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Case Report

Gastric perforation following blunt abdominal trauma

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ABSTRACT

Gastric perforations following blunt abdominal trauma are rare, accounting for <2% of all blunt abdominal injuries. Isolated blunt gastric ruptures are uncommon. They are usually associated with other solid visceral injuries. Injuries to the stomach are associated with the highest mortality of all hollow viscus injuries. Severity of the injury, timing of presentation and presentation following the last meal as well as concomitant injuries are important prognostic factors. Imaging modalities may be unreliable in making a diagnosis and thus clinical vigilance is mandatory. We present a patient with gastric perforation following blunt abdominal trauma and review the literature.

Introduction

Blunt abdominal trauma (BAT) following assaults, motor vehicle accidents and falls not uncommonly results in solid organ (liver, spleen and kidney), diaphragmatic, pancreatic and retroperitoneal injury. Hollow viscera injuries to duodenum, jejunum, urinary bladder and the colo-rectum are also not uncommon with an incidence that varies between 4 to 15% [1,2]. However, by contrast, gastric perforations following BAT have an incidence of between 0.02 to 1.7%. A multicentre retrospective analysis of blunt gastric injuries from four trauma centres in Brazil over a 14 year period yielded only 33 cases of gastric perforation [3]. The rarity of gastric perforation developing following BAT in civilian practice together with the inconsistent diagnostic yield from standard investigations has led to this condition being invariably recognised at laparotomy. In this case report we describe an anterior gastric perforation following BAT due a motor vehicle accident.

Case report

A 29 years old male patient presented with severe abdominal pain and distention as a result of blunt abdominal trauma sustained in a motor vehicle accident 2 h previously. At presentation, the patient was fully conscious, normotensive with a pulse rate of 112/min. The haemoglobin was 13.6 g/dL. Multiple bruises and abrasions were evident over the anterior chest and epigastrium. The abdomen was distended and peritonitic. The patient was also tender over the left lower anterior chest wall; air entry was normal. Radiological investigations revealed a pneumoperitoneum and fractures involving ribs 10, 11 and 12 along the left anterior chest wall.

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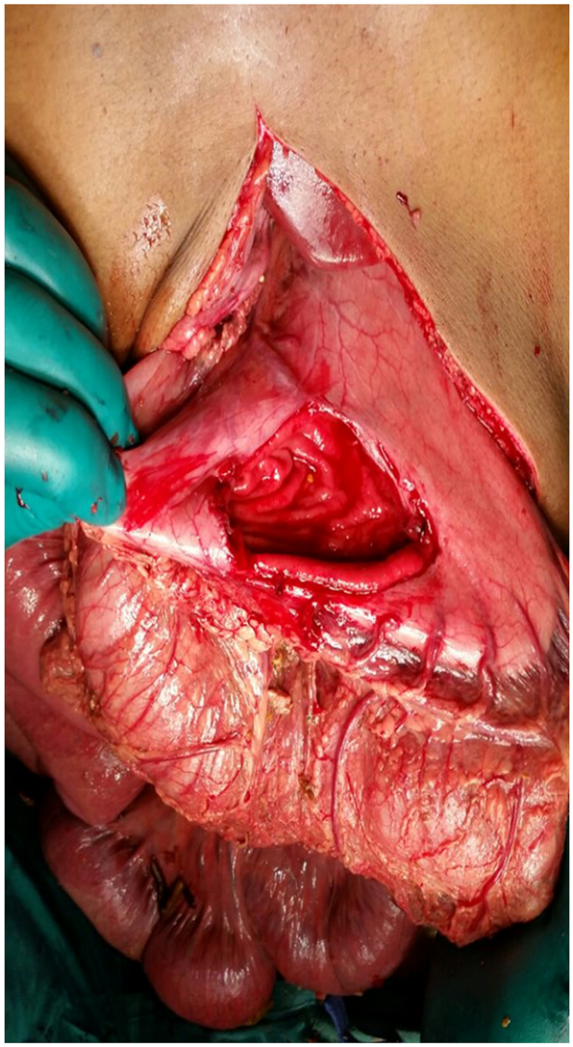

Fig. 1. Full thickness perforation along the anterior gastric wall measuring 5 cm extending proximally from the juxta-pylorus along the longitudinal gastric axis.

At laparotomy serosanguinous fluid with undigested food particles was noted. A full thickness anterior gastric perforation measuring 5 cm extended proximally from the pylorus along the longitudinal gastric axis (Fig. 1). This was classified as a Grade II gastric injury (Table 1) [4].

Haematomas were noted over the transverse colon, jejunum and ileum and were managed conservatively. The gastric perforation was repaired in a standard fashion. The patient made an unremarkable and was discharged on the 7th post-operative day.

Table 1
Grading of gastric injuries [4].

	Grading of gastric injuries	
Grade I	Intramural hematoma < 3 cm Partial thickness laceration	
Grade II	Laceration:	<2 cm in GE junction/pylorus <5 cm in proximal one-third <10 cm in distal two-third
Grade III	Laceration:	>2 cm in GE junction/pylorus ≥5 cm in proximal one-third ≥10 cm in distal two-third
Grade IV	Vascular:	Tissue loss/devascularisation ≤ two-third stomach
Grade V	Vascular:	Tissue loss/devascularisation ≥ two-third stomach

Discussion

The infrequency of gastric perforation following BAT is due to several factors that include the protective anatomy afforded by the thoracic cage, the relative mobility of the stomach and gastric mural thickness [5].

Classically, gastric perforations due to BAT have been attributed to 3 mechanisms:

1) External compression resulting in an acute and intense rise of intra-abdominal pressure. This mechanism applies in particular to a distended stomach with a consequent massive increase of intra gastric pressure. This mechanism may explain the development of gastric perforation following the Heimlich manoeuvre.
2) Rapid deceleration causes differential movement among adjacent structures resulting in shear forces causing hollow, solid, visceral organs and vascular pedicles to tear, especially at relatively fixed points of attachment.
3) Crushed intra-abdominal contents between the anterior abdominal wall and the vertebral column or posterior thoracic cage.

Gastric perforations due to BAT may develop in any location of the stomach. The most common location for gastric perforation is the anterior wall (40%) followed by the greater curvature (23%), lesser curvature (15%) and posterior wall (15%). Such perforations are invariably solitary; to date only 3 cases of a double gastric perforation following BAT have been described (refer Table 2) [6].

Gastric perforations following BAT are usually associated with other intra- and extra-abdominal injuries; isolated blunt gastric ruptures are uncommon. The most common associated injury is to the spleen, followed by thoracic injury [5,6]. The successful management of gastric perforations due to BAT is contingent on an accurate clinical evaluation. Injuries to the stomach are associated with the highest mortality of all hollow viscus injuries [7]. Morbidity and mortality increases parallel with time to operative intervention (intervention within 8 h is associated with a 2% mortality, intervention within 8 to 16 h with a 9% mortality, intervention within 16 to 25 h, a 17% mortality and intervention after 24 h over 30% mortality) [6]. The overall reported mortality ranges from 0–66% [5,6,8].

The majority of complications are septic in nature with the reported incidence of intra-abdominal abscesses being up to 24% [9]. In contrast to the fasted patient with a low gastric pH and bacterial load, the fed patient has a higher gastric pH that predisposes to a greater bacterial load with potential to predispose to much contamination and infective complications. In the instance of unequivocal peritonitis prompt laparotomy will afford early diagnosis and appropriate treatment. However, the physical examination may be misleading when the patient is intoxicated or has associated injuries (head injury, spinal cord injury, thoracic or long bone trauma) [10].

Although shock on presentation has been reported as a fairly common occurrence, it was reported in <20% of cases [3]. Vassey et al. have suggested that aspiration of dark coloured fluid on peritoneal lavage or paracentesis is probably the best pre-operative diagnosis of gastric perforation [11]. Serum amylase has been suggested as a biochemical marker to diagnose upper gastrointestinal rupture [12]; the unpredictability of this test makes this an unreliable diagnostic marker. Plain abdominal radiographs may show pneumoperitoneum, retroperitoneal air or the obliteration of psoas muscle shadow, which though non-specific, will prompt surgical intervention.

When there is a diagnostic dilemma, recourse to ultrasonography and computed tomography is advised. Ultrasonography has value in identifying intra-abdominal fluid which, in the presence of haemodynamic instability, strongly suggests free blood and an indication for laparotomy. The failure to reliably distinguish hollow visceral injury from solid visceral injury is vital and compromises the decision to undertake laparotomy, particularly in haemodynamic patients. Presently, helical (spiral) computed tomography (CT) is advocated when there is diagnostic doubt in the setting of haemodynamic stability. The alarm features on CT scan which prompt further intervention (diagnostic peritoneal lavage if single abnormality, laparotomy if several abnormalities) include unexplained intraperitoneal fluid, pneumoperitoneum, bowel wall thickening, mesenteric fat stranding, mesenteric haematoma, extravasation of bowel contents and free blood [9]. Notwithstanding this, clinical vigilance is mandatory as a negative CT scan may miss a bowel perforation in 13% of cases [13].

Table 2
Summary of the features of gastric injury due to blunt trauma.

Mechanisms of injury	Increase in intra gastric pressure Deceleration shear force tears Crush between anterior abdominal wall and vertebra
Location of injury	Anterior wall (40%) Greater curve (23%) Lesser curve (15%) Posterior wall (15%)
Most common associated injury	Spleen
Mortality	Increases with time to operative intervention
Complications	Abdominal abscess (24%) [more common in post-prandial trauma]
Diagnosis	Abdominal radiograph: pneumoperitoneum Peritoneal paracentesis: dark coloured fluid Computed tomography: free fluid with thickened wall and mesenteric fat standing
Management	According to grade (see Table 1)

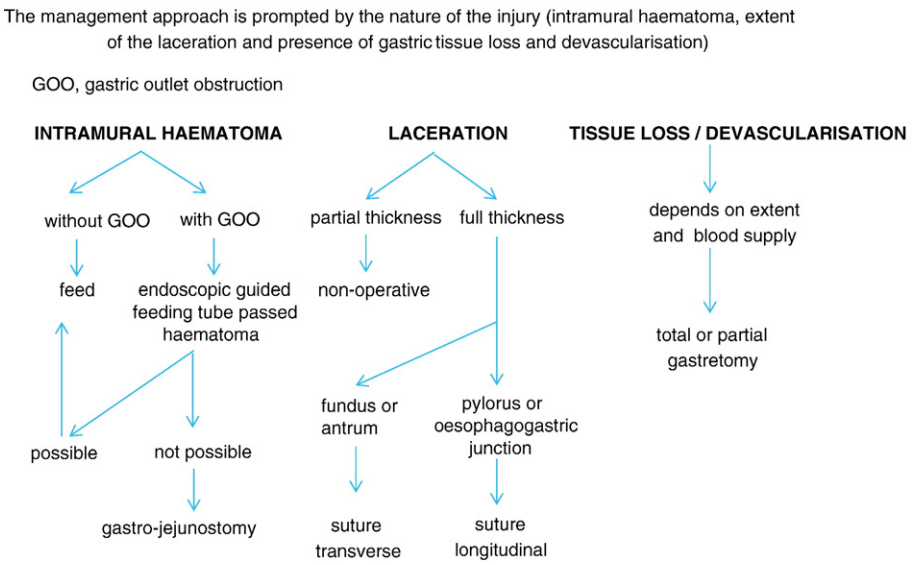

Fig. 2. Suggested management of blunt gastric injury.

The surgical management of gastric injury is largely dictated by the grade of injury [which reflects the nature (haematoma vs laceration), extent and location of the injury – Table 1] as well as by the presence of associated injuries. A management algorithm has been suggested (Fig. 2) and a summary of the literature is provided in Table 2.

At laparotomy it is mandatory to exclude a separate gastric laceration (for example, along the posterior gastric wall). Grades 1 to 3 gastric injuries (the majority of gastric injuries) are amenable to primary repair; a 2 layer closure is advocated to effect haemostasis. Grade 4 (tissue loss with devascularisation affecting <50% of stomach) and Grade 5 (tissue loss with devascularisation affecting >50% of stomach) gastric injuries are uncommon, associated with other organ and major vascular injuries; affected patients rarely reach hospital alive.

In the light of the extent of the injuries, primary repair will not be feasible in patients with Grades 4 and 5 gastric injuries. Depending on the location of the tissue loss (proximal vs distal stomach) and extent of devascularisation, sub-total or rarely total gastrectomy may have to be undertaken. The options to restore gastrointestinal continuity will be influenced by the presence of associated injuries (to duodenum, bile duct and pancreas) and include a gastro-duodenostomy, gastro-jejunostomy or a Roux-en-Y reconstruction.

Conclusion

Severity of the injury, timing of presentation and presentation following the last meal as well as concomitant injuries are important prognostic factors. Prompt diagnosis and timely intervention greatly limits mortality and morbidity associated with blunt gastric injuries. Majority of which are septic in nature.

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