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Case Report

Small bowel obstruction through an internal hernia caused by a congenital mesenteric defect: an early diagnosis and prompt treatment of a rare condition saved a young life

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ABSTRACT

Congenital internal hernias causing small bowel obstruction are extremely rare in adults, with only approximately 28 cases reported in the literature. The condition carries a high mortality rate-up to 50% even with treatment, and nearly 100% without intervention. Diagnosis is particularly challenging due to the nonspecific nature of symptoms and often inconclusive imaging findings. We present a patient diagnosed with a trans mesenteric hernia resulting from a congenital defect. The patient failed conservative management, which included bowel rest, nasogastric tube placement, and monitoring with KUB X-rays. A diagnostic laparoscopy was performed and subsequently converted to a laparotomy. Intraoperative findings revealed a 6 cm defect in the mesentery with approximately 60 cm of small bowel herniating through, showing signs of early ischemia. Bowel viability was restored following reduction, and the mesenteric defect was closed. Congenital mesenteric defects, though rare, are an important consideration in the differential diagnosis of de novo small bowel obstruction, particularly in adults with a virgin abdomen and no history of prior abdominal surgery. Early recognition and prompt surgical intervention are essential, as timely management can significantly reduce the risk of morbidity and mortality.

Keywords: Small bowel obstruction, Internal hernia, Congenital mesenteric defect, Transmesenteric hernia, Diagnostic laparoscopy, Bowel ischemia

INTRODUCTION

Congenital internal hernias are a rare cause of small bowel obstruction, predominantly described in infants and pediatric populations. In adults, these hernias are exceedingly uncommon, with only 28 documented cases reported to date in the literature.[1] They typically occur near the ileocecal region or the ligament of Treitz in adults. Volvulus or bowel strangulation occurs in 30% to 40% of cases, with mortality rates reported as high as 50% in treated patients and approaching 100% in untreated cases. Autopsy studies reveal that congenital transmesenteric hernias account for 0.2% to 0.9% of all internal hernias and are responsible for intestinal obstruction in 4.1% of cases.[2] Acquired internal hernias are more prevalent than congenital ones, with paraduodenal hernias being the most common congenital type, particularly in males.[3,5] The diagnosis of congenital transmesenteric hernias is challenging due to nonspecific symptoms and signs that can mimic other common causes of acute abdomen, such as appendicitis, gastroenteritis, or cholecystitis. Despite their rarity, transmesenteric hernias represent surgical emergencies requiring prompt diagnosis and intervention to prevent progression to bowel strangulation, necrosis, perforation, peritonitis, sepsis, and death.[6] Authors present a case of a 34-year-old male with abdominal pain diagnosed with an internal hernia secondary to a congenital transmesenteric defect, successfully treated by prompt surgical exploration without the need for bowel resection.

CASE REPORT

A 34-year-old Asian male with no significant past medical history, presented to the emergency department with a 4-hour history of abdominal pain, nausea, vomiting, and chills that began while resting at home after consuming a large dinner. He reported his last bowel movement being after the onset of his symptoms.

Figure 1: Gross view of congested and dusky-appearing small bowel loops immediately following exteriorization, before reduction.

Figure 2: Intraoperative image demonstrating early ischemia of approximately 60 cm of small bowel herniated through a congenital mesenteric defect.

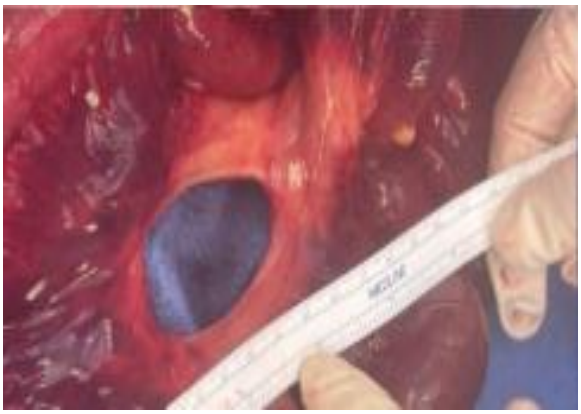

Figure 3: Identification of a 5 cm congenital mesenteric defect in the small bowel mesentery through which the bowel herniated.

Figure 4: Restoration of bowel perfusion following reduction of the herniated segment, and closure of the mesenteric defect.

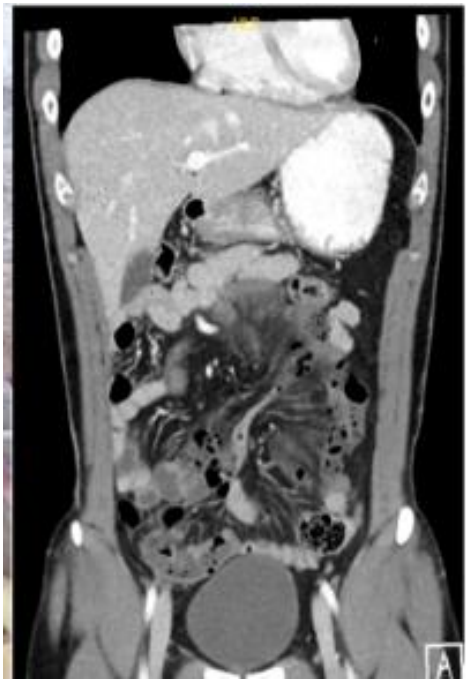

Figure 5: Coronal CT scan showing central mesenteric stranding and swirling mesenteric vessels-classic features suggestive of internal hernia.

On examination, the patient appeared to be in moderate discomfort, but vital signs were stable. Abdominal examination revealed mild, poorly localized tenderness without guarding, rebound tenderness, or distension. Laboratory tests showed leukocytosis of $20.2\times10^3/\mu l$ with a left shift of 90.6%, and an elevated lactate level of 2.9 mmol/l. Computed tomography of the abdomen and pelvis demonstrated central mesenteric stranding with a swirling mesenteric pattern and clustered small bowel loops, suggestive of a possible internal hernia, although there was no significant bowel dilation to confirm an obstruction.

The patient was then started on intravenous fluids, Mefoxin, and pain management. As the patient's pain worsened despite nasogastric tube decompression, subsequent flat and upright radiographs revealed no

progression of contrast beyond the stomach, and the decision was made to proceed with surgery. Five hours later, the patient was taken to the operating room for diagnostic laparoscopy, which was converted to an exploratory laparotomy. A 5 cm mesenteric defect was identified, through which approximately 60 cm of small bowel had herniated. The herniated bowel segment appeared at first significantly congested and dusky but without necrosis.

Gentle traction and blunt manipulation were used to reduce the bowel. After reduction, the herniated segment regained peristalsis and improved in color, indicating viability. The mesenteric defect was closed primarily with interrupted 3-0 silk sutures, carefully preserving mesenteric vascular integrity. A closed-suction drain was placed adjacent to the repair site. The abdominal cavity was irrigated and inspected for hemostasis. The fascia was closed in a standard fashion, and the skin was approximated with staples. The patient had an uneventful postoperative course, with return of bowel function by postoperative day 2. His diet was slowly advanced, and he was discharged on day 7.

DISCUSSION

Internal hernias are defined as the protrusion of a viscus through a normal or abnormal aperture within the peritoneal cavity.[7] Congenital internal hernias are a rare cause of small bowel obstruction, predominantly described in infants and pediatric populations. In adults, these hernias are exceedingly uncommon, with only 28 documented cases reported to date in the literature.[1]

They typically occur near the ileocecal region or the ligament of Treitz in adults. Volvulus or bowel strangulation occurs in 30% to 40% of cases, with mortality rates reported as high as 50% in treated patients and approaching 100% in untreated cases. Autopsy studies reveal that congenital transmesenteric hernias account for 0.2% to 0.9% of all internal hernias and are responsible for intestinal obstruction in 4.1% of cases.[2] Congenital internal hernias are classified based on the location of the defect into paraduodenal, transmesenteric, pericecal, foramen of Winslow, intersigmoid, and paravesical types. Acquired internal hernias usually arise through potential spaces that developed after surgery, trauma, or inflammation.[8]

Congenital transmesenteric hernias result from developmental defects in the mesentery, likely due to incomplete fusion or ischemic injury during embryogenesis.[9] They may be asymptomatic until bowel herniation occurs. The majority are located near the ileocecal region or small bowel mesentery, consistent with the present case. Pediatric cases differ as these hernias may present earlier and sometimes involve the small bowel mesentery more diffusely.[4] Internal hernias often present with nonspecific gastrointestinal symptoms, making early diagnosis difficult. Fan et al reported abdominal pain in 100%, vomiting in 71%, and abdominal distension in 63% of internal hernia cases. Preoperative diagnosis is challenging, achieved in only 16% of cases due to intermittent and vague symptoms.[5] The patient's presentation with mild, poorly localized tenderness, stable vitals, preserved bowel function highlights the subtle clinical picture that can precede catastrophic bowel compromise. High clinical suspicion is critical for early intervention.

Imaging is paramount in diagnosing internal hernias. Initial modalities such as plain abdominal radiographs and ultrasound have limited sensitivity and specificity, often showing nonspecific signs like bowel dilation or air-fluid levels. Ultrasound is limited by operator dependency and poor visualization of deep structures.[2] Upper gastrointestinal series can occasionally demonstrate abnormal bowel positioning but are seldom used acutely due to practical constraints.[3] Computed tomography remains the gold standard, providing high diagnostic accuracy. Key CT findings may be subtle, necessitating experienced radiologist input for timely detection. They include clustered small bowel loops in abnormal locations, mesenteric vessel engorgement and crowding, displacement of adjacent structures, and the whirl sign indicative of mesenteric volvulus.[2,9] Magnetic resonance imaging offers similar anatomical detail but is rarely used emergently.[2]

Prompt surgical intervention is necessary to reduce the herniated bowel, assess viability, resect necrotic segments if needed, and close the mesenteric defect to prevent recurrence. Delayed treatment results in bowel ischemia, necrosis, and perforation, with mortality rates reaching up to 75%.[7] Meticulous closure of the mesenteric defect is essential to avoid suture breakdown or incomplete closure, which may lead to postoperative complications or recurrence. Postoperative care involves close monitoring for anastomotic leaks, obstruction, or sepsis, maintaining fluid-electrolyte balance, early nutritional support, antibiotic prophylaxis, and monitoring of surgical drains.

Early mobilization and pulmonary hygiene reduce respiratory complications. Follow-up imaging may be warranted if clinical signs suggest complications.[7] Recurrence after surgical repair is rare but can occur if the mesenteric defect is incompletely closed or sutures fail.[7] Re-herniation may cause recurrent obstruction or strangulation. Vigilant postoperative surveillance and early investigation of recurrent symptoms are critical. Routine imaging follow-up is not standardized but may be considered based on clinical suspicion.[3] Prognosis depends heavily on early diagnosis and intervention. Patients treated prior to bowel strangulation typically experience excellent outcomes. Conversely, delays result in higher morbidity and mortality, with reported death rates between 20% and 75%, primarily from ischemia, perforation, and sepsis.[1] Prompt surgical reduction and

defect closure remain the cornerstone of favorable prognosis.[3,6]

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