A report of three cases and review of the literature on rectal disruption following abdominal seatbelt trauma

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ABSTRACT
Seatbelt associated blunt trauma to the rectum is a rare but well recognised injury. The exact mechanism of hollow visceral injury in blunt trauma is unclear. Stress and shear waves generated by abdominal compression may in part account for injury to gas containing structures. A ‘seatbelt sign’ (linear ecchymosis across the abdomen in the distribution of the lap belt) should raise the suspicion of hollow visceral injuries and can be more severe with disruption of the abdominal wall musculature.

Three consecutive cases of rectal injury following blunt abdominal trauma, requiring emergency laparotomy and resection, are described. Lumbar spine injury occurred in one case and in the other two cases, there was injury to the iliac wing of the pelvis; all three cases sustained significant abdominal wall contusion or muscle disruption. Abdominal wall reconstruction and closure posed a particular challenge, requiring a multidisciplinary approach. The literature on this topic is reviewed and potential mechanisms of injury are discussed.

KEYWORDS
Blunt abdominal trauma – Seatbelt – Hollow visceral injury – Stress waves – Shear waves – Degloving

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Hollow visceral injury following vehicular blunt abdominal trauma is well recognised but relatively unusual and injury to the rectum is seen rarely1,2 with an incidence of 0.1–0.5% in the civilian population.3–6 With decreasing mortality from motor vehicle collisions, largely on account of seatbelt legislation, deceleration injury related to the restraining effect of the seatbelt has become more common.6,7

The presence of a ‘seatbelt sign’ (linear ecchymosis in the abdominal wall from seatbelt trauma) is considered to be a strong indication for exploratory laparotomy, even if cross-sectional radiological imaging is negative, in view of the likelihood of intestinal injury.8 Intestinal injury following blunt abdominal trauma is also considered more likely in the presence of lumbar spine injury and the term ‘seatbelt syndrome’ has been coined for this combination.9

The exact mechanism of hollow visceral injury in blunt trauma, however, is unclear. While sudden trunk flexion and extension combined with inertial resistance during deceleration may lead to rapid compression and tearing, the true mechanism of rectal injury remains speculative. Stress and shear waves generated by abdominal compression may in part account for injury to gas containing structures,10 and they deserve further consideration in this regard.

Three consecutive cases of rectal injury following blunt abdominal trauma, requiring emergency laparotomy and resection, are described. Lumbar spine injury occurred in one case and in the other two cases, there was injury to the iliac wing of the pelvis; all three cases sustained significant abdominal wall contusion or muscle disruption. The literature on this topic is reviewed and potential mechanisms of injury are discussed.

Case 1
A 56-year-old female driver was involved in a head-on collision with a combined speed of 40mph. On admission, she was alert but hypotensive, and complained of neck, abdominal and pelvic pain. On examination, there was bruising from seatbelt compression on the lower abdomen below the umbilicus, a puncture wound over the right anterior superior iliac spine and pelvic tenderness.

Computed tomography (CT) revealed bilateral rib fractures, oblique fractures of the manubrium sternum and the superior body of the manubrium, small traumatic pneumatoceles in the right upper lobe, an open comminuted right iliac wing fracture, a closed left iliac wing fracture and dis-
section injury of the right common iliac artery. The small bowel and right colon were seen to have herniated through the right iliac wing defect to lie subcutaneously but there were no signs of visceral perforation or haemorrhage (Fig 1). In addition, there was disruption of the anterior abdominal wall muscles, central spinal canal stenosis with fracture dislocation at C5/C6, contusion of the left cerebral frontal lobe, a comminuted fracture of the right olecranon and a small avulsion fracture of the medial epicondyle of the right humerus.

The patient underwent combined midline laparotomy and pelvic fracture surgery. Reduction of the caecum and small bowel back into the abdomen allowed fixation of the comminuted pelvic fracture through an extension of the external wound (Fig 2). There was complete seromuscular degloving of the rectosigmoid and associated mesenteric injury. The upper rectum and sigmoid colon were mobilised, and the rectosigmoid colon was resected, leaving the stapled ends inside the abdomen. The omentum was mobilised off the colon and used to cover the right pelvic defect. The abdomen was closed with the ABThera™ (KCI, Gatwick, UK) open abdomen negative pressure therapy system and the patient was transferred to the intensive care unit.

At the ‘relook’ laparotomy 48 hours later, a circular stapled colorectal anastomosis was performed uneventfully. Partial closure of the iliacus muscle was possible, augmented by omentoplasty, and several days later, a tensor fascia lata flap was raised to fill the wound over the iliacus. The rectus sheath defect was closed with a mesh followed by an extensor thigh muscle flap to cover the external pelvic and abdominal wall defects. The tensor fascia lata flap failed two weeks later, requiring debridement. A vastus lateralis flap was raised from the left thigh and inserted successfully into the defect.

The patient underwent a two-stage C5/C6 corpectomy and C4–C7 fixation a week later. She was eventually discharged to a spinal rehabilitation unit with a healed abdomen and normal bowel function.

Case 2
A 77-year-old female front seat passenger was involved in a head-on collision at a combined speed of 60mph. Clinical assessment revealed an open fracture of the left pelvic ilium, ecchymosis in the distribution of the lap component of the seatbelt in line with the anterior superior iliac spines and signs of peritonism on abdominal palpation. Whole body CT confirmed C5 posterior and C7 anterior left tubercle fractures, a displaced fracture of the left occipital condyle, bilateral rib fractures, a manubrium sternal fracture and a comminuted fracture of the left ilium with associated bilateral fractures of the lumbar transverse processes. There was also pneumoperitoneum from a possible sigmoid perforation with associated mesenteric injury (Fig 5).

The patient proceeded to a midline laparotomy, at which there was free blood, small bowel content and faeces in the pelvis, with almost complete transection of the lower
sigmoid colon. There was a small perforation of the distal ileum, two small bowel mesenteric injuries (one associated with a 10cm segment of ischaemic mid-ileum) and several small bowel intramural haematomas.

The rectosigmoid colon and ischaemic small bowel were resected, leaving the closed stapled ends inside the abdomen. The small bowel perforation was repaired primarily with sutures. Following lavage, an ABThera™ negative pressure dressing was inserted and the patient transferred to the intensive care unit.

At the relook laparotomy 48 hours later, the small bowel was healthy and was reanastomosed. The upper rectum and distal sigmoid ends were resected, a circular stapled colorectal anastomosis was performed, with a covering ileostomy, and the abdomen was closed.

The spinal injury was managed conservatively. The patient was eventually discharged to outpatient follow-up with plans for ileostomy reversal.

Case 3

An 18-year-old, belt-restrained, rear seat passenger was admitted to a neighbouring hospital following a high speed, head-on collision. On abdominal examination, a lap seatbelt sign was noted (Fig 4) and she was peritonitic. She sustained intra-abdominal injuries, including a thrombus in the abdominal aorta, sigmoid and small bowel perforations, and fracture dislocation of the L3/L4 vertebrae. The anterior abdominal wall muscles were also disrupted. An emergency laparotomy was undertaken at which there were perforating injuries to a section of ileum and rectosigmoid colon. These were managed by resection, leaving the closed stapled ends inside. An aortic thrombectomy was also performed and the abdomen was closed temporarily prior to hospital transfer.

At the relook laparotomy 48 hours later, small bowel anastomosis and a circular stapled colorectal anastomosis were carried out uneventfully. The abdominal wall muscles from the midline to the flanks were reconstituted by suture repair over suction drains (Fig 4). Spinal fixation was undertaken after a further 48 hours and the patient ultimately made a full recovery.

Discussion

Abdominal seatbelt injuries are becoming more common as mortality from motor vehicle collisions diminishes following improvements in road safety. These cases highlight the importance of recognising the likelihood of visceral trauma, even if CT appears normal. The presence of abdominal pain with external signs of seatbelt compression should lead to a low threshold for laparotomy, especially when there is concomitant musculoskeletal injury. The energy necessary to cause a flexion distraction spinal injury and disruption of the abdominal wall during deceleration is likely to be sufficient to produce visceral trauma. The presence of a seatbelt compression sign may be predictive of visceral injury in over 60% of cases, regardless of skeletal injury.

Visceral injuries from blunt abdominal trauma occur from either high speed, low momentum impacts (such as blast injuries in the battlefield) or low speed, high momentum impacts that predominate in road traffic collisions. Intestinal injuries are found in 5–16% of laparotomies performed for blunt abdominal trauma but the mechanism of hollow viscus injury following blunt abdominal trauma is debatable. Colorectal injuries are even rarer, with an incidence in the civilian population reported at 0.1–0.5% in some series. Seatbelts are designed to protect by
dissipating energy at impact over a larger surface area through contact with the most resistant body structures. However, body inertia and compression generated on impact lead to deformation of the abdominal and chest wall with a rise in intra-abdominal and thoracic pressures, and the generation of stress waves and shear waves.

Stress waves, generated from a rapid rise in air pressure, are longitudinal pressure waves that travel faster than the velocity of sound in tissues. Peak body wall velocity and acceleration determine the magnitude of the stress wave, and the amount of energy transmitted rises exponentially with the velocity at impact. The ability of stress waves to be reflected (and reinforced) enhances their injury potential.

Multiple air–tissue interfaces in the gastrointestinal tract render the gut particularly susceptible to injury owing to differences in acoustic impedance. Spalling occurs as the stress wave is reflected at fluid–gas interfaces, such as exist in the wall of the intestine, creating tension and injury as the energy is given up. Implosion occurs when the stress wave, coming into contact with small compressible air bubbles, creates very high local pressures and local damage as the pressure falls, allowing the bubbles to rapidly re-expand. The ensuing intraparticulate disruption leads to intramural haematomas and perforations.

Shear waves are transverse waves of long duration and low velocity that cause gross distortions of tissues and viscera. Shearing results in tissue disruption if the energy applied to a structure is greater than its tensile strength or elasticity, and injury is produced by asynchronous motion of adjacent connected structures with inertial differences, stretching at sites of attachment or by collision of viscera with more resistant structures (eg bone). Consequently, shearing mechanisms account for mesenteric injuries, aortic rupture and some solid organ injuries, and possibly also disruption between the layers of the bowel wall.

It is probable that the rectosigmoid disruption encountered in the cases described occurred as a result of a combination of these mechanisms. Stress wave injury may be more likely if the rectum contains gas at the moment of impact and, conversely, shear wave injury may occur if the colon is loaded with faeces. Shearing of mesenteric attachments, with haematoma formation or ischaemia, or shearing due to fixation by adhesions from previous surgery (as in the first case) are also likely to have contributed. The increased susceptibility to injury or perforation because of pre-existing colonic pathology (such as the diverticulosis seen in the second case) remains speculative but it is important when considering surgical treatment.

Less likely in the reported cases is rectal injury as a result of pelvic fracture. However, in more severe pelvic ring disruption, this may be important, with crush injury or perforation by bone fragments; there was a higher risk of small bowel perforation from this mechanism in the first case.

Three-stage damage control surgery was used effectively in managing these three patients' abdominal injuries, allowing rapid control of haemorrhage and contamination source control, the emphasis being on resuscitation and correction of deranged physiology (predominantly the lethal triad of hypothermia, acidosis and coagulopathy) rather than immediate restoration of normal anatomy. Leaving the abdomen open (laparostomy) aims to prevent intra-abdominal hypertension and abdominal compartment syndrome, and the use of commercially available negative pressure therapy systems seems to be most effective in achieving this and ultimate primary fascial closure as well as improving overall long-term survival. In the reported cases, this technique also facilitated pelvic fracture fixation and planning for definitive delayed abdominal wall repair and reconstruction.

Conclusions

These cases illustrate the need to be vigilant in cases of blunt abdominal trauma and to have a high index of suspicion for hollow visceral injury in the presence of signs of significant deceleration from a seatbelt, even when CT is negative or equivocal. There should be a low threshold for laparotomy under such circumstances as the consequences of overlooking hollow visceral injury and risking intraperitoneal contamination by intestinal content may be considerable, particularly in patients with multiple injuries.

Conversely, the management of cases with subtle abdominal signs, haemodynamic stability and negative initial CT should include serial examinations, close observation and monitoring of surrogate markers for compromised tissue perfusion (eg lactate). Changes in patient status should prompt reimaging or surgery. Examples of such delayed manifestations include a hollow visceral wall or mesenteric haematomas leading to ischaemia and, eventually, perforation resulting in peritonitis. Biswas et al detail a list of considerations used in their practice when assessing cases where a seatbelt injury is coupled with subtle abdominal signs. They suggest that the presence and pattern of visceral injury can be foretold to some degree by the site of the seatbelt sign and abdominal wall bruises and the position of the patient in the vehicle at the time of impact, and these factors should be documented clearly at the time of initial assessment.

Management will usually need to be via a damage control approach with a high chance of ultimate restoration of intestinal continuity.

References
