CASE REPORT

Gastroenterology



An indolent case of traumatic bowel injury as a superior mesenteric artery syndrome mimic

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Abstract

Traumatic bowel injury is an uncommon injury pattern that can have a delayed presentation after an initial trauma hospitalization and present to pediatricians with nonspecific symptoms. This syndrome is often missed and can mimic other common gastrointestinal conditions. Our case presents a previously healthy 16-year-old girl with recent trauma admission who re-presented to the hospital with a presumed superior mesenteric artery syndrome and had a mixed response to initial management. Given persistent symptoms despite standard care, subsequent management consisted of an exploratory laparotomy that led to findings of a strictured segment of the bowel that was resected and led to clinical recovery. These patients can initially present with normal imaging and have an evolving inflammatory-mediated process due to microvascular injury and abscess formation. These injuries should be included in the differential diagnosis of patients with nausea, vomiting, abdominal pain, weight loss, and fever in the setting of recent blunt abdominal trauma.

KEYWORDS

abdominal imaging, bowel stricture, trauma

INTRODUCTION 1

Traumatic bowel injury (TBoI) is an uncommon injury pattern after trauma, only present in 1.2% of cases of blunt abdominal trauma. It can present both immediately, in an acute rupture, or in a delayed manner, with microvascular or mesenteric injury. 1,2 In delayed presentations, symptoms are often nonspecific like anorexia, nausea, vomiting, weight loss and generalized abdominal pain which can mimic other gastrointestinal disorders, particularly with recent history of critical illness.^{3,4} These injuries can require surgical intervention and are often difficult to diagnose because they may present to pediatricians and pediatric gastroenterologists outside the context of the initial trauma hospitalization. We describe a case of TBol discharged and readmitted to the hospital with an initial diagnosis of superior mesenteric artery (SMA) syndrome and a prolonged course.

CASE REPORT

A previously healthy 16-year-old girl was a restrained passenger in a high-speed motor vehicle collision (MVC) arriving with a Glasgow Coma Scale of 7. An initial survey of injuries revealed anterior abdominal wall ecchymoses (seatbelt sign), chance fracture of L4 and additional fractures of the L1-L4 right transverse processes, traumatic brain injury, and cauda equina syndrome. Computed Tomography of the Abdomen and Pelvis (CTAP) showed trace-free fluid without signs of intraabdominal injury to the bowel, such as free air or pneumatosis. The patient was admitted to the surgical intensive care unit and underwent a spinal fusion, with a repeat CTAP on post-trauma day (PTD) 6 showing resolution of previous findings. The postoperative course was complicated by fever, leukocytosis, abdominal pain, and nausea on PTD 8, thought to be related to

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postoperative infection and ileus. Symptoms resolved after a 7-day course of piperacillin-tazobactam, and she was discharged to an inpatient rehabilitation facility.

Two weeks after discharge, the patient developed nausea, bilious vomiting, low-grade fevers, and a 30% weight loss compared to baseline. Workup was significant for a CTAP showing a fluid-filled dilation of the duodenum, with a normal angle of the SMA and aorta (35°) and elevated inflammatory markers. On readmission, the patient had a normal upper gastrointestinal (UGI) series without small bowel followthrough and normal abdominal ultrasound. Given bilious vomiting, recent critical illness, weight loss, and duodenal dilation, the patient was diagnosed with SMA syndrome and potential concomitant gastroenteritis and admitted to the general pediatrics service, receiving nasojejunal tube feeding, parental nutrition, and prokinetics. After the initiation of tube feeding, the symptoms resolved, and a repeat CTAP showed a reduced angle of the SMA and aorta at less than 20° and distension of the first and second portions of the duodenum.

After 2 weeks, the patient re-developed similar symptoms. The abdominal film showed tube migration proximally, terminating in the third portion of the duodenum. Repeat UGI series showed a 3-cm focal segment stenosis of the duodenojejunal junction, hyperperistalsis of the proximal bowel, and minimal contrast passage on small bowel follow-through—likely a TBoI with development of jejunal stricture (Figure 1).

The patient was rehabilitated for several weeks with more than 60 kilocalories/kilogram/day of total parental

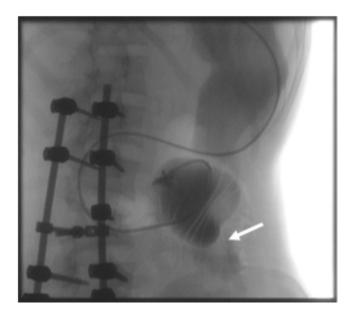


FIGURE 1 Fluoroscopic UGI series image of a dilated bowel segment concomitant to a stricture of the bowel at the duodenojejunal junction, with subsequent minimal contrast passage (marked with arrow). UGI, upper gastrointestinal.

nutrition before exploratory laparotomy on PTD 97. Laparotomy revealed copious adhesions, a short segment of strictured proximal jejunum with a small abscess just past the duodenojejunal junction, and an edematous fourth portion of the duodenum. The remaining bowel appeared grossly normal. The patient underwent resection of the affected segment with anastomosis. Histopathology showed transmural mixed inflammatory infiltrates with neutrophilic predominance, fibrosis, edema, and hypoxic-ischemic changes of the mucosa. The postoperative course was uncomplicated, and the patient was discharged home tolerating a regular diet orally with return to baseline at 3-month follow-up.

3 | DISCUSSION

The seatbelt syndrome has been described in the literature as a triad of the "seatbelt sign" on exam, intraabdominal injury (IAI), and spinal fractures for restrained child victims of MVCs. Nearly half of the patients with these Chance-type spinal fractures will have associated IAI, with the majority being injuries of the bowel and mesentery.⁵ In a study of patients presenting to a pediatric trauma center, a quarter of patients with gastrointestinal trauma initially presented with a reassuring exam, with one third of this group having a normal CTAP on admission and others with minor bowel edema or unexplained free fluid that did not require immediate laparotomy. Ultimately, a constellation of increasing peritoneal irritation, pain, fever, and leukocytosis leads to a delayed diagnosis of TBol.²⁻⁴ Previous studies have noted that the severity of the TBol is not necessarily correlated to the severity of the mechanism of injury but rather may be more related to proper seatbelt positioning and implementation of three-point seatbelts.3 The phenomenon of delayed presentation despite normal imaging appears more specific to children, as negative CTAP has a high sensitivity for ruling out TBol in adults.⁶

Our patient had a more indolent presentation than previously seen in the literature, perhaps owing to a milder predisposing microvascular injury. Over time, the stricture evolved with corresponding chronic inflammation and abscess formation, and later, it was more readily seen on imaging. With weight loss and critical illness, there is likely a component of SMA syndrome also involved in the pathogenesis of our patient, which may explain our repeated observations of improvement with a bypass of the obstructive segment with a nasojejunal tube before the luminal narrowing of the affected bowel illustrated on serial imaging and the characteristic findings on initial CTAP. Previous reviews showed that for children with delayed traumatic injury diagnosis, the delay was not attributable to inadequate initial imaging, suggesting these injuries are difficult to appreciate initially.⁷

In summary, delayed TBol is an important part of the differential diagnosis in children with a recent history of trauma presenting with nausea, vomiting, and fever. A UGI series is an important tool for diagnosis but becomes more specific with the progression of stenosis and may be normal in the initial weeks following the injury. Consultation with a pediatric surgeon is recommended if a stricture is seen or there is minimal clinical progress with conservative management. Alternative diagnoses, like SMA syndrome, should still be considered, but if unresponsive to therapy, these patients would need re-imaging and re-evaluation for a delayed presentation of TBol.

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The authors have nothing to report.

CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest.

ETHICS STATEMENT

The authors thank the patient and her family for their informed consent to publish this case.

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