Isolated Gallbladder Perforation Following Blunt Abdominal Trauma: a case report

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ABSTRACT

Although infrequent, gallbladder injury occurs in approximately 2% of blunt abdominal trauma cases, which are most commonly caused by motor vehicle accidents. Furthermore, isolated gallbladder injury, including gallbladder perforation, is even more rare.

A 39 year-old male patient fell down accidentally from a height of 3 m and was sustained injuries, causing right upper quadrant pain and laceration of the left facial region.

Computed tomography (CT) of the facial bones showed fractures of the nasal bone and left maxilla. Additionally, CT of the abdomen revealed a 4-cm, round, mildly hyperdense gallbladder lesion. Intraperitoneal fluid was also observed. No further imaging evidence suggestive of additional internal organ injuries was found. Ultrasonography (US)-guided paracentesis was performed and 50 c.c. of dark green fluid was aspirated. The fluid was confirmed to be bile ascites. US revealed heterogeneously hypoechoic content inside the gallbladder. Therefore, the patient was diagnosed with gallbladder hematoma and perforation. Emergent laparotomy uncovered a 2-cm perforation in the anterior wall of the gallbladder and 1500 c.c. of turbid ascites was removed. Cholecystectomy was performed and no additional intra-abdominal injuries were noted. This case demonstrates that accurate preoperative diagnosis with CT and US, and, if necessary, paracentesis can prompt emergent laparotomy to reduce patient morbidity and mortality.

CASE REPORT

A 39 year-old male patient sustained injuries after an accidental fall from a height of 3 m. Physical examination revealed laceration of the left facial region and pain in the right upper quadrant. CT of the facial bones performed at another hospital showed fractures of the nasal bone and left maxilla. The patient was then transferred to our emergency department. Blood analysis results were as follows: white blood cell count (WBC), 14380/µL; glutamate pyruvate transaminase (GPT), 102 U/L; total bilirubin, 3.4 mg/dL; direct bilirubin, 1.94 mg/dL; and C-reactive protein, 7.98 mg/dL. Multidetector row CT (MDCT, Brilliance CT 64-channel, Philips Medical Systems, Haifa, Israel) of
the abdomen with contrast-enhanced arterial and delayed venous phases revealed a 4-cm, round lesion in the minimally wall-thickened gallbladder (Fig. 1). The lesion was hyperdense, with an attenuation value of 70 H.U. on precontrast-enhanced CT. Intraperitoneal fluid with an attenuation value of approximately 5 H.U. was also seen in the abdominal and pelvic cavities. No increased contrast accumulation was identified inside the gallbladder on delayed venous phase. Furthermore, no additional imaging evidence of injuries to other internal organs was found.

Therefore, intraluminal hematoma of the gallbladder was suspected based on CT findings. The interval between the traumatic event and the performance of abdominal CT was more than 12 hours.

Due to the patient’s persistent right upper quadrant pain, paracentesis was performed in the right lower anterior abdomen under US guidance 2 hours after CT. A total of 50 mL of dark green fluid was aspirated and confirmed to be bile ascites with a total bilirubin level of 40 mg/dL and direct bilirubin level of 26 mg/dL. US of the gallbladder

**Figure 1.** Multidetector row computed tomography with precontrast a, post-contrast-enhanced arterial b, delayed venous c, phases, and reformatted coronal images d, show mildly hyperdense content inside the gallbladder with no increased contrast accumulation on the delayed venous phase (c). Diffuse and minimal wall thickening of the gallbladder is noted, with a 4-mm wall thickness (b-d). Hypodense ascites is seen around the liver, and in the pelvic cavity (a-d).
showed heterogeneously hypoechoic content inside the
gallbladder as compared to the diffusely hyperechoic fatty
liver parenchyma (Fig. 2). Therefore, gallbladder hematoma
and perforation were diagnosed based on imaging findings.
Emergent laparotomy uncovered a 2-cm perforation in
the anterior wall of the gallbladder (Fig. 3) and 1500 c.c.

**Figure 2.** Ultrasonography at the right intercostal a. and right subcostal approaches b. reveals heterogeneously hypoechoic
content inside the gallbladder relative to the hyperechoic fatty liver. The content is more hyperechoic than the renal cortex
(figure not shown).

**Figure 3.** The resected gallbladder specimen demonstrates a blood clot a. overlying the perforation, which can be inserted
by a straight Kelly forceps b.
Isolated gallbladder perforation

of turbid ascites was removed. No other intra-abdominal injuries were found. Cholecystectomy was performed. Follow-up blood analysis 4 days later revealed a return of the elevated blood findings to normal levels (WBC, 5510/µL; GPT, 33 U/L; total bilirubin, 1.1 mg/dL; direct bilirubin, 0.56 mg/dL), with the exception of a consistently high level of C-reactive protein (7.18 mg/dL). Postoperative fever subsided on the day 5 after initiation of treatment with antibiotics, and the patient was discharged uneventfully on postoperative day 10.

DISCUSSION

Although gallbladder injury is uncommon, it has been reported in approximately 2% of cases of blunt abdominal trauma most commonly caused by motor vehicle accidents or by direct blows and falls, as in our case [1-5]. Injury to the liver is most commonly associated with gallbladder injury, whereas spleen, stomach, bowel loop, pancreas, and kidney injuries are less common [1]. Isolated gallbladder injury, including gallbladder perforation is even rarer. The low incidence of gallbladder injury is likely attributable to its protection by the surrounding liver and rib cage [5].

The clinical signs and symptoms that arise from isolated traumatic gallbladder injuries may be vague and insidious, and range from acute abdomen to slowly progressive abdominal complaints. As clinical signs and symptoms may take up to 6 weeks to become apparent, diagnosis may be delayed and result in significant increase in morbidity and mortality associated with traumatic gallbladder injuries [1-2, 6-8]. The delayed presentation of a gallbladder perforation may be attributable to leakage of sterile bile into the abdomen, which leads to bile peritonitis that can be accompanied by tenderness, or with few to no symptoms. Subsequently, the omentum may wrap around the gallbladder to prevent further bile leakage and peritoneal irritation, and lead to a dull pain and clinically palpable mass in the right upper abdomen [7-8].

Injury to the gallbladder is usually caused by a direct blow in blunt abdominal trauma, or by a shearing force, which may lead to an avulsion injury [4]. The spectrum of gallbladder injuries includes contusions, lacerations (also referred to perforations), and avulsions [1-2, 5, 8-9]. Gallbladder contusion, or intramural hematoma, is most commonly encountered during laparotomy.

Gallbladder perforations, the most often reported gallbladder injuries, account for 3.2% of all injuries to the gallbladder [2]. Most perforations occur at the fundus and neck of the gallbladder, which are the weakest points in response to direct blows. [5, 8]. The factors that predispose to traumatic gallbladder perforation are distention of the gallbladder, a thin-walled gallbladder, and alcohol ingestion [5]. Alcohol not only relaxes the abdominal wall muscles, but also increases the tone of the sphincter of Oddi, resulting in gallbladder distention. Conversely, the thickened gallbladder wall in a chronically diseased gallbladder protects it from injury [8]. Gallbladder thickening may also occur secondary to acute or chronic cholecystitis, extracholecystic inflammatory processes (e.g. acute hepatitis, peritonitis, acute pancreatitis, and acute pyelonephritis), systemic causes (e.g. liver cirrhosis and congestive heart failure), or gallbladder tumors, including benignity (e.g. adenomyomatosis and polyps) and malignancy (either primary or secondary malignant tumors) [10]. Non-traumatic causes of gallbladder perforation may be due to acute cholecystitis, infectious processes, or malignancy [5].

Gallbladder avulsion is the most severe form of injury, and may be partial or complete [2, 4, 8]. Partial avulsion, in which the gallbladder is partially torn from the liver bed, may compromise venous or lymphatic drainage and lead to gallbladder edema, gangrene, and subsequent rupture [8]. Complete avulsion results in complete separation of the gallbladder from the liver and its attachments (i.e. traumatic cholecystectomy) [2, 4].

Several methods can be used for the diagnosis of gallbladder perforation. Diagnostic peritoneal lavage is useful only if the fluid is bile (as in our case), but may not be helpful due to the high rate of false negative results, especially in cases where the bile leak and hematoma from the perforated gallbladder are within the extraperitoneal gallbladder fossa [2-4, 6].

US can be effective in diagnosing gallbladder perforations. Heterogeneously hyperechoic blood inside the gallbladder, pericholecystic fluid accumulation, hypoechoic and edematous gallbladder wall thickening, and disruption of the gallbladder wall with focal loss of reflectivity are expected US findings [4-5]. In our case, the patient had a fatty liver, causing the liver parenchyma to be diffusely hyperechoic on US. Therefore, the hematoma inside the gallbladder was hypoechoic relative to the hyperechoic liver parenchyma, but was hyperechoic as compared to the renal cortex (figure not shown).

CT has been shown to be the most effective diagnostic tool for detecting gallbladder injuries [2, 4]. Blood within the gallbladder is hyperdense on CT. Other CT findings associated with gallbladder injuries include pericholecystic fluid, thickened and indistinct gallbladder walls, a mass effect on the adjacent organs (liver, duodenum, and right kidney) due to gallbladder distention, and active arterial extravasation into the gallbladder lumen. The latter may be confirmed by increased amounts of hyperdense fluid within the gallbladder on delayed contrast-enhanced CT images, which may show an increased amount of hyperdense gallbladder fluid as the hemorrhage progresses in the injured gallbladder [2]. However, this is not always present, as in our case. Cholelithiasis, hyperechogenic sludge, milk-of-calcium, and vicarious contrast excretion of intravenous contrast medium may mimic the hyperdense blood in the gallbladder lumen.
In equivocal cases with a high level of clinical suspicion of gallbladder injury, hepatobiliary scintigraphy, especially in delayed imaging, can detect the extravasation of radioisotopes due to bile leakage from the biliary tract [4, 6]. Magnetic resonance imaging can be used for detection of subtle areas of mural discontinuity in the gallbladder wall due to superior soft tissue contrast resolution [11]. However, it is not widely used due to high costs and long examination times.

In conclusion, isolated gallbladder perforation is rare after blunt abdominal trauma. The clinical signs are often non-specific and may result in delayed diagnosis. Correct diagnosis with US, CT, and, if necessary, paracentesis, can prompt emergent laparotomy and reduce patient morbidity and mortality.

REFERENCES