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Brief report

BRIEF REPORT

Gallbladder bleeding along with cholecystocolonic fistula: a case report

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Introduction

Gallstone disease is a major public-health problem, with a high prevalence ranging from 10% to 15% [1]. Acute cholecystitis, chronic cholecystitis, choledocholithiasis, and gallstone pancreatitis are all common complications in clinical practice. However, when gallstones migrate to adjacent viscera or vascular structures, the clinical challenge is far less familiar [2].

Gallbladder (GB) bleeding is seldom reported, which mostly manifests as hemobilia. Some cases are caused by cystic artery aneurysm and rupture and others are due to GB rupture [3]. Gallstones can erode through the gallbladder wall or bile duct into the adjacent portion, resulting in a cholecystocolonic fistula (CCF), which is a rare complication of gallstones with cholecystitis [4].

Here, we share a case of gallbladder bleeding along with cholecystocolonic fistula that has not yet been reported. We hope that, through our unusual case, clinicians gain an enhanced understanding of rare complications caused by gallstones.

Case report

A 65-year-old male was admitted, complaining of hematochezia for 5 days, with intermittent abdominal pain and jaundice for 2 years. The patient reported no fever, hematemesis, or bellyache, but a history of gallstones and type II diabetes mellitus. Physical examination revealed yellow sclera, as well as abdominal tenderness over the epigastrium and right hypochondrium. Blood tests revealed severe anemia (hemoglobin 60 g/L), increased white blood cell count (13.18×10^9/L), procalcitonin level (1.64 ng/mL), bilirubin levels (total bilirubin 45.1 μmol/L, direct bilirubin 44.3 μmol/L), transaminase levels (alanine aminotransferase 108 IU/L, aspartate aminotransferase 130 IU/L), alkaline phosphatase levels (682 IU/L), and gamma-glutamyl transferase levels (784 IU/L). Platelet, creatinine, electrolytes, and coagulation index levels were within normal ranges. Furthermore, blood culture of both aerobe and anaerobe showed negative results.

Abdominal computed tomography (CT) revealed gallstones with cholecystitis and a little gas bubble in the gallbladder, while the surrounding tissues, including the gastric wall, transverse colon, and hepatic flexure, were all inflamed (Figure 1). In consideration of cholecystitis, the patient was given antibiotic therapy: sulperazone (150 mg/kg per day) for 7 days. Surgery was not recommended by the surgeons for the first time. As hemobilia was suspected, an esophagogastroduodenoscopy was performed and revealed a Forrest III duodenal bulbar ulcer (1.0 cm in diameter), without duodenal papilla and surrounding abnormalities (Figure 2). Proton-pump inhibitors (esomeprazole, 40 mg intravenous infusion twice a day for 7 days) were given and the bleeding stopped. However, on the 7th day of hospitalization, 200 mL of bright-red blood stool with blood clots suddenly appeared. Therefore, digital subtraction angiography (DSA) was performed immediately and the contrast medium was found to extravasate from the cystic artery into the

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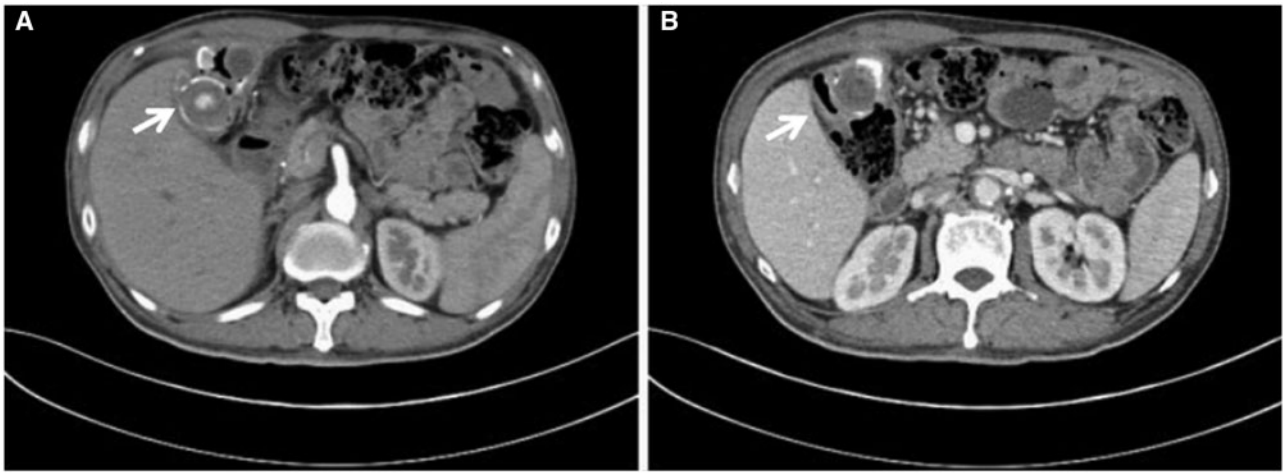

Figure 1. Abdominal computed tomography scan. (A) Gallstones with cholecystitis. (B) A little gas bubble can be seen in the gallbladder, and the surrounding tissues, including the gastric wall, transverse colon, and hepatic flexure, are all inflamed.

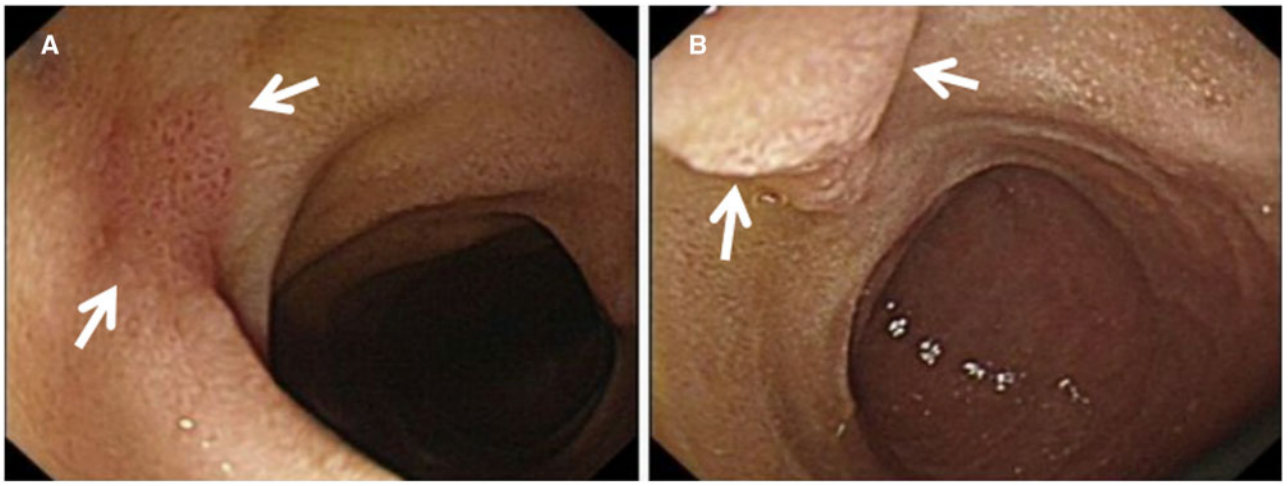

Figure 2. Esophagogastroduodenoscopy. (A) The endoscopy shows a Forrest III duodenal bulbar ulcer, which is 1.0 cm in diameter. (B) No signs of bleeding are presented around the duodenal papilla and surroundings.

gallbladder. Endovascular embolization was successfully performed with a spring coil and the bleeding was immediately stopped (Figure 3). However, 2 days after the DSA, the patient unfortunately presented with fresh hematochezia. A colonoscopy was attempted but failed due to bad bowel preparation.

After we talked with the surgeons again, an exploratory laparotomy was given. During surgery, a fistula between the gallbladder and the transverse colon was detected, as well as massive stones and blood clots. The largest stone was $6 \times 4 \times 3$ cm and crossed the gallbladder and transverse colon. A cholecystectomy with fistula resection and bilioenteric anastomosis were performed. Histopathological examination of the resected gall bladder and partial transverse colon confirmed acute suppurative inflammation; no tumor cells were observed. The colonic glands were arranged regularly and the cell morphology was normal. The structural integrity of the gallbladder wall disappeared, with manifestations of hemorrhages and inflammatory reaction (Figure 4). The final diagnosis was chronic cholecystitis with acute exacerbation complicated by cholecystocolonic fistula and bleeding.

The patient had an excellent post-operative clinical course and was discharged 1 week after the operation. No bleeding was reported after a follow-up over 6 months.

Discussion

In general, GB bleeding manifests as hemobilia with classic presentation as Quincke's triad; it includes gastrointestinal hemorrhage, biliary colic, and jaundice [5]. Typical cases are often characterized by a minor hemorrhage. In these cases, clots are formed, causing biliary obstruction and resulting in pain and jaundice [6].

In our patient, the hemorrhage could not be explained by a peptic ulcer; with his clinical manifestation and history of gallstones, hemobilia was suspected. Our case was confusing as the patient did not present with the standard Quincke's triad, for no

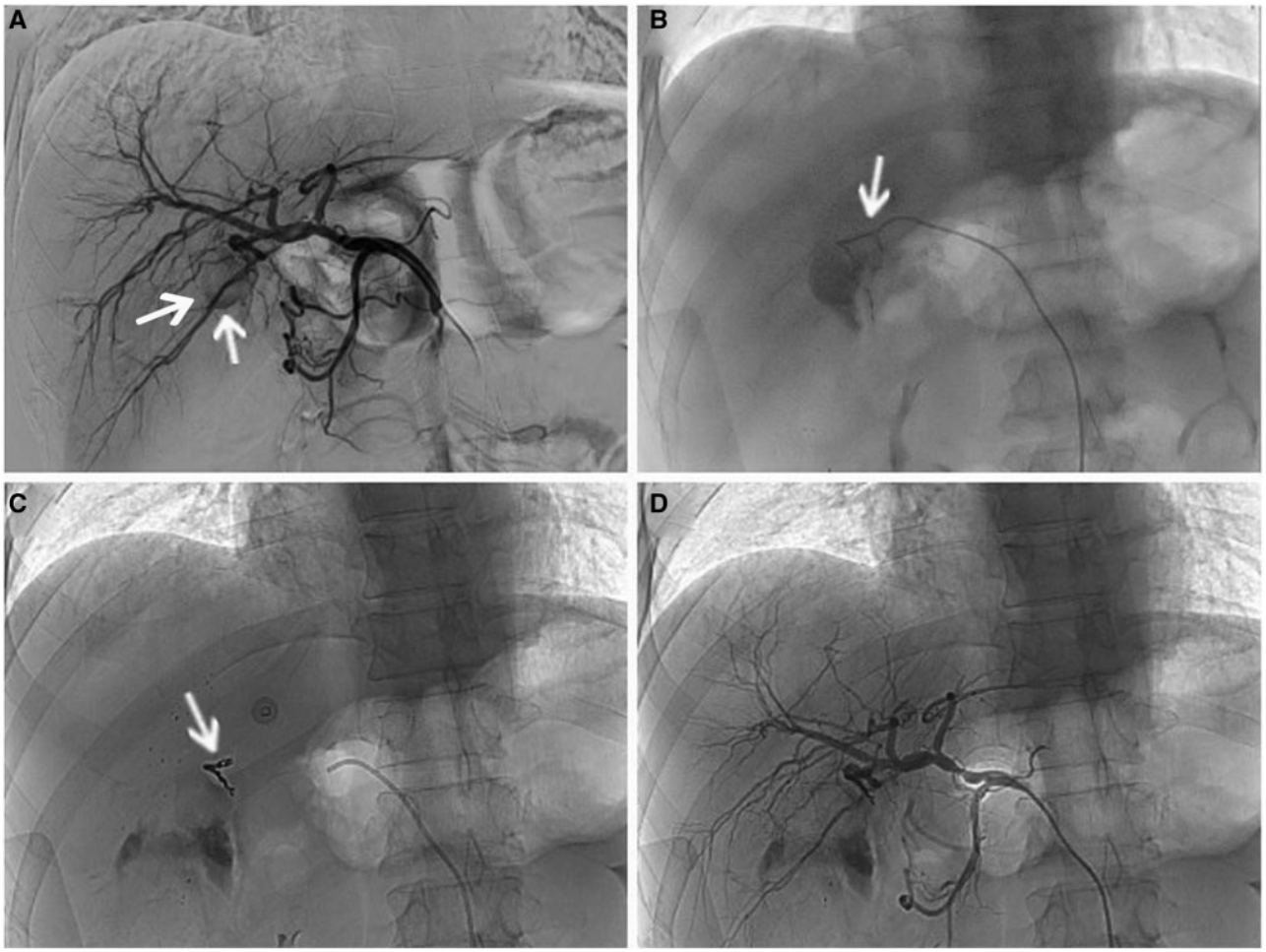

Figure 3. Digital subtraction angiography. (A) Hepatic arteriography shows contrast medium extravasated into the gallbladder. (B) Selective angiogram of the cystic artery reveals areas of bleeding. (C) The cystic artery into the gallbladder is embolized using coils. (D) Post-coil-embolization angiogram shows no contrast-agent extravasation.

obvious biliary colic appeared. Emergent DSA proved that the bleeding came from the gallbladder artery. However, 2 days after the embolization, the patient underwent re-bleeding. Finally, we performed a laparotomy and found a CCF. The existence of GB bleeding explains the cause of hematochezia at first. Furthermore, the existence of a CCF as the major drainage explains the atypical presentation of hemobilia. Finally, blood clots may have blocked the fistula, preventing fresh bleeding into the intestinal tract for a period of time, which explains the delayed re-bleeding after DSA.

What can be learned from our case? First, gallstones with cholecystitis may cause GB bleeding. Second, when the hemobilia is suspected but not presented with typical Quincke's triad and the patient appeared to have continuous gastrointestinal bleeding, the possible existence of a CCF should be considered. Although it is infrequent, gallbladder cancer may co-occur with CCF [7]; we can exclude it by pathologic observation. Finally, surgical treatment consisting of cholecystectomy and resection of the fistula tract should be recommended [8, 9].

Authors' contributions

Q.L. and T.T.L. conceived and designed the report. B.L. collected the data. H.L.Y. reviewed the literature and wrote the manuscript. H.S.M. and J.L.Y. revised and edited the manuscript. All authors read and approved the final manuscript.

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Conflicts of interest

None declared.

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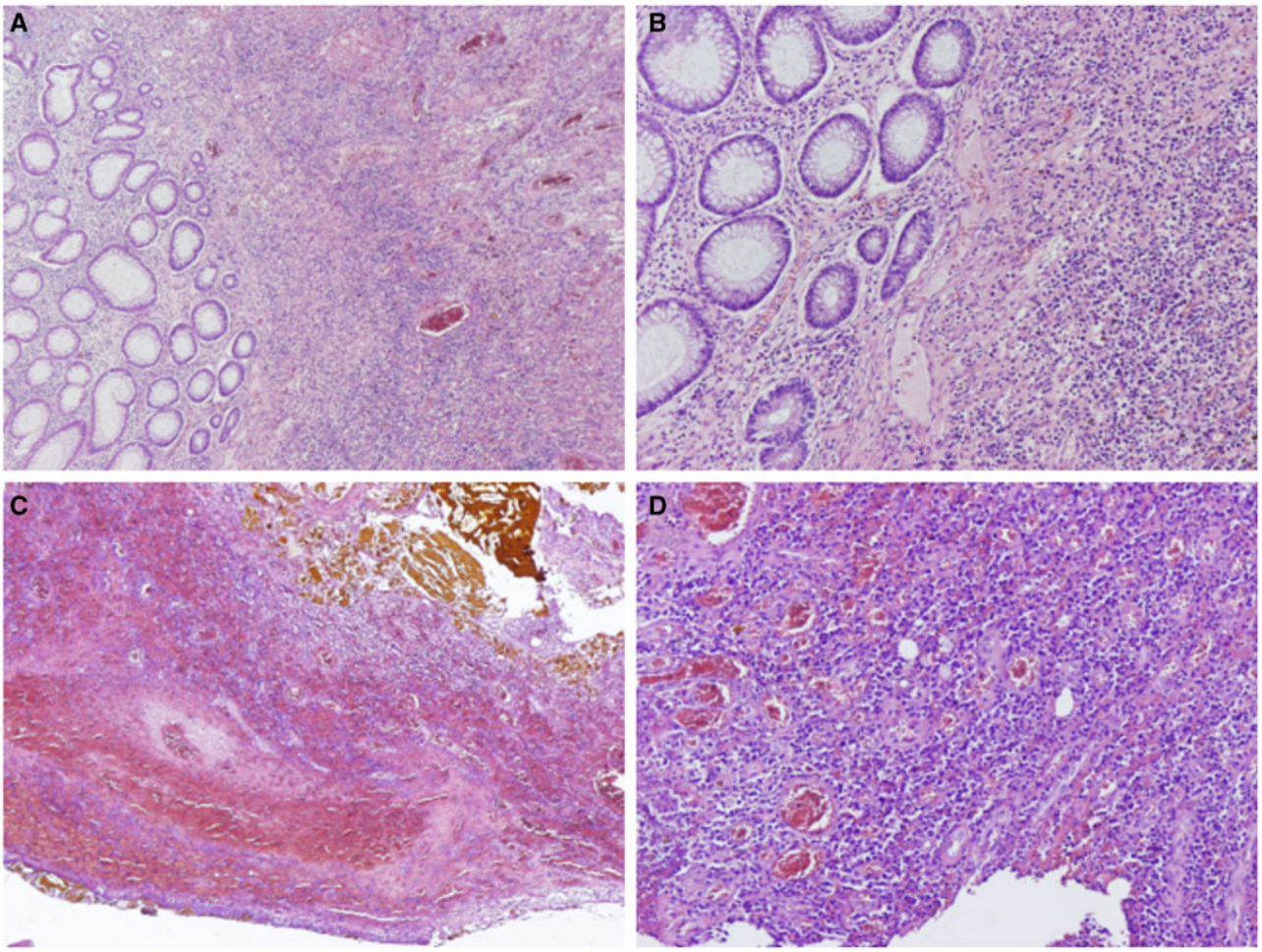

Figure 4. Histopathological pictures. (A) Acute inflammatory cell has infiltrated the mucosa and the submucosa of the resected transverse colon (H&E stain, ×40). (B) The colonic glands are arranged regularly and the cell morphology is normal (H&E stain, ×100). (C) The structural integrity of the gallbladder wall disappears, with manifestations of hemorrhages and inflammatory reaction (H&E stain, ×40). (D) There is an exteive neutrophil infiltration and no tumor cells can be observed (H&E stain, ×100).

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