**Isolated gallbladder rupture following blunt abdominal trauma**

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**Abstract**

The gallbladder is a relatively well-protected organ; consequently its rupture following blunt abdominal injury is rare and usually associated with other visceral injuries. Isolated gallbladder rupture is extremely rare. We report a healthy Nigerian adult male who sustained isolated gallbladder rupture following blunt abdominal injury from riding a motor cycle (Okada). A high index of suspicion with positive bile aspirate might lead to early diagnosis. Open cholecystectomy is a safe option of treatment in a resource poor centre especially in delayed presentation and has a good outcome.

**Key words:** Blunt abdominal trauma, gallbladder rupture, isolated

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**Introduction**

The gallbladder is relatively a well-protected organ being partially embedded in the liver substance, cushioned by the surrounding omentum and intestine and shielded by the rib cage.[1,2] Consequently gallbladder rupture following blunt abdominal trauma is rare and is usually associated with other visceral injuries.[1-6] Isolated gallbladder rupture due to blunt abdominal trauma is even rarer.[1-3,5] Most literature on the subjects are case reports because of the rarity of the disease and contrary to earlier reports, in which falls, kicks or blows were the most common factors causing blunt gallbladder injuries. Road traffic accidents (RTA) that are on the increase are now the primary causes of such injuries.[2] We report a case of isolated gallbladder rupture due to blunt abdominal trauma from a motor cycle accident; which to our knowledge is the first of such report.

**Case Report**

G M M, a 22-year-old male commercial motorcyclist, was referred from a General Hospital to University of Maiduguri Teaching Hospital on 30th December 2011 with a 2-week history of progressive abdominal distension a week after a motorcycle accident.

He was carrying a passenger on his motorbike 3 weeks earlier and collided with another motorcycle. He had lost consciousness transiently on falling to the ground, but no bleeding from any orifice or convulsion. He complained of abdominal pains and abrasions on his left hypochondrial region for which he was treated and discharged the same day. He had no fever, jaundice, vomiting or change in bowel habit and his last meal was over 12 hours.

One week after the trauma he developed a progressive painless abdominal distension. He presented again to the General Hospital where an unspecified amount of darkish yellow fluid was aspirated from his abdomen and was referred to us 3 weeks after the trauma. On examination he was not febrile with a temperature of 36.9°C, not pale and not jaundiced. His pulse rate was 78/min, blood pressure 130/70 mmHg, and his respiratory rate was 22. The chest was clinically clear. The abdomen was grossly distended with dull percussion note and a positive fluid thrill. Abdominal
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Ultrasound scan reported an intraabdominal fluid collection with internal echoes and strands within it. However, the liver echo texture was preserved but the gallbladder was not visualized. The spleen appeared normal with no evidence of rupture. Abdominal paracentesis yielded bile stain peritoneal aspirate. His PCV was 37%; urea, electrolyte and creatinine were within normal limits.

Exploratory laparotomy revealed bilious peritoneal fluid amounting to about 5 l in both supra and infra colic compartments forming 3 pseudo cysts; the larger one in the infra-colic compartment and two smaller ones in the supra colic compartment, one extending into the Morrison's pouch and communicating with a perforation about 3 × 2 cm at the fundus of the gallbladder Figure 1. He had cholecystectomy and peritoneal lavage after the wall of the cyst was gently teased out Figure 2. He made an uneventful recovery and was discharged from the hospital 10 days after the surgery. Histopathology report confirmed the perforation and pseudo cyst.

Discussion

Injury to the gallbladder (GB) following blunt abdominal trauma is rare and accounts for about 2% of all other intraabdominal injuries. Isolated damage to the GB is even rarer as it is protected under the rib cage and the liver in which it is embedded. GB rupture occurs predominantly in males as was the case in our patients; this is in keeping with their propensity for involvement in violent accidents. Children especially are involved because of their increased vulnerability to direct abdominal trauma and also poor anterior abdominal wall muscular developments.

The thin-walled GB is more prone to rupture from blunt abdominal trauma than a diseased GB that is thickened and fibrotic from chronic inflammation. For GB rupture to occur following blunt abdominal trauma, GB distension is almost always present. This was the case in this patient whose last meal was over 12 h before the trauma. Alcohol enhances gastrin and secretin secretions which in turn stimulate bile flow and high serum alcohol elevates the common bile duct pressure by increasing the sphincter of Oddi’s tone. This combined effects causes GB distension which makes it more susceptible to injury, especially since alcohol also relaxes the abdominal wall musculature. The majority of GB injuries occur following motor vehicular incidents from road traffic accidents followed by significant falls and direct kicks or blows from sporting activities and fights. The underlying mechanism is the shearing acceleration – deceleration force with impacts against the steering wheel or seat belts or the direct blows. The exact mechanism of injury in our patient remains unclear; he was riding a motor cycle that collided with another, direct blow with parts of the motor cycle to the abdomen may be the cause as there were only bruises in the left hypochondrium that led to the earlier suspicion of splenic injury.

Several types of GB injuries may result from blunt abdominal trauma. Blunt gallbladder injuries are classified as contusion, avulsion, laceration and traumatic cholecystitis. Laceration of the gallbladder, also termed “traumatic rupture” or “perforation,” is the most common reported type of GB injury secondary to blunt trauma the type our patient had. Avulsion injury of the GB is the second most common reported injury, and has three subtypes: Partial avulsion, in which the gallbladder is partially torn from the liver bed: Complete avulsion, in which the gallbladder is completely torn from the liver bed but with intact cystic duct and artery; and total avulsion also termed “traumatic cholecystectomy,” in which the gallbladder lies free in the abdomen, torn from all attachments. Traumatic cholecystitis is caused by cystic duct obstruction by blood clots from liver or gallbladder injury. Delayed perforation following blunt abdominal trauma can also occur, following a hematoma of the GB wall developing into an area of necrosis, or blood clot occluding the cystic duct and precipitating infection, gangrene and late perforation. Nontraumatic perforation of the gall bladder

Figure 1: Laparotomy showing the liver and perforated gallbladder

Figure 2: Gallbladder with 3 × 2 cm perforation
as may occur in acute cholecystitis is much commoner than traumatic perforation. The two cases of perforated gallbladder as a complication of acute cholecystitis secondary to typhoid fever and cholelithiasis has been reported. Isolated GB rupture due to blunt injuries to the abdomen, though very rare, can occur and may constitute a diagnostic challenge especially with delayed presentation. Persistent bile leak causes sequestration of fluid resulting in intraperitoneal bilious fluid collection. The clinical presentation is variable and nonspecific, resulting in delay in diagnosis as was the case in our patient. Damage to a noninfected gallbladder can cause leakage of sterile bile into the abdomen. This in itself does not present acutely and such injuries can take up to 6 weeks to become apparent. Unless bacterial peritonitis supervenes, generalized biliary peritonitis may be innocuous. In fact, it is not unusual to have the patient discharge from the hospital, only to return days or weeks later with a few or numerous signs and symptoms of peritonitis, including weight loss, nausea, vomiting, abdominal distension, jaundice, ascites, low-grade fever, and abdominal pain. This is exemplified by our patient that was managed conservatively for suspected splenic injury and was discharge only to return with progressive abdominal distension. Diagnosis of GB rupture secondary to blunt abdominal injury is mostly incidental at laparotomy for evaluation and treatment of other visceral injuries. Delay in making diagnosis may be due to no, or vague symptoms or subtle signs coupled with low index of suspicion. Thus, delay in making diagnosis is a rule rather than the exception. Bile stained aspirate with ultrasound report of normal spleen and liver suggest possible injury to the biliary tract in our patient though delayed splenic injury was initially entertained clinically. Diagnostic procedures are not always helpful in rupture of the gallbladder because a bile stained aspirate may be from trauma to the biliary tract, GB, the liver or upper GI tract and thus not specific. A high incidence of false negative results has been reported especially if the bile leak is entirely intraperitoneal. A contrast study depends on the ability of the gallbladder to concentrate media which may be distributed in an injured GB. Ultrasonography and CT scans are found valuable when bile leak becomes encapsulated forming a mass, while Tc99m-HIDA cholecystoscintigraphy has a more accurate means of detecting bile leak. Magnetic resonance imaging and endoscopic retrograde cholangiopancreatography are other possible diagnostic modalities in blunt abdominal trauma.

Various treatment options have previously been suggested for the management of a gallbladder injuries, including: Expectant observation, drainage, cholecystorrhapsy or cholecystostomy, but cholecystectomy is now the treatment of choice. Laparoscopic cholecystectomy is advocated to be safe and effective procedure in the diagnosis and treatment of traumatic gallbladder injuries. In our case, exploratory laparotomy and cholecystectomy were done due to the delay in presentation and uncertainty of the diagnosis. High morbidity and mortality in patients with gallbladder injuries is usually due to associated intraabdominal injuries. However, no deaths have ever been reported in patients with isolated gallbladder rupture treated surgically. Our patient did well postoperatively and was seen 2 weeks after discharge at SOPD and was lost to follow-up subsequently.

We conclude that isolated GB rupture due to blunt abdominal trauma is extremely rare and that resulting from a motor cycle rider has not been reported previously. High index of suspicion with positive bile stain aspiration might lead to early diagnosis. Open cholecystectomy is a safe option of treatment in a resource poor facility and has a good outcome.

References

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