Gangrenous Small Bowel Obstruction Secondary to Congenital Internal Herniation: A Case Report

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SUMMARY

Congenital internal herniation is a rare condition presenting as recurrent abdominal pain or acute intestinal obstruction. In cases in which bowel incarceration or strangulation develop, rapid progression to bowel ischemia, necrosis or perforation is inevitable. Mortality in such cases has been reported to be as high as 50%. Despite advances in imaging modalities, arriving at a pre-operative diagnosis of a congenital internal herniation remains a challenge. We report such a case where imaging was unsuccessful in determining the cause of intestinal obstruction in a 3 year old child. Congenital internal herniation may result in disastrous consequences if not addressed in a timely fashion due to its rarity. Hence a high index of clinical suspicion is needed to avoid missing this diagnosis in a child presenting with recurrent abdominal pain or acute intestinal obstruction.

KEY WORDS:

Congenital internal hernia; Transmesenteric, Gangrenous bowel; Small bowel obstruction; Children

INTRODUCTION

Hernias are of two main types, external and internal. Internal hernias are defined as protrusion of a viscus through a normal or abnormal peritoneal or mesenteric aperture within the confines of the peritoneal cavity. The orifice can be either acquired or congenital; through normal apertures or abnormal apertures - arising from anomalies of internal rotation and peritoneal attachment. It is a rare cause of small bowel obstruction in children, occuring in all age groups and equal sex distribution. Most cases are diagnosed only intraoperatively due to non-specific symptoms and signs. Plain radiographs, ultrasound and CT scan may be useful, but accurate diagnosis is rarely made. This report presents a child with small bowel gangrene as a result of obstruction cause by a congenital internal hernia.

CASE REPORT

A previously healthy, 3- year- old Malay boy presented with sudden onset of severe abdominal pain to emergency department, lasted for an hour at home associated with intractable projectile, non bilious vomiting and abdominal distension. The child had normal bowel opening prior to this and his mother did not notice any change in stool colour and consistency.

Clinical examination revealed a dehydrated child, but haemodynamically stable. Abdominal examination revealed a scarless, distended abdomen with no localized tenderness, no palpable mass and with sluggish bowel sound. Per rectal examination revealed an empty rectum. Other systemic examination were normal.

His blood counts on admission revealed a hemoglobin level of 14.4 g/dL, hematocrit level of 43.2% elevated total white count 32.8 x10°/L (predominantly leucocytosis) and a normal platelet count and electrolytes level. Both erect and supine abdominal X-rays revealed grossly dilated small bowels and an abdominal ultrasound showed only presence of free fluid, but no features to suggest cause of intestinal obstruction. Thus, the child was kept nil orally on fluid resuscitation, 4-hourly gastric aspiration and commencement of intravenous co-amoxyclav and treated as septic ileus probably secondary to mesenteric adenitis.

Within the next 24 hours, the child deteriorated as his abdominal distension worsened, with increasing amount of gastric aspiration and no bowel opening. Repeat blood count revealed a sudden drop of hemoglobin and haematocrit level to 5.7g/dL and 17.2% respectively and metabolic acidosis. An urgent CT abdomen with oral contrast was performed showing increasing amount of free fluid, grossly dilated and fluid-filled small bowel loops, centrally clustered, abutting on the anterior abdominal wall, with no abnormalities detected on the appearance or location both superior mesenteric artery or inferior mesenteric vein to suggest malrotation, volvulus or features of an internal hernia; thus the cause of mechanical obstruction was not demonstrated. There appeared to be a swollen tubular structure in continuity with the caecum, measuring about 1 cm in length; reported as possibly an inflamed or ruptured appendicitis with intrabdominal collection causing septic ileus.

An urgent explorative laparatomy was performed. In contrary to the CT scan report, we found long segment of gangrenous small bowels, involving distal jejunum and whole of ileum; from approximately 150 cm from Treitz ligament up to 15 cm just before ileocaecal junction, secondary to strangulation caused by a congenital transmesenteric internal herniation. No other bowel abnormalities were found intraoperatively. Resection of gangrenous segment and anastomosis was performed and remaining bowel length was approximately 170 cm. Child was nursed in paediatric intensive care unit for 5 days with full parenteral nutrition support. Subsequent post operative course was uneventful. Short bowel syndrome did not develop in this case.

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Fig. 1: CT abdomen: Intraabdominal free fluid with grossly dilated small bowel; centrally clustered abutting on anterior abdominal wall, fluid-filled with thickened bowel wall. Major vessels (SMA, SMV, IMV) locations were normal.

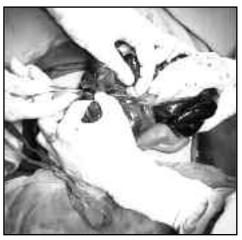


Fig. 2: Intraoperative finding of extensive length of gangrenous small bowel secondary to bowel strangulation. The bowel loops appeared to have herniated through a congenital mesenteric defect. The non-viable bowels were too distended to be reduced to demonstrate the mesenteric defect. Resection and end-to-end anastomosis performed.

DISCUSSION

Overall incidence of internal hernias is 0.2–0.9%¹. Meyers et al have described several types of internal hernia based on locations; most common being paraduodenal (53%) followed by pericecal (13%), foramen of Winslow (8%), transmesenteric and transmesocolic (8%), intersigmoid(6%), retroanastomotic (5%) and paravesical (7%) from historical data.

Transmesenteric hernias in children are due to congenital defect rather than a surgically created mesenteric defect that are often seen in adults. Rokitansky reported the first case of transmesenteric hernia found in autopsy in 1936, in which the caecum had herniated through a defect in the mesentery near the ileocaecal valve. In 1885, Treves described a part of the mesentery near the terminal ileum that was circumscribed by the junction of the ileocolic artery and the last branch of the ileal artery- later known as Treves "bloodless" field. Congenital mesenteric defects usually occur in this thin and avascular mesentery – approximatedly 15 cm from ileocaecal junction, as in this case.

The pathogenesis of mesenteric defects is uncertain. One popular theory relates the cause to prenatal intestinal ischemia and subsequent thinning of the mesenteric leaves because the prenatal intestinal ischemia is associated with bowel atresia in 5.5% of the pediatric population []. Other causes postulated include intraperitoneal inflammation, trauma, partial development regression, and fenestration of the mesentery by the colon during the embryologic displacement into the umbilical cord.

Although a rarity, internal hernia constitute up to 5.8% of all small-bowel obstructions, which, if left untreated, have been reported to have an overall mortality exceeding 50% if strangulation is present². Blachar *et al*⁴ have described several

CT signs common to all types of internal hernias as evidence of small-bowel obstruction by reviewing 17 cases retrospectively. On the other hand, Ghiassi *et al* ⁵ reported that 16% of retrospective reviews of forty-nine CT scans were suspicious for internal hernias, and yet, an accurate preoperative diagnosis using CT scan is rarely made, just like in this case.

In view of this, a high index of clinical suspicion is required in diagnosing congenital internal herniations causing intestinal obstruction. This is especially so, in situations where radiographic imaging were inconclusive and non operative management failed. A high index of suspicion leading to timely operative intervention, while not only justifiable, could have limited the risks of morbidity for the patient in this case.

The treatment is based on the operative findings. If the bowel is viable, simple reduction should be performed. Gangrenous bowel should be resected with an end-to-end anastomosis to restore bowel continuity. The hernial defect should be closed with non-absorbable sutures. A mesenteric defect, regardless of its size, should be closed when encountered by chance.

CONCLUSION

In conclusion, transmesenteric hernias usually rapidly progress to bowel ischaemia once strangulated and have no definitive predictors. Because of the difficulty with diagnosis preoperatively and its association with high mortality in delayed diagnosis, congenital internal herniation should be suspected in children presenting with recurrent abdominal pain or small bowel obstruction without any history of previous abdominal surgery or trauma. Early decision for exploratory laparotomy based on clinical suspicions could prevent lethal complications.

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