Severe Hemobilia from Hepatic Artery Pseudoaneurysm after Laparoscopic Cholecystectomy: A Case Report and Review of Literature

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Abstract

Background: Hemobilia is a rare, jeopardizing complication of laparoscopic cholecystectomy coming upon patients generally within 4 weeks from surgery. The first line management is angiographic coil embolization of hepatic arteries, which checks the majority of bleedings whereas in a minority of cases, a second embolization or even laparotomy is needed.

Case presentation: We describe the case history of a patient who had laparoscopic cholecystectomy complicated three weeks later by massive hemobilia. The cause of hemorrhage was a pseudoaneurysm of a right hepatic artery branching off the superior mesenteric artery. This complication was managed successfully by one stage angiographic embolization with full recovery of the patient.

Keywords: Hemobilia, Laparoscopic cholecystectomy, Angiographic embolization, Thermal damage, Ultrasonic dissection.

INTRODUCTION

Severe hemobilia complicating laparoscopic colecystectomy (LC) is a rare, unpredictable, life-threatening vascular complication commonly occurring within four weeks from surgery. In the literature, more than 60 cases have been reported by now. Pre-existing aneurysms and postsurgical pseudoaneurysms of hepatic arteries are the cause of hemobilia in 10% of cases. LC related iatrogenic pseudoaneurysms of right hepatic artery (RHA) account for around 60% of cases, of common hepatic artery for around 30%, of cystic artery for around 10%. Pseudoaneurysms are often close to surgical clips and may reach 7 cm in size; bile duct leaks may be associated, but clear visualization of presence of an arteriobiliary fistula by imaging radiologic techniques is seldom obtained. In more than 80% of cases, transarteriographic embolization (TAE) is the first and definite treatment; in some cases re-embolization is necessary, while open or laparoscopic surgery ought to be chosen only in case of unsuccessful coil embolization or when embolization is impossible to accomplish. The pathogenesis of this uncommon but sometimes fatal complication still remains unclear. Mechanical or thermal injuries have been considered responsible, but at the moment precise suggestions to prevent hemobilia after LC are still lacking. We report the clinical history of a 55-year-old woman who presented severe hemobilia with anemia three weeks following a LC with uneventful immediate postoperative course.

CASE PRESENTATION

A 55-year-old woman from Eastern Asia who had been living in Europe for many years underwent LC for cholecystitis. Her past medical history included only asthma, no previous laparotomy. She had been suffering from abdominal pain for five months and 20 days before LC. She was admitted to a medical unit for jaundice where abdominal percutaneous ultrasound examination showed the gallbladder was thick walled (9 mm) with an obstructing gallstone impact in the infundibulum without dilation of intra- and extrahepatic bile ducts. The last time she had been to her native country was one year before. Biochemical tests demonstrated that the alanine aminotransferase (AST) level was within the normal range of 3 to 45 U/L, the total bilirubin level was 4 mg/dl (nonconjugated bilirubin 3.3 mg/dl), coagulation tests and platelets were normal. The markers of hepatitis B and C were negative, the white blood cells count was normal (8.000/mmc), the eosinophiles count was normal and...
Entamoeba histolytica was absent in stool. Consequently, no infectious disease was found and the cause of jaundice remained unexplained. Endoscopic percutaneous cholangiopancreatography (ERCP) with endoscopic papillotomy had definitely ruled out obstruction of the biliary tree while laboratory tests confirmed the persistence of nonconjugated bilirubin values comprised between 3 and 3.5 mg/dl, insofar as her jaundice was attributed to Gilbert's disease. Besides, she became asymptomatic in a fortnight and was discharged. Elective LC was scheduled, but another 20 days later she complained again of abdominal pain in the upper right quadrant and was admitted to our surgical unit where we decided to perform LC in emergency. Because the walls of gallbladder were thick and cohesive, dissection by monopolar coagulation from liver bed was demanding and took longer time than usual, although no intraoperative complication occurred. After excision of the gallbladder, an intraoperative cholangiography was carried out by laparoscopy confirming the complete patency and normality of the biliary tree, and the absence of stones in bile ducts. The early postoperative course of operation was uneventful and the patient was discharged five days after surgery. The histologic examination was consistent with acute inflammation arisen in the context of lithiasis chronic cholecystitis. Oral feeding continued at home, the patient remained asymptomatic for two weeks until she referred a mild epigastric pain irradiated to the right quadrant, although she did not see a doctor. One week later she experienced sudden hypotension with melena and was admitted to our emergency service. Blood pressure was 100/60 mm Hg, pulse rating was 86 beats/minute, hemoglobin level was 8 gr/dl, hematocrit level 23%, white blood count was 9.700/mmc, alanine aminotransferase (ALT) level was increased to 838 U/L (normal values 0-40), alkaline phosphatase level was within the normal ranges of 35 to 129 U/L, coagulation tests and platelets were normal, total bilirubin level was 3.5mg/dl. Digestive endoscopy showed the presence of blood in the upper gastrointestinal tract without evidence of ulcers or other diseases causing bleeding from stomach or duodenum. Abdominal computed tomography (CT) showed a small hematoma of 3 cm in the gallbladder bed with no hemoperitoneum or any other peritoneal fluid collection and iatrogenic pseudoaneurysm of RHA beside titanium clips sized 4 mm without arterial blushing. Resuscitation with transfusional support (3 units of packed red blood cells) allowed the patient to reach hemodynamic stability then she was sent to our surgical ward. Hemoglobin level increased to 12 gr/dl and hematocrit level to 36%. We excluded surgical primary repair because of the high risks related to possible misinterpretation of anatomy after three weeks of local inflammation. Further intermittent episodes of melena occurred in the following days with hemodynamic stability and minimal decrease of Hb and Ht levels. White blood cells count was 11.000/mmc, alanine aminotransferase (ALT) level lowered to 192 U/L and aspartate aminotransferase to 141 U/L while the total bilirubin level raised to 12 mg/dl with 2.6 mg/dl of nonconjugated bilirubin. Since the patient persisted stability in her hemodynamic parameters without fever or abdominal pain and tenderness, we planned to perform angiography only in case of rebleeding, so much more because CT had not shown any arterial blushing which could make angiography inconclusive. The patient underwent ERCP that demonstrated a biliary leak in the gallbladder bed at the level of biliary branch for the V segment, therefore a nasobiliary drainage (NBD) was placed. Two days later occurred another episode of severe melena with hemodynamic instability, hence transfemoral angiography was performed revealing the presence of pseudoaneurysm sized 2 cm sited on a replaced RHA with extravasation between the V and VIII segmental branches (Figs 1A and B) RHA was an arterial branch arising from the superior mesenteric artery; TAE was achieved by filling the entire artery and pseudoaneurysm with coils of 3 and 4 mm (Fig. 2). The patient had an uneventful clinical course without rebleeding, NBD was removed and she started oral intake. CT scan proved revascularization of the right hepatic arterial branches with no ischemia of right liver lobe. The patient was discharged two weeks later with no impairment of liver function tests and a magnetic resonance cholangiography showed a normal biliary tree.

**DISCUSSION**

The case, herein presented of LC-related hemobilia, has been the only one we have registered over the last 10 years accounting for 0.001% of patients with acute cholecystitis operated on in emergency (within 72 hours of admission) and including elective surgery accounting for 0.0003% of all the patients undergoing LC over the same span. Hemobilia complicating LC has become a well-known serious event reported in a plenty of issues. The onset of symptoms and signs is within four weeks from LC in 80% of cases, and only in three cases this complication has occurred one year after surgery or even later. Upper gastrointestinal bleeding with melena is the commonest sign of hemobilia and observed in 90% of cases whereas abdominal pain is
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frequently false aneurysms of common hepatic artery or cystic artery are found. In the present case, small pseudoaneurysm of RHA arising from the superior mesenteric artery was the cause of hemobilia, ERCP could show a biliary leak in the hepatic bed, the existence of an arterobiliary fistula remained unvisualized by the imaging techniques. Allegedly the incidence of vascular injuries during LC ranges between 0.25 and 0.8% whereas the incidence of biliary injuries ranges between 0.2 and 1%. LC related hemobilia due to pseudoaneurysm accounts for 4.5% of biliary lesions that is around 0.0004% of LC procedures nearly the same as in our experience. TAE of hepatic branches is the first line procedure whereas open or laparoscopic surgery should be advocated only in case of unsuccessful coil embolization. TAE may be followed by rebleeding and requires a second embolization or emergency laparotomy. In the case presented, one single coil embolization of RHA could obtain the definite management of hemorrhage. To date, no definite pathogenetic explanation of hemobilia following LC has been given, but titanium clips are often found in the vicinity of pseudoaneurysms and generally monopolar coagulation is adopted by laparoscopic surgeons, hence mechanical and thermal injuries both to biliary and vascular structures have been considered responsible for this complication. If an inadvertent thermal damage occurs, a char of biliary duct may ensue followed weeks later by its detachment. Bile

![Image](https://via.placeholder.com/150)

Figs 1A and B: (A) The angiogram shows the sac of 2 cm pseudoaneurysm, with no radiologic evidence of arterobiliary fistula. (B) A few days before, the CT scan revealed a vascular lesion of 4 mm, successively enlarging to the size reached at the moment of bleeding (smaller arrow). The pseudoaneurysm was located on a replaced right hepatic artery branching off the superior mesenteric artery (greater arrow)

![Image](https://via.placeholder.com/150)

Fig. 2: After embolization, the arteriogram shows 3-4 mm coils obstructing the replaced right hepatic artery with complete disappearance of pseudoaneurysm

present in 70% and jaundice in 60% of patients. The classic Quincke's triad comprehending melena, pain in the right upper quadrant and jaundice is present in 20 to 40% of patients. In the case hereby described, a nonobstructive jaundice was present even before LC, and therefore this sign could not be used for diagnostic suspicion. In around 60% of cases, a pseudoaneurysm of RHA is found. In some cases branching off the superior mesenteric artery, less
erosion of a vascular char may also play a role in the pathogenesis of bleeding while fistulization into the biliary tree explains hemobilia. Hemobilia may also occur after elective hepatobiliary surgery and emergency, open or converted cholecystectomy during which clips are never or seldom employed; instead, severe local inflammation may entail difficult dissection and thermal damage must be the real causes of inadvertent vascular injuries in such cases.\(^26^-30\) Pseudoaneurysms of hepatic or cystic artery can be even secondary to acute or chronic cholecystitis,\(^31^-33\) and perhaps in some cases this vascular lesion was present even before LC. The size of pseudoaneurysms increases with the time and may reach the noticeable size of 7 cm as observed when cholecystitis is managed nonoperatively for long time,\(^22,31\) or less frequently, when the vascular lesion complicates LC and becomes symptomatic much time later.\(^19\)

In the case presented, the patient had been suffering for months from abdominal pain, the histologic examination showed a thick-walled gallbladder with acute inflammation and chronic cholecystitis but the pseudoaneurysm was tiny (4 mm), hence inadvertent thermal damage must have been the only real cause of vascular complication herein described. The cases reported in the literature often refer to surgical histories of difficult, time consuming LC carrying the risk of inadvertent vascular injuries and pseudoaneurysm thereafter. Suggestions about prevention of such events cannot be found in the specific literature on this complication, but we have enough data to argue that the adoption of bipolar coagulation or better of ultrasonic dissection when dealing with thick-walled gallbladders represents a good piece of advice, especially when dissection digs deep into the liver bed. Under such circumstances, possible thermal damages may be prevented by employing ultrasonic coagulation, since the potential carbonization to surrounding tissues is minimal compared to laser,\(^1\) monopolar and even bipolar coagulation.\(^34^-37\) Hence, the consequences of inadvertent injuries to biliary structures should be minimized by using ultrasonic instrumentation, and hemostasis in the hepatic bed should be achieved by absorbable hemostat products rather than by coagulation. When the cystic artery arises low in Calot's triangle, below the cystic duct, the surgeon can suspect the presence of a replaced or aberrant RHA branching off the superior mesenteric artery, which can be found in 5 to 25% of subjects.

A replaced RHA is an artery supplying the right hepatic lobe whereas an aberrant RHA is an additional branch of RHA.\(^38\) If the suspicion of such anatomical variations is present, the surgeon ought to be particularly cautious with coagulation in that area and ultrasonic dissection should be preferred. Determining if thermal damage is transmitted or not through the clips, is impossible, and it is unlikely either, since dissection in Calot's triangle is commonly carried out before firing clips whereas dissection in the gallbladder bed requires no clip application. Other causes of vascular lesion after LC have been described in a case occurring in a child, the pseudoaneurysm of 8 mm arose in a branch of RHA that had been ligated during LC.\(^17\) Some authors speculate that laparoscopic titanium clips are often found near the pseudoaneurysms and may be partially responsible for arterial or biliary injuries.\(^12,14,15,19,22\)

If surgeons share this opinion, the cystic artery and cystic duct may be interrupted by a clip and ligated by an endoloop then the clips may be gently removed to avoid contacts with surrounding vascular or biliary structures. Obviously, there is no evidence that this strategy is effective in preventing chars of arterial or biliary structures. Adopting these strategies or not is quite up to each surgeon's choice.

**CONCLUSION**

The occurrence of severe hemobilia following LC is a life-threatening vascular complication that can be managed successfully by TAE. Nevertheless, a means to prevent this unpredictable vascular complication has not yet been indicated. Within the purpose of minimizing lateral thermal damage, the adoption of ultrasonic devices during difficult dissections might be proposed to be evaluated in further studies.

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