, its use during laparoscopic cholecystectomy is difficult to justify.

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