

Acute Pancreatitis Caused by Hemobilia: An Unusual Complication of Laparoscopic Cholecystectomy

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ABSTRACT

Acute pancreatitis (AP) in the early postlaparoscopic cholecystectomy (LC) period is a rare complication. The cause is often a missed common bile duct stone. Having been reported only once before, we present a second case of AP after LC caused by hemobilia secondary to hepatic artery pseudoaneurysm. The management of this complication is distinctly different from the treatment for AP caused by a stone and must be done on an emergency basis.

INTRODUCTION

With reported rates between 0.05% and 0.34%, acute pancreatitis (AP) in the early postlaparoscopic cholecystectomy (LC) period is a rare complication, often caused by a missed common bile duct (CBD) stone.^{1,2} Hemobilia from a hepatic artery pseudoaneurysm after LC causing AP has been reported only once.³ Hemobilia is bleeding into the biliary tree. It is characterized by the Quinke's triad of abdominal pain, jaundice, and an upper gastrointestinal (UGI) bleed, which can be present in approximately 25% of cases. Hemobilia is an uncommon cause of UGI and can occur following liver biopsy, transjugular intrahepatic portosystemic shunt (TIPS) placement, trauma, hepatocellular carcinoma, cholangiocarcinoma, gallstones, pancreatitis, polyarteritis nodosa, biliary ascariasis, and hepatic artery aneurysm, among others.^{4,5}

CASE REPORT

A 52-year-old woman underwent LC for chronic calculous cholecystitis. The surgery was unremarkable except for some difficulty in the dissection in Calot's triangle due to dense adhesions. There was no significant bleeding or any intraoperative event. She had an uneventful recovery and was discharged on the second postoperative day. Twelve days after the surgery, she was readmitted with complaints of severe epigastric pain and vomiting for the previous 3 days.

On examination, the patient was afebrile, with heart rate 98/min and blood pressure 100/60 mm Hg. There was no jaundice or pallor. She had tenderness and guarding in the epigastric region. Liver dullness was not obliterated, and there were no signs of generalized peritonitis. An abdominal ultrasound showed bulky pancreas, mild peripancreatic free fluid, and normal biliary radicals. Laboratory results showed hemoglobin 13.6 mg/dL, leukocyte count 21,000 cells/mm³, total bilirubin 2.5 mg/dL (direct, 1.5 mg/dL), aspartate aminotransferase 295 IU/L, alanine aminotransferase 471 IU/L, alkaline phosphatase 474 IU/L, serum amylase 1,250 IU/L, and serum lipase 9,826 IU/L. Her coagulation profile was normal. Acute pancreatitis was evident. Magnetic resonance cholangiopancreatography (MRCP) showed features of edematous pancreatitis but no filling defects or dilatation of the CBD (Figure 1).

With a diagnosis of a passed stone that caused AP, the patient was managed conservatively. She responded favorably and was discharged after 7 days. Two days later, she was again readmitted with similar complaints. Her clinical

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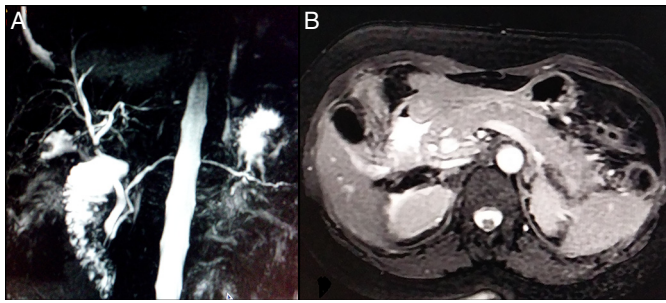


Figure 1. (A) Maximum intensity projection image of abdominal magnetic resonance imaging showing normal biliary tree with mild prominence of the pancreatic duct. (B) Axial section of abdominal magnetic resonance imaging T2 FS images showing a bulky tail of pancreas with mild fat stranding in the tail region.

findings and laboratory parameters were similar to her previous admission. Because she had persistent vomiting, a nasogastric tube was placed. The next morning, she had a small quantity of altered blood in her nasogastric tube. She was hemodynamically stable, and there was no significant drop in her hemoglobin levels.

Bleeding from the second part of the duodenum was evident on UGI endoscopy, and a side-view endoscopy identified a blood clot blocking the ampulla (Figure 2). We did not perform a sphincterotomy to evacuate the clot or a cholangiogram for fear of precipitating a massive bleed. Computed tomography angiography showed a 21-mm hepatic artery pseudoaneurysm in the region of the cystic artery stump (Figure 3). She was immediately taken for a selective celiac artery angiography. Computed tomography angiography findings were confirmed, and 1,000 IU thrombin was injected via a microcatheter into the pseudoaneurysm with complete occlusion of the sac with normal preserved natural artery. In addition, a 1 mm x 30 mm coil was deployed close to the point of origin. Check angiogram showed occluded pseudoaneurysm with well-preserved native vessel (Figure 4).

After the procedure, the patient was comfortable, improved rapidly, and did not require an endoscopic retrograde cholangiopancreatography (ERCP) for evacuation of the clots. She was discharged 4 days after the procedure, and at her 6-month follow-up she remained asymptomatic.

DISCUSSION

Acute pancreatitis after LC is rare and is usually due to missed or small CBD stones that drop from the cystic duct and subsequently block the pancreatic duct.¹ Often, these stones pass spontaneously; if not, ERCP is required to clear the stones from the CBD.

Hepatic artery pseudoaneurysm after LC is rare, but it is a known complication and can present with hemobilia.⁶ Hepatic

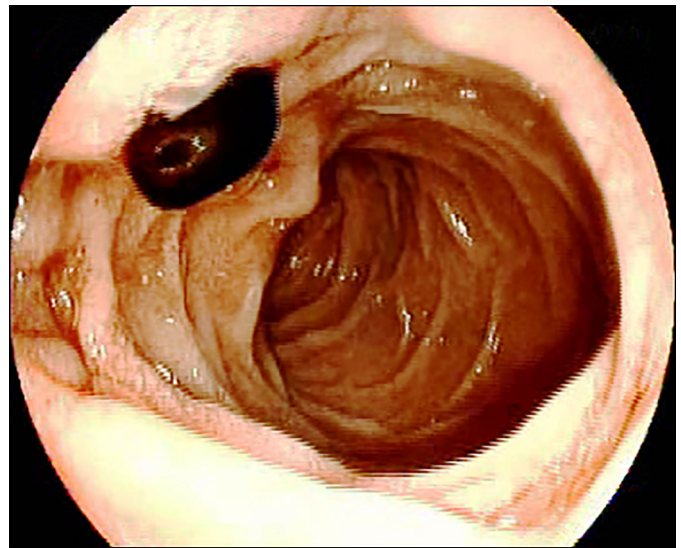


Figure 2. Endoscopic view showing the blood clot at the ampulla.

artery pseudoaneurysm is a delayed manifestation of intraoperative vascular injury. This could be due to mechanical trauma caused by dissection in Calot's triangle, thermal damage due to diathermy, or erosion by metal clips. Occasionally, these can arise secondary to bile leaks. In our case, it is possible that it was caused by mechanical trauma to the right hepatic artery due to the difficult dissection in the Calot's triangle.

The association of hemobilia and AP is rare. It has been reported after liver biopsy, percutaneous transhepatic biliary drainage, and hepatocellular carcinoma.⁷⁻⁹ To date, there is one report of a case of AP caused by hemobilia due to

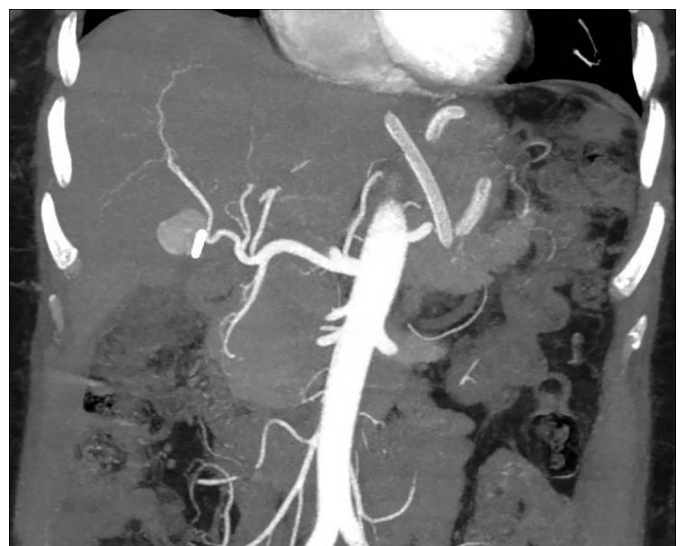


Figure 3. Coronal section of computed tomography angiography showing the pseudoaneurysm from right hepatic artery.

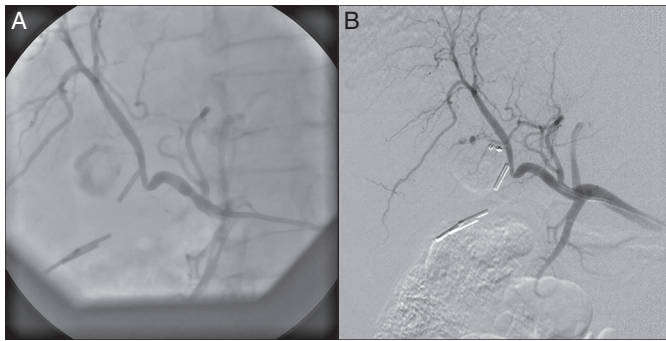


Figure 4. (A) Angiography image demonstrating the pseudoaneurysm. (B) Postembolization/coiling image showing obliterated pseudoaneurysm with preserved flow in the right hepatic artery.

hepatic artery pseudoaneurysm after LC.³ The pathogenesis involves the obstruction of the pancreatic duct by the blood clots and reflux into the pancreatic duct. By the same mechanism, it can lead to jaundice and cholangitis.⁸

The clue to the diagnosis of AP due to hemobilia is a manifestation of UGI bleeding in the background of AP. The rate of bleed in our case was slow, so it was picked up only after insertion of a nasogastric tube during the patient's second readmission. This can be confirmed with a side-view endoscopy, which will show a blood clot or bleeding from ampulla. Another useful investigation is MRCP, which can show biliary dilatation and irregular filling defects.⁹ In our case, the clot may have passed into the duodenum, which is why the MRCP did not show any filling defects. When a hepatic artery pseudoaneurysm is likely to be the cause for hemobilia, a computed tomography angiography will clarify the diagnosis, as was the case in our patient. Because hemobilia was not suspected during the patient's first readmission, we did not administer contrast during the magnetic resonance imaging session, which could have made the diagnosis clear at that time.

A selective transcatheter embolization is both diagnostic and therapeutic, in that the hepatic artery pseudoaneurysm is obliterated using thrombin or other embolic material such as coils, gelfoam (Pfizer, Michigan, USA), or polyvinyl beads.¹⁰ Covered stents are valuable in the management of hepatic artery pseudoaneurysm by excluding the pseudoaneurysm while preserving the flow in hepatic artery.¹¹ We deployed a coil close to the point of origin in addition to thrombin. In the previously reported case of acute pancreatitis after LC, ERCP endoscopic retrograde cholangiopancreatography, and clot retrieval by balloon sweep was performed in addition to embolization.³ In our case, this was not necessary. This can

happen due to spontaneous passage of clots into the duodenum, to dissolution of the clot by the fibrinolytic properties of bile, or a combination of both processes.¹²

DISCLOSURES

Author contributions: All authors collected data. A. Kumar drafted and edited the manuscript, and is the article guarantor. D. Kumar drafted the manuscript. A. Singh critically revised the manuscript. CK Jakhmola edited the manuscript.

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Informed consent was obtained for this case report.

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