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Case Report

CASE REPORT

Seatbelt syndrome with superior mesenteric artery syndrome: leave nothing to chance!

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Abstract

The introduction of seatbelts to legislation has dramatically reduced mortality and morbidity from motor vehicle accidents. However, overtime evidence has emerged of 'seatbelt syndrome' (SBS), particularly in the paediatric population. The report describes the diagnosis and management of this rare injury in a 12-year-old boy who sustained a chance lumbar fracture and mesenteric tear resulting in small bowel obstruction. His stay was subsequently complicated by superior mesenteric artery (SMA) syndrome. This is the first documented case of SBS with SMA syndrome. High index of suspicion and continuity of care, particularly in the setting of a 'seatbelt sign', is paramount to timely diagnosis and management.

INTRODUCTION

'Seatbelt syndrome' (SBS) was first coined by Garrett and Braunstein in association with adult lap belt injuries [1]. It includes abdominal wall contusions known as 'seatbelt sign', hollow viscus injury and flexion–distraction lumbar spine injuries. The paediatric population is particularly vulnerable due to their physical and behavioural characteristics [2].

This report presents the diagnosis and surgical management of a 12-year-old boy who was involved in a high-speed motor vehicle accident with an inappropriately fitted seatbelt. His stay was complicated by superior mesenteric artery (SMA) syndrome requiring prolonged total parental nutrition (TPN). The importance of maintaining high index of suspicion with frequent physical examination for timely diagnosis is highlighted. The literature on paediatric SBS is reviewed.

CASE REPORT

A.O. is a 12-year-old boy who was a backseat passenger, involved in a high-speed (70 km/h) motor vehicle collision. He was only wearing the lap portion of the lap-sash seatbelt. There was no loss of consciousness; however, immediately following the accident, he had complete loss of motor and sensory function to his lower limbs. On presentation to hospital, he was haemodynamically stable with a Glasgow coma scale of 15. Seatbelt bruising was noted across lower abdomen crossing his anterior superior iliac spine. Remainder of abdominal examination was unremarkable. On palpation of his spine, an upper lumbar spine step and left paraspinal contusions were noted. There was 0/5 power in both lower limbs and loss of fine-touch and pinprick below L1 dermatome. There was no voluntary anal contraction and an absent anocutaneous reflex. Remainder of full body examination was unremarkable. Routine blood investigations are highlighted in Table 1.

Whole body computed tomography (CT) demonstrated only identified a Grade 1 left kidney laceration with retroperitoneal haematoma and flexion/distraction chance-type fracture of L2–L3 vertebrae. Magnetic resonance imaging was performed, which showed complete rupture of all ligaments at L2–L3 level and unstable fracture. He underwent L2–L3 posterior spinal fusion with autologous bone graft harvested from iliac crest on the same day.

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Over the next 4 days, his abdomen remained soft and non-tender. However, he progressively began to have small bilious vomits, and on Day 5, his abdomen was noted to be more distended. A repeat abdominal X-ray and CT was suggestive of acute obstruction with a transition point in the small bowel. He was taken for exploratory laparotomy on Day 5, which identified an internal herniation of jejunal loops through a mesocolic laceration (Fig. 1). There was a short segment of involved jejunum, which was slightly narrower in calibre but patent and well perfused and left *in situ* after reduction and repair of the mesocolic defect.

Initially he progressed well tolerating per oral (PO) intake. However, 10 days post-op his abdominal pain and nausea returned. Repeat imaging suggested a partial bowel obstruction and repeat laparotomy identified significant adhesions and stenosis of the previously involved segment of jejunum (Fig. 1C). He was commenced on TPN, and PO intake was slowly reduced post-operatively. His PO intake remained poor, and he continued to have ongoing intermittent abdominal pain with significant post-prandial discomfort. A barium meal and follow-through study was performed 1 week post-relaparotomy, and a diagnosis of SMA syndrome was made (Fig. 2).

The nasogastric (NG) was advanced past the point of obstruction (now nasojejunal tube), and feeding was commenced. Since his admission, he had lost over 10 kg at this point. He was eventually tolerating PO intake and off TPN one 40 days post-admission and was transferred for rehabilitation and ongoing bowel management with regular laxatives.

Table 1: Admission blood tests

Na	139 mmol/l
Potassium	4.3 mmol/l
Chloride	104 mmol/l
Bicarbonate	23 mmol/l
White cell count	14.3×10^9/l
Neutrophils	12.3×10^9/l
Lymphocytes	0.7×10^9/l
Monocytes	1.3×10^9/l
Eosinophils	0.0×10^9/l
Liver function tests	
Bilirubin	6 µmol/l
Alanine aminotransferase	149 U/l
Aspartate aminotransferase	67 U/l
Alkaline phosphatase	179 U/l
Gamma-glutamyl transpeptidase	13 U/l
Coagulation study	
Prothrombin time	14.7 s
International normalized ratio	1.0
Activated partial thromboplastin time	29.7 s
Amylase	74 U/l
Lipase	67 U/l

DISCUSSION

Paediatric cases of 'SBSs' started appearing since 1980s when child restraint became common in car seats [1, 3–5]. Small bowel mesenteric tear and perforations are the most common pattern of abdominal injury [5]. Jejunum and ileum are typically affected due to their mesenteric attachment to posterior abdominal wall [1]. Two main types of lumbar injuries can occur: compression and chance fractures [2]. The mechanism of injury relates to migration of lap belt towards the anterior abdominal wall due to small pelvic size, tendency of children to sit further over edge of seat and weaker abdominal muscle layers. With sudden deceleration, direct compression of viscera occurs between the seatbelt and the spine. Lumbar injuries result from the anterior position of the flexion fulcrum relative to the spine and the more sagittal placed spinal facets in the lower thoracic and lumbar joints [6]. Even with appropriate three-point seatbelt, patients can 'submarine' under the seatbelt, which then acts as a lap belt [6]. In the case presented, however, the seatbelt was worn inappropriately without the sash component, effectively converting to a two-point seatbelt.

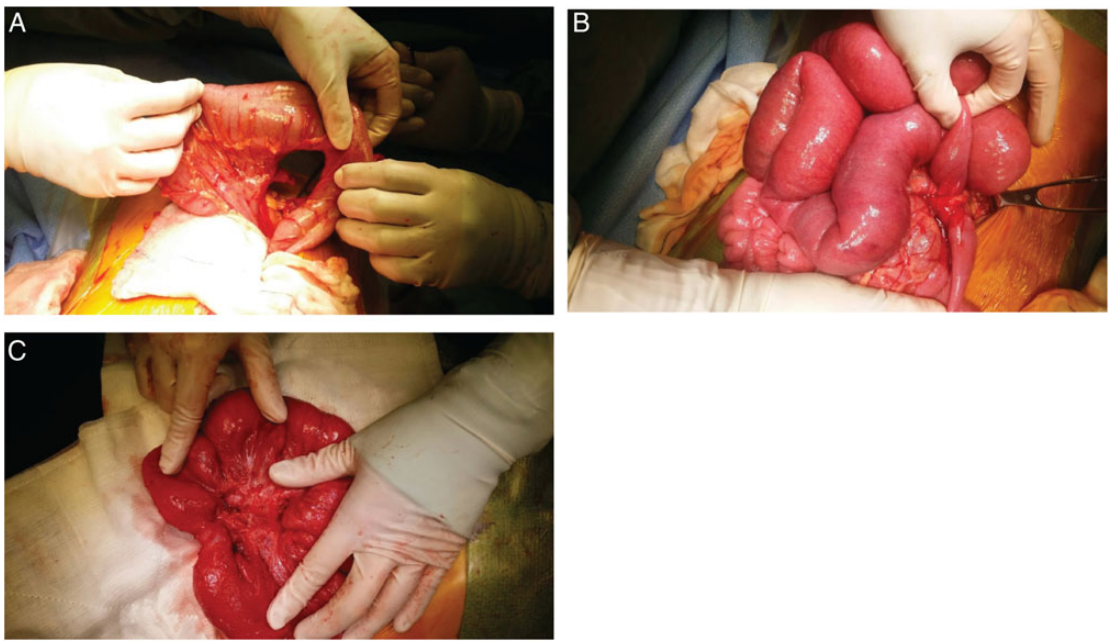

Figure 1: Intra-operative pictures (**A**) internal hernia of jejunal loop resulting in small bowel obstruction with proximal bowel dilatation. (**B**) Mesenteric defect in mesocolon. (**C**) Subsequent stricture in area of jejunal loop previous caught in mesenteric defect.

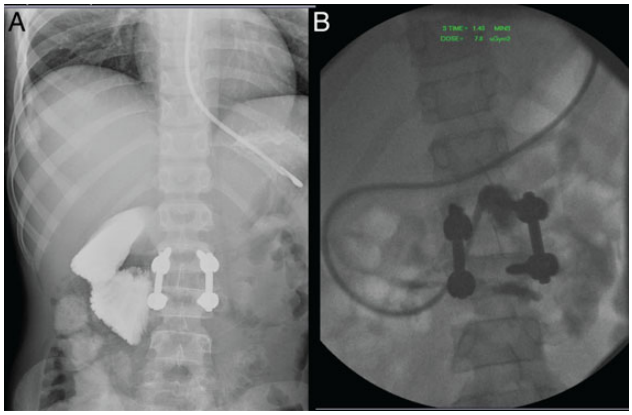

Figure 2: (**A**) Barium swallow through NG demonstrating SMA syndrome with cut-off at D3. (**B**) Advancement of NG tube past D3 into jejunum prior to commencing feeds.

In SBS, up to 80% of flexion–distraction spinal injuries have concomitant intra-abdominal injury [7]. Relying on CT and ultrasound can lead to delay in diagnosis [4]. In the case presented, initial abdominal CT did not identify any significant gastrointestinal pathology. The presence of 'seatbelt sign' should heighten index of suspicion. Abdominal wall ecchymosis is associated with significant intra-abdominal injury (particularly hollow viscus injury) in up to 75% of cases [8]. Patients with 'seatbelt sign' versus those without are 3, 13 and 22 times more likely to have intra-abdominal, gastrointestinal and pancreatic injuries. Vascular injuries, although more rare, have also been documented including to abdominal aorta, superior mesenteric and iliac vessels [9]. When diagnosis remains uncertain, there is no substitute for frequent examinations. Early exploratory laparotomy is encouraged to prevent delay in potentially lethal outcome [8].

SMA syndrome (Wilkie's syndrome) results from compression of the third part of the duodenum from the overlying SMA. This typically results from loss of cushioning from retroperitoneal fat and lymphatic tissue, anatomically reducing the angle between SMA and duodenum. Known risk factors include significant weight loss, spinal cord injuries and burns. Only a handful of case reports exist of SMA syndrome in paraplegic patients [10]. In the case presented, several risk factors may have predisposed to it including: over 10 kg weight loss, spinal surgery and use of extension spinal braces. Physicians need to be aware of this potential diagnosis in patients who fail to return to normal oral intake.

This report highlights an important case of 'SBS' in a paediatric patient. In the presence of 'seatbelt' sign, high index of suspicion and frequent re-examination are necessary. Low threshold for exploratory laparotomy is recommended. SMA syndrome is a rare complication but must be kept in mind in this cohort of patients. Paediatricians, surgeons and public health officials need to work together and continue to provide community education and campaigning in appropriate fitting of seatbelts in children.

CONFLICT OF INTEREST STATEMENT

None declared.

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