

Masquerading as adhesions: left paraduodenal hernia, a congenital cause of small bowel obstruction

Amy Van der Sluis, Divyansh Panesar, Richard Tapper

Internal hernias are an uncommon cause of small bowel obstruction (SBO), responsible for only 0.2–0.9% of cases. Among these, paraduodenal hernias are the most frequent, representing 53–75% of congenital internal hernias. Left paraduodenal hernia (LPDH), also known as Landzert's hernia, occurs more often than its right-sided counterpart and arises from an embryological anomaly during midgut rotation. Although rare, LPDH is clinically significant due to the potential for strangulation and bowel ischaemia if diagnosis is delayed. The clinical presentation is often vague and intermittent, which may result in misdiagnosis or delayed surgical intervention. With increasing use of computed tomography (CT), pre-operative diagnosis has improved, yet many cases are still recognised intraoperatively. This report describes a young patient with SBO due to LPDH, managed with laparoscopic exploration and conversion to open repair. Contemporary literature is reviewed to highlight diagnostic strategies and evolving operative approaches, demonstrating the importance of early recognition and meticulous surgical technique to prevent bowel compromise.

Case presentation

Mr X, a 22-year-old man, presented with a 2-day history of severe, periumbilical pain that radiated throughout the abdomen. The pain came in waves, progressively worsening over 24 hours. His last bowel motion was the day prior, and he was no longer passing flatus. He was nauseous and had vomited twice. He had no significant medical history and, notably, no previous abdominal surgery. His observations remained within normal range. On examination, he was distended with generalised tenderness but no guarding or peritonism. All blood tests including inflammatory markers were within normal range. He proceeded to have a contrast enhanced CT scan, which showed dilated small bowel loops with a focal transition point in the mid-distal ileum, suggestive

of an SBO and suspicious for a band adhesion (see Figure 1 and Figure 2). Initial management included nasogastric tube placement, intravenous (IV) fluid resuscitation, IV analgesia, mechanical and pharmaceutical venous thromboembolism prophylaxis and gut rest. Over the following hours his clinical condition failed to improve and his pain continued to deteriorate. It was deemed unlikely that he would resolve without operative management, and he made an informed decision to proceed with an operation.

Initial laparoscopy revealed multiple distended proximal small bowel loops emerging from behind the transverse mesocolon, with collapsed distal loops disappearing beneath, suggesting an internal transition point (see Figure 1 and Figure 2). Congenital adhesions between small bowel, sigmoid colon and pelvic side wall were noted, but did not account for the obstruction. Further exploration revealed a segment of jejunum herniating through a defect to the left of the duodenojejunal (DJ) flexure, beneath the transverse mesocolon, consistent with a left paraduodenal hernia. The herniated bowel was gently reduced but the distended viscera began to dominate the operative field, leading to loss of space and visibility for safe laparoscopic manoeuvres. At this point, the procedure was converted to an open midline laparotomy.

Intraoperatively, a large hernia defect was visualised and bounded by the DJ flexure and the left colic artery (see Figures 3–5). The hernia sac persisted into the lesser sac, causing the gastrocolic omentum to billow out. Interestingly, this was fully reducible without the need for further dissection. Redundant sac was then resected with electrocautery and plicated with a 2-0 Vicryl by approximating the peritonealised mesentery to the serosal edge of the DJ flexure and imbricating the sac. The patient recovered uneventfully, with resumption of oral intake on day 5, cessation of IV analgesia on day 2 and discharge on post-operative day 6. High-quality intraoperative

Figure 1: Key image from contrast enhanced computed tomography (CT) scan.



Figure 2: Key image from contrast enhanced computed tomography (CT) scan.

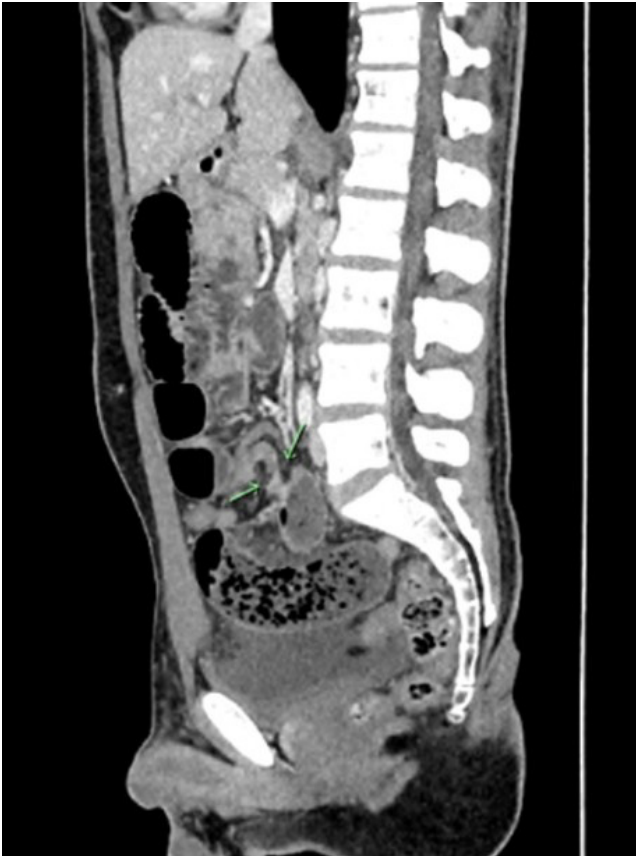


Figure 3: Defect with left colic artery.

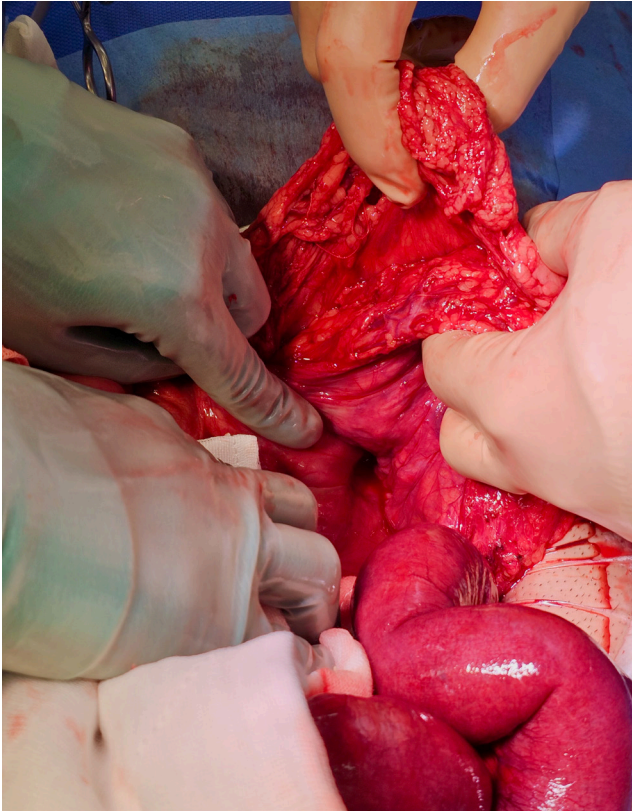


Figure 4: Extent of defect extending up to 10cm.



Figure 5: Thickened peritoneum of hernia sac.



photographs demonstrate the large hernia defect, sac and the relationship to surrounding structures.

Discussion

Congenital internal hernias account for only 0.2–0.9% of all SBO.¹ Paraduodenal hernia (PDH) are the most common congenital internal hernias, responsible for up to 53% of reported cases.² Left-sided PDH (Landzert's hernia) arise from a failure of mesenteric fusion between the descending mesocolon and posterior parietal peritoneum during embryogenesis, resulting in a peritoneal recess to the left of the DJ flexure, allowing herniation of viscera underneath the transverse mesocolon.³

The defect, known as the fossa of Landzert, is bordered anteriorly by the inferior mesenteric vein (IMV) and variably by the left colic artery or its ascending branch, structures that make surgical repair technically challenging.⁴ Patient presentation can be variable, ranging from vague, intermittent symptoms to acute high-grade

obstruction. Congenital hernias should always remain a differential diagnosis of consideration in younger people presenting with symptoms of obstruction.

PDHs most commonly affect males in their fourth to sixth decade of life, but cases in younger patients are also documented.⁵ Adhesions, although possible without prior surgery, are only present in approximately 50% of cases.⁶ CT can aid diagnosis but its diagnostic accuracy is highly variable in patients without prior surgery as evidenced in our case where imaging (highly suggestive of adhesive SBO) was discordant with operative findings.⁷ Typical CT findings include encapsulated jejunal loops in the left upper quadrant, mesenteric vessel crowding or swirl, and abnormal location of the bowel posterior to mesenteric vessels.⁸ However, diagnosis is often made intraoperatively.⁹ Our intraoperative images illustrate the classic anatomical relationships of a left PDH, reinforcing intraoperative recognition and serving as a valuable teaching aid.

Surgery is the definitive treatment. The primary objectives are reduction of the herniated bowel,

assessment of viability, and management of the hernial orifice, either by closure or enlargement.¹⁰ Defect closure is preferred when the vascular anatomy allows safe suturing; however, widening may be chosen when the IMV or left colic artery are at risk.¹¹ Laparoscopic repair is feasible but may require conversion, particularly when distended bowel limits working space.^{10,11} In our case, a laparoscopic approach facilitated diagnosis but the procedure was converted to open to ensure safety. Careful defect closure avoided injury to the left colic artery.

Laparoscopic management has become increasingly reported since the mid-2010s, with contemporary series showing favourable outcomes.¹² However, laparoscopy is technically demanding in the presence of distended small bowel or when vascular landmarks are obscured.¹⁰ Conversion to open surgery should not be viewed as a failure but as an essential safety measure where adequate views or safe operating field cannot be attained.¹³ In our case, conversion allowed safe reduction, clear identification of the IMV and left colic artery, and secure defect closure. This mixed approach is consistent with

current best-practice recommendations.

When diagnosed and treated early, LPDH has an excellent prognosis. Mortality rates have fallen dramatically with advances in imaging and early operative intervention. Recurrence after complete closure is rare, though long-term data remain limited. Delayed diagnosis, however, carries a high risk of strangulation and ischaemia, which is reported in up to half of untreated cases.¹⁴ Elective repair is recommended even for incidentally discovered LPDH, given the risk of catastrophic obstruction.

Overall, LPDH is a rare but important differential in young patients, and those without prior abdominal surgery, presenting with SBO. Definitive management involves reduction and closure of the defect, with laparoscopic repair preferred where feasible. Conversion to open surgery should be performed when anatomy is unclear or bowel is distended. Timely recognition and surgical management are critical to avoid complications. The provided intraoperative photographs offer invaluable educational insight into this anatomical anomaly.

COMPETING INTERESTS

Nil.

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