Case Report

Gallbladder perforation presenting with hemorrhagic shock: Two case reports and an approach to management

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Abstract

Gallbladder perforation is an uncommon surgical condition usually secondary to severe acute cholecystitis. In this report we present a comparison of management for two comorbid patients with gallbladder perforation who presented atypically with hemorrhagic shock, symptomatic large volume hemoperitoneum, and did not have evidence of acute cholecystitis. One patient had a clearly identified gallbladder wall perforation on initial imaging with hemodynamic instability and underwent urgent laparotomy and cholecystectomy. The other patient did not have a clearly identified source for the hemoperitoneum and was later found to have a biliary leak through a HIDA scan. He was a poor surgical candidate due to extensive co-morbidities and was stabilized with resuscitative measures. He was managed conservatively for several days before he clinically deteriorated and passed away. Both patients had different outcomes based on their underlying co-morbidities, presentations and treatment plans.

Keywords: gallbladder perforation, hemorrhage, treatment, surgical versus medical management

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Background

Spontaneous gallbladder perforation is a relatively uncommon complication of acute cholecystitis (1). Management of spontaneous perforation is variable based on patient presentation and characteristics due to a lack of large-scale randomized controlled trials (1). Hemoperitoneum is a rare finding with few reported cases, of which even fewer have been outside the context of acute cholecystitis (2-4). There is a paucity of evidence in current literature regarding identification and management of hemoperitoneum with reference to gallbladder perforation. Operative versus conservative management should be carefully considered on an individual patient basis, taking into consideration patient co-morbidities and state.

Case Report

Patient A is a 70-year-old male who presented with a 12-hour history of worsening abdominal pain, nausea and two syncopal episodes. Past medical history was significant for non-alcoholic fatty liver disease with cirrhosis, coronary artery disease with bypass surgery, hypertension, dyslipidemia, type 2 diabetes mellitus, atrial fibrillation with ablation, erosive gastritis, and intracranial hemorrhage. On initial evaluation, he was afebrile and found to be in hemorrhagic shock with a blood pressure of 72/34 mmHg and pulse of 80 bpm. His abdomen was generally soft with focal tenderness in the right upper quadrant.

Patient B is a 73-year-old male with a 24-hour history of abdominal pain near the site of a parastomal hernia, reduced stoma output and one episode of emesis. The patient had previous episodes of obstruction which presented in similar fashion and had been conservatively managed in the past. This presentation was complicated by findings on cross sectional imaging of hemoperitoneum without a specific source. The patient had severe co-morbidities including insulin-dependent type 2 diabetes mellitus, chronic renal failure with a baseline creatinine of 250 mg/dL, Protein C deficiency with a history of pulmonary embolism and deep vein thrombosis for which he was on chronic Coumadin. His surgical history included a subtotal colectomy for ulcerative colitis with ileostomy and a parastomal hernia. Additionally, he had a substantial cardiac history including a previous episode of ventricular tachycardia and cardiac arrest, previous ST-elevation myocardial infarction (MI), and congestive heart failure with reduced ejection fraction of 30%. The former had been previously managed with an implantable cardioverter-defibrillator, and the MI with percutaneous intervention. On initial presentation, this patient was hemodynamically unstable with a blood pressure of 95/60 and a pulse rate of 62 beats per minute. His abdomen was non-distended with a large midline incisional hernia, which was firm without signs of peritonitis.

Investigations

Initial investigations for Patient A revealed a very low hemoglobin of 40.4 g/L, a low platelet count of 98 x 10^9/L, and elevated lactate of 7.8 mmol/L. A full list of the patient's laboratory findings is summarized in Table 1. Given the hemodynamic instability, a massive transfusion protocol was initiated, and he received 2L of crystalloid solution, 6 U of packed red blood cells, 2 U of platelets and 4 U of fresh frozen plasma. Computed tomography (CT) scan revealed soft tissue hematoma measuring 2 x 6 x 2.2 cm within the gallbladder fossa, perforation of the posterior wall of gallbladder and extravasation of contrast into a large hematoma in the right flank (Figure 1). There were no obvious features of cholecystitis such as wall thickening or hyperemia.

Patient B's initial laboratory findings are summarized in Table 1. The CT scan showed dense free fluid, suggestive of hemoperitoneum in the hepatic, splenic and pelvic regions, without an identified source although a perforated gallbladder was conjectured by the reporting radiologist (Figure 2A and 2B). The gallbladder was filled with stones, dense material, and was completely herniated through the parastomal hernia (Figure 2C). The stomach had surrounding inflammatory changes and fluid along the greater curvature. Loops of small bowel were seen in the hernia without evidence of obstruction. Similarly, there were no obvious features of cholecystitis.

Differential Diagnoses

Patient A's initial imaging was evident for a hemoperitoneum with clear signs of gallbladder perforation. As such, they were immediately taken for surgical management which confirmed the diagnosis.

Patient B's presentation of hemoperitoneum was unusual. There were no overt signs of gallbladder perforation. Additionally, the bleeding initially was suspected to be due to a tear of the liver capsule, or from the short gastric arteries which might be consistent with stranding observed on imaging around the greater curve of the stomach. While being managed medically, he developed elevated transaminases and bilirubin levels and spiked a fever of 38.5 C. This prompted investigation with an abdominal ultrasound to look for evidence of cholecystitis or cholangitis. The ultrasound demonstrated hyperdense material in the gallbladder suspicious for sludge or blood clot, but no evidence of CBD dilation and minimal gallbladder wall thickening. A bile leak was then considered given the location of the fluid on cross sectional imaging, the febrile response, and the biochemical changes. This was confirmed by a hepatobiliary iminodiacetic acid (HIDA) scan showing a collection of radiotracers along the inferior edge of the liver on delayed images in keeping with a gallbladder perforation. Notably, there was filing of the gallbladder, ruling out calculous cholecystitis as an underlying cause of the perforation.

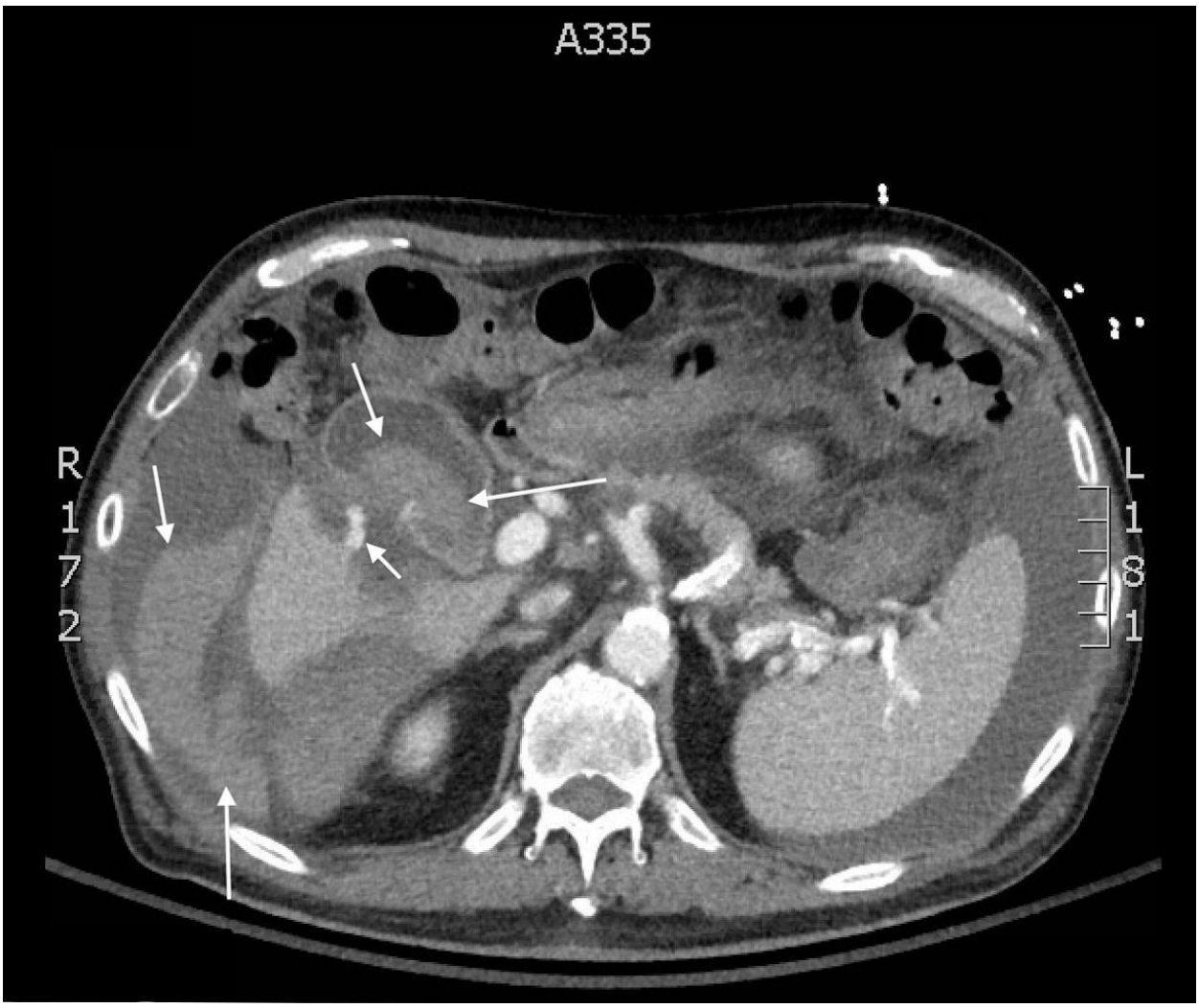

Figure 1. Axial view of contrast-enhanced computerized tomography scan of patient A's abdomen and pelvis soft tissue hematoma within the gallbladder fossa, perforation of the posterior wall of gallbladder, and extravasation of contrast on large hematoma in the right flank.

Treatment

For management of Patient A, a consideration was made for embolization by the interventional radiologist, who could not locate an appropriate target on the initial scans. Given refractory hemodynamic instability, the patient was brought to the operating room urgently for exploratory laparotomy. Upon entering the peritoneum, a large volume of blood was evacuated from the right subphrenic and the subhepatic space. The bleeding was controlled with surgical packs to give time for resuscitation before exposing the gallbladder which demonstrated obvious rupture at the fundus with active bleeding directly from the gallbladder wall. This was controlled with cautery and Surgicel and cholecystectomy was subsequently performed. No additional injuries, bile leak, or anatomical variants were identified.

For Patient B, based on initial presentation and imaging, hemoperitoneum was thought to be secondary to a small tear in the liver capsule or the short gastric vessels, as a gallbladder perforation was felt to be unlikely to cause such a large volume of hemorrhage. He was considered a poor surgical candidate given his significant co-morbidities. He responded well to initial resuscitation and was admitted to a step-down unit for conservative management with empiric broad-spectrum antibiotics and transfusion as necessary.

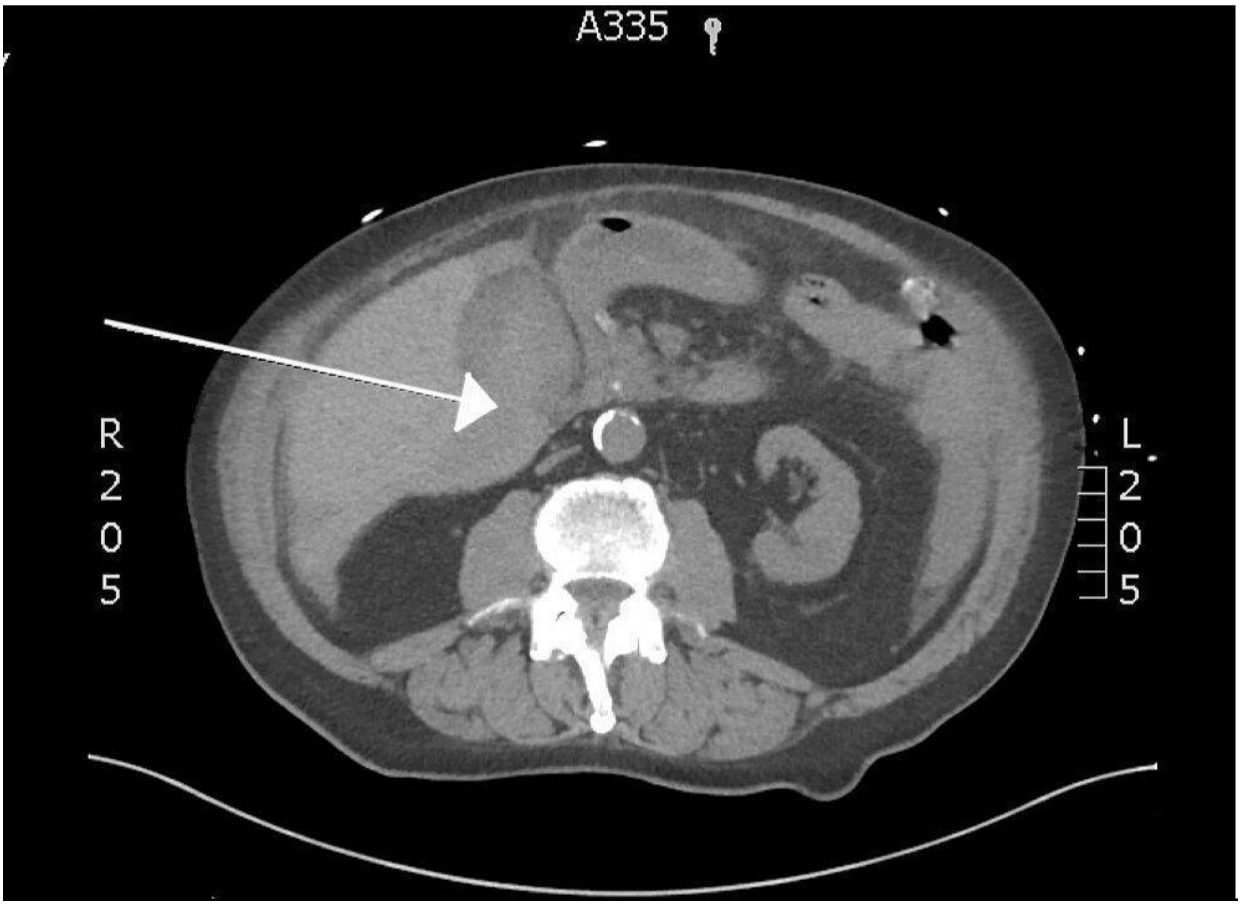

Figure 2A. Axial view of computerized tomography scan of patient B's abdomen and pelvis showing ?hemoperitoneum or biliary leak in gallbladder fossa

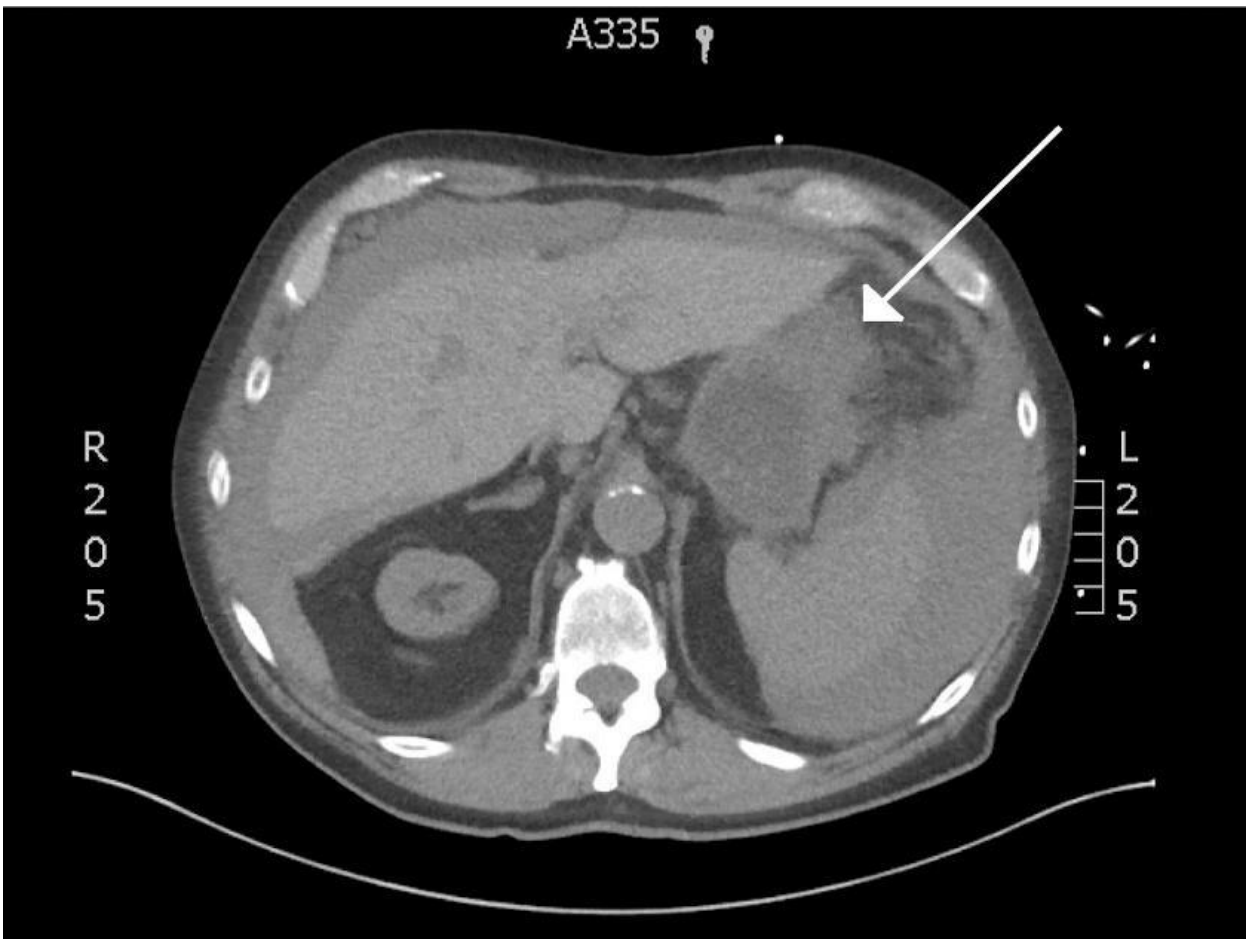

Figure 2B. Axial view of computerized tomography scan of patient B's abdomen and pelvis showing ?hemoperitoneum

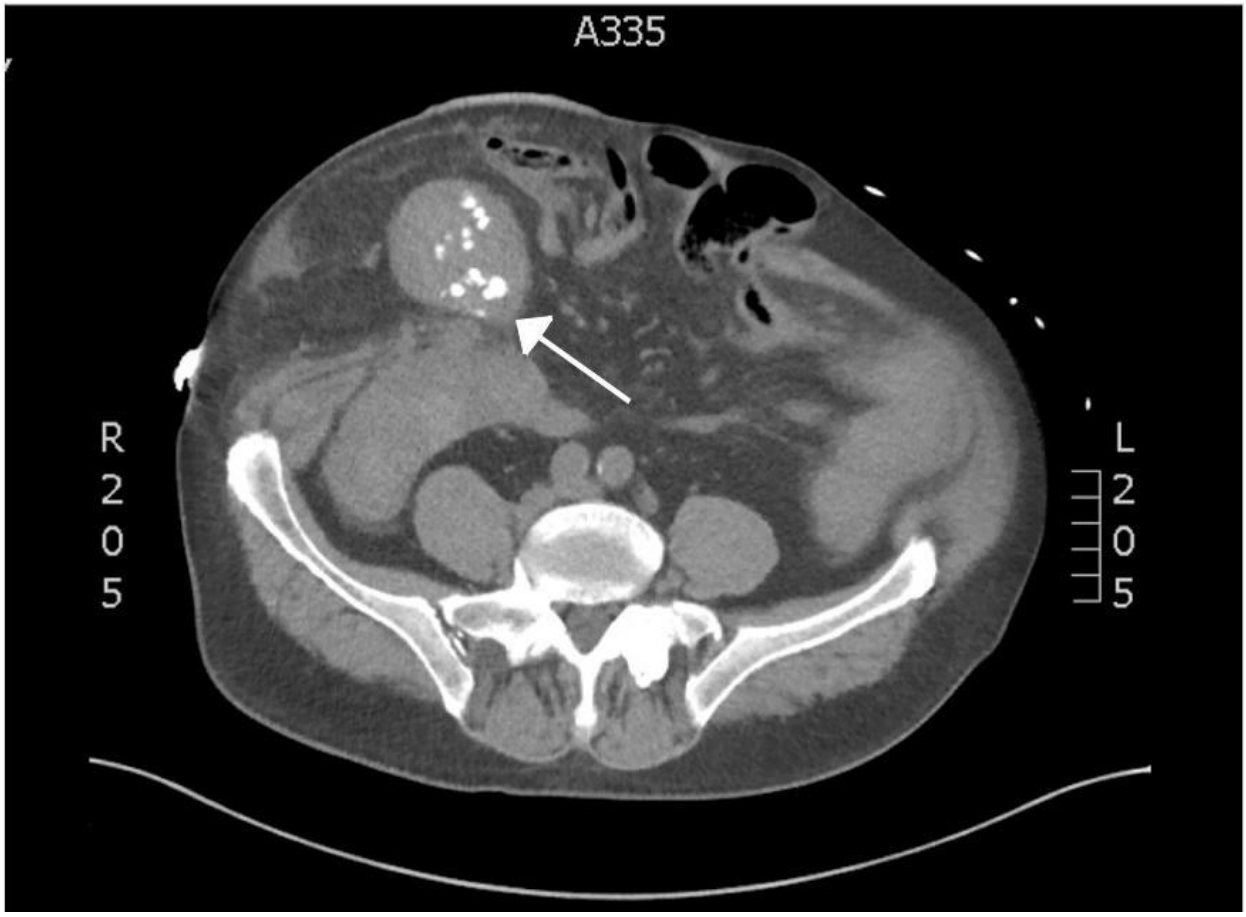

Figure 2C. Axial view of computerized tomography scan of patient B's abdomen and pelvis showing herniation of the gallbladder through the stoma defect

Table 1. Laboratory Values

Patient A

Characteristics	Value	Normal Value
Complete blood cell count		
White blood cell count, × 10^9/L	11.3	4.0-11.0
Hemoglobin, g/L	44	135-175
Platelet count, × 10^9/L	98	150-400
Coagulation Profile		
INR	2.0	0.8-1.2
Fibrinogen, g/L	1.00	1.7-4.0
Biochemical Examination		
Total bilirubin, umol/L	35.9	3-22
Direct bilirubin, umol/L	9	0-5
Alanine aminotransferase, U/L	18	5-68
Alkaline phosphatase, U/L	97	50-136
Creatinine, umol/L	84	58-110
Lactate, mmol/L	7.8	0.4-2.1

Table 1 (continued)

Patient B

Characteristics	Value	Normal Value
Complete blood cell count		
White blood cell count, × 10^9/L	12.2	4.0-11.0
Hemoglobin, g/L	120	135-175
Platelet count, × 10^9/L	218	150-400
Coagulation Profile		
INR	3.3	0.8-1.2
Fibrinogen, g/L	4.80	1.7-4.0
Biochemical Examination		
Total bilirubin, umol/L	49.4	3-22
Direct bilirubin, umol/L	27	0-5
Alanine aminotransferase, U/L	188	5-68
Alkaline phosphatase, U/L	294	50-136
Creatinine, umol/L	278	58-110
Lactate, mmol/L	6.6	0.4-2.1

Outcome and Follow-up

Patient A tolerated the procedure well. He was initially admitted to the intensive care unit where he remained hemodynamically stable and was quickly transferred to a surgical ward for post-operative management. The pathology revealed no signs of calculi, malignancy, or acute inflammation and was reported as chronic cholecystitis. After a ten-day course at hospital without further complications, the patient was safely discharged home.

Patient B, whose work up was previously described, was transitioned to antimicrobial therapy of Ceftriaxone and Metronidazole once a gallbladder perforation was more confidently diagnosed. There was no evidence of ongoing bleeding, obviating the need for embolization for control of hemorrhage. Consideration was given to endoscopic retrograde

cholangiopancreatography (ERCP) or cholecystectomy to control an ongoing bile leak but given his comorbidities it was decided to continue with conservative measures. In the subsequent days, the patient clinically declined, with worsening kidney function and development of cardio-renal syndrome with fluid overload. The patient was not responsive to hemodialysis and subsequently passed away.

Discussion

Hemoperitoneum and hemorrhagic shock from gallbladder rupture is an extremely rare presentation. Few case reports in the past have discussed this phenomenon, with the majority describing an element of hepatic injury from gallbladder rupture, leading to hemoperitoneum (2). Additionally, the cause for gallbladder rupture varies including, malignancy (2), acute cholecystitis (3) and blunt trauma (4). Patients in all these cases typically presented with right upper quadrant/abdominal pain and nausea/vomiting. In contrast, the presentation of both patients was atypical in this report with neither having a known history of cholecystitis, malignancy, or trauma. Patient A presented with syncopal episodes in addition to abdominal pain and nausea. The CT imaging was pivotal in recognizing gallbladder rupture as the potential source of hemoperitoneum. The pathology report for patient A revealed chronic cholecystitis, without any identified source. Regardless, we hypothesize chronic inflammation led to ulceration of gallbladder wall and subsequent hemorrhage in patient A.

Patient B's presentation initially mimicked his previous presentations of bowel obstruction and a lack of a clear source of hemoperitoneum delayed recognition of gallbladder rupture as the underlying diagnosis. It is more difficult to decipher the cause for rupture in this patient. Although the CT scan did not show any signs of bowel obstruction, there was complete herniation of the gallbladder through the parastomal hernia. A previous case report showed that mobile structures like bowel more commonly get incarcerated and perforate through parastomal hernia, but non-naturally mobile structures like the stomach can also herniate and perforate (5). The first ever case of gallbladder perforation through a parastomal hernia was reported in 2021 (6). The patient presented with acute abdominal pain and no output from ostomy, like patient B in this case, however they did not have hemodynamic instability or evidence of hemoperitoneum on the CT scan. In the case of patient B, in retrospect, the hemoperitoneum may have included some component of bile, however there were no features such as peritonitis or hyperbilirubinemia at initial presentation to suggest this.

Treatment for spontaneous perforation

Management of gallbladder perforation has traditionally been emergency cholecystectomy or drainage from gallbladder fossa using image-guided percutaneous techniques. The method of management is dependent on multiple factors, including patient co-morbidities, type of perforation and patient preference. Most commonly, patients with Type I perforations (acute, free perforation), who are good surgical candidates and amenable to the procedure, undergo

surgery (7). At times, percutaneous drainage can be used in patients who are not fit or refuse surgery (7).

In this report, the prompt identification of gallbladder rupture through CT imaging in patient A, in combination with him being a reasonable surgical candidate, led to him receiving emergent surgical management and fared well. By contrast, the delay in identification of gallbladder perforation as a potential source of hemoperitoneum and shock in patient B, in combination with patient's poor surgical candidacy was fatal.

Conclusion

Angioembolisation and percutaneous drainage with or without ERCP are potential treatment options, which may be offered to patients with bleeding or bile leak, who are poor surgical candidates. Future studies can focus on evaluating large databases on patient outcomes to provide further information to establish guidelines for management of similar patients.

This case report was conducted after obtaining informed consent from patient A and from patient B's substitute decision maker.

Learning Points/Take Home Messages

- Spontaneous gallbladder rupture can rarely present atypically with hemodynamic instability and hemoperitoneum secondary to hemorrhage.
- Management in such patients should start with prompt resuscitation as necessary, considering gallbladder perforation as a potential cause for the presentation
- Management of gallbladder perforation with hemorrhagic shock is emergency cholecystectomy. Angioembolization followed by percutaneous drain with or without ERCP stenting depending on the presence of cholecystitis can be considered in non-operative candidates to control ongoing bleeding and bile leakage.

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