Left Paraduodenal Hernia: Case Report and Literature Review

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ABSTRACT
Paraduodenal hernia is the most common form of congenital internal hernia and constitutes a protrusion of bowel into the retroperitoneal space through peritoneal defects near the third and fourth portion of the duodenum. The lifetime risk of obstruction and bowel strangulation is around 50% with a mortality of 20% and higher. Despite the rarity of the disease, it poses a serious surgical problem. The high risk of obstruction and the associated mortality mandate repair once the diagnosis is established. We present the interesting case of a 24-year-old woman with a left paraduodenal hernia who presented with abdominal pain and vomiting and underwent subsequent laparotomy with hernia repair. The purpose of this paper is to review the etiology, pathology, diagnosis and treatment of paraduodenal hernia and to heighten the awareness of this rare but significant disease.

Key words: Paraduodenal hernia, Internal hernia, Intestinal obstruction, Bowel ischemia

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ÖZET
Sol Paraduodenal Herni: Olgu Sunumu ve Literatürün Gözden Geçirilmesi

Anahtar kelimeler: Paraduodenal herni, İnternal herni, Bağır sak tıkanıklığı, Bağır sak iskemisi
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INTRODUCTION
Paraduodenal hernia (PDH) is a protrusion of bowel into the retroperitoneal space through peritoneal defects near the third and fourth portion of the duodenum\(^1\). PDH is the most common form of congenital internal hernia accounting for half of all cases and 1% of all small bowel obstructions. 75% of these hernias are left-sided and due to failed embryological development. The reported lifetime risk of obstruction and bowel strangulation is around 50% with a mortality of 20% and higher\(^2,3\). The diagnosis of PDH is difficult as symptoms are often nonspecific and many clinicians are unfamiliar with this rare condition. We present the interesting case of a young woman with a left paraduodenal hernia and discuss etiology, diagnosis and treatment according to the current literature on PDH.

CASE REPORT
A 24-year-old morbidly obese woman presented to the emergency room with a one-day history of acute onset sharp abdominal pain. She described the pain as diffuse and had not been able to tolerate food for the last day. She had not had a bowel movement in several days and was not passing flatus. She reported several episodes of vomiting prior to admission. The patient stated that she had several similar episodes of abdominal pain in the past, which had been attributed to pelvic adhesions as a result of a previous cesarean section and a ruptured ectopic pregnancy that necessitated exploratory laparotomy. She had further undergone laparoscopy with lysis of pelvic adhesions in the past. Her past medical history was significant for polycystic ovarian syndrome, hypertension and morbid obesity. She was taking ibuprofen and hydromorphine/acetaminophen for recurrent abdominal pain.

On presentation she was afebrile, with normal heart rate and blood pressure. Her abdomen was diffusely tender without rebound tenderness or guarding. She had scarce bowel sounds. Her white blood cell count was 7800/μL. Her chemistry and liver function tests were all within normal limits. She underwent a computerized tomography (CT) scan with oral and intravenous contrast that showed clustered loops of small bowel in the left upper quadrant posterior to the descending colon (Figure 1 A,B). This was consistent with a left paraduodenal hernia. After informed consent was obtained the patient was taken to the operation room for exploratory laparotomy and closure of the paraduodenal hernia. A left subcostal incision was used to gain access to the peritoneal cavity. The omentum was found adherent to the previous c-section incision and was sharply lysed. Omentum and transverse colon were now retracted cephalad and the paraduodenal hernia was instantly visible to the left of the duodeno-jejunal junction (Figure 2). Small bowel that had herniated underneath the inferior mesenteric vein through the paraduodenal fossa into the retroperitoneal space was now easily reduced without enlarging the hernial orifice. The bowel initially appeared dusky and decompressed but was found to be fully viable after several minutes. The paraduodenal hernia was now closed using several sutures.

Figure 1. Computerized tomography scan of the abdomen in axial and coronal cuts demonstrating the left paraduodenal hernia. Arrows are outlining the hernia sac, with clustered, sharply circumscribed loops of small bowel in the left upper abdomen.
interrupted 3-0 silk sutures, attaching the peritoneal rim next to the inferior mesenteric vein to the retroperitoneal tissues to the left of the duodeno-jejunal junction (Figure 3). The abdomen was now closed in several layers and the patient was then extubated and recovered from anesthesia. Postoperatively her pain was controlled with intravenous narcotics and diazepam. On postoperative day (POD) 1 her nasogastric tube was removed and she was started on a clear liquid diet. By POD 2 she was passing flatus, her diet was advanced to regular food and she was discharge home later in the day on oral pain medication. She was readmitted to the hospital on POD 4 with ileus that resolved with bowel rest and intravenous fluid administration. She was discharge home on POD 6 with bowel function present. She was seen in the office one week later with well healing wounds and no further complaints of abdominal pain.

**DISCUSSION**

In 1857 Treitz defined an internal hernia as a retroperitoneal protrusion of an abdominal organ through a peritoneal fold[1]. These hernias can be classified according to their etiology as either congenital or acquired. Post-operative small bowel obstruction (SBO) due to internal hernia in certain patient populations (e.g. after liver transplantation or bariatric surgery) is increasing in incidence and as likely as obstruction from adhesions[2]. The focus of this discussion will be on congenital hernias, specifically paraduodenal hernias, as described in our case presentation. Among the numerous etiologies of SBO internal herniation represents only about 1% of all cases, with paraduodenal hernia (PDH) being the most common entity half of the time. The true incidence of these hernias is difficult to establish as many cases are either completely asymptomatic or misdiagnosed as functional gastrointestinal abnormalities. Only around 50% of paraduodenal hernias for example present with SBO or bowel strangulation[3]. In large autopsy series the prevalence of PDH has been reported between 0.02 and 0.2% however it might be higher as suggested by a recent retrospective review of 294 upper gastrointestinal X-ray series that revealed PDH in 2% of the reviewed cases[4,5]. To date there have been around 500 reports in the literature and although PDH is a rare condition, they pose a significant surgical problem, as the lifetime risk of obstruction/strangulation is 50-66% with a mortality in these cases of 20% and more[6,7]. The abdominal surgeon should therefore be facile with the diagnosis and treatment of PDH.

Meyers classified internal hernias according to their location[2]. Among the congenital hernias (paraduodenal, transmesenteric, pericecal, transmesosigmoid, peri- and supravesical, further hernias through Winslow’s foramen, the mesoappendix, broad ligament, omentum or mesentery of a Meckel’s diverticulum) PDH are most common, representing 50% of the cases. 3/4 of these are left sided and there is a sex
predilection with a male to female ration of 3:1[2,8]. Peritoneal fossae develop between the 5th and 11th gestational week due to an incomplete fusion of the posterior parietal peritoneum and the posterior abdominal wall. Superior and inferior duodenal fossae are most commonly found, in about 1-2% of cases a left paraduodenal fossa lateral to the fourth portion of the duodenum and posterior to the inferior mesenteric artery (IMV) and left colic artery is present (fossa of Landzert)[9,10]. The pathophysiology of hernia formation is not entirely clear. One theory suggests mechanical forces of undulating intra-abdominal pressure to lead to herniation in places where the peritoneum is yet incompletely fused (as in the case of left PDH)[10]. In the case of right PDH (lateral and posterior to the third portion of the duodenum in Waldeyer’s fossa) another mechanism seems to be responsible. Andrews described right PDH as a consequence of malrotation of the prearterial limb of the midgut in embryologic development[11]. In the 5th gestational week the midgut leaves the peritoneal cavity, with the superior mesenteric artery (SMA) defining the long axis of the bowel loop, dividing the midgut into a prearterial and postarterial segment depending on their relationship to the SMA axis. The prearterial limb undergoes a counterclockwise rotation and enters the peritoneal cavity first. After completion of the rotation the prearterial limb and therefore its derivate lie posterior and to the left of the SMA. The post arterial limb now follows and delivers the cecum into a prearterial and postarterial segment depending on their relationship to the SMA axis. The prearterial limb undergoes a counterclockwise rotation and enters the peritoneal cavity first. After completion of the rotation the prearterial limb and therefore its derivate lie posterior and to the left of the SMA. The post arterial limb now follows and delivers the cecum into the right lower quadrant. In the case of right PDH, the prearterial loop does not complete its rotation and gets positioned to the right of the SMA. As the postarterial limb now rotates and the cecum descends, bowel gets trapped behind the ascending mesocolon. In right PDH features of malrotation are often seen on imaging or intraoperatively. In either case, both left and right PDH lack a true hernial sac and therefore should be termed prolapse or procidentia rather than hernia[10]. The diagnosis of PDH is challenging and is almost never achieved clinically. Patients are often asymptomatic or present with recurrent vague and general abdominal symptoms. The pain can be periumbilical or epigastric, colicky or constant[2]. Nausea and vomiting are frequently present, often pronounced postprandially. The symptoms can be dependent on patient position, with aggravation while standing and resolution in a supine position[12,13]. Pain is often intermittent due to herniation and spontaneous reduction and attempts at imaging the hernia during the asymptomatic interval may be unsuccessful[14]. The symptoms are often mislabeled as functional gastrointestinal problems such as irritable bowel syndrome or non-ulcer dyspepsia[15]. Mean age of diagnosis is 29-38.5 years, and most patient report an average of 1.8 years of symptoms[7,10]. SBO with strangulation and ischemia occurs in up to 66% of all patients during their lifetime, with a mortality of at least 20% mainly from abdominal sepsis[7]. The presence of rebound tenderness, leukocytosis > 18.000/mL or bandemia > 6% have been associated with bowel ischemia in cases of both congenital and acquired internal hernias (intermittent abdominal pain is negatively correlated with ischemia)[16]. This serves more as an academic discriminator however, as it is generally agreed upon that any congenital internal hernia such as PDH should be repaired once diagnosed, given the high risk of obstruction with associated mortality. Improvements in imaging technology have increased the preoperative diagnosis of PDH. Barium studies, CT and magnetic resonance imaging (MRI) have been used to diagnose internal hernias, the yield is highest when the patient is imaged during a symptomatic episode[5,17]. Features, which are similar in all three modalities, are the presence of clustered and well- circumscribed loops of small bowel in an abnormal location. In the case of left PDH, bowel loops are found interposed between the descending colon and the adrenal gland, they can displace the stomach, pancreas and duodeno-jejunal junction. The IMV runs along the anteromedial border of the hernia and is laterally and superiorly displaced[17]. CT imaging shows changes in the mesenteric vasculature such as engorgement, twisting, stretching or crowding. These vascular findings are the key to diagnosis as the hernial defect itself is not visible as both hernial orifice and herniating contents are fat attenuating. In right PDH the small bowel is clustered behind the ascending and proximal half of the transverse mesocolon. The SMA and ileocolic artery run along the anterior border of the hernial neck and both bowel loops and mesenteric vessels can loop around the SMA. Evidence of malrotation such as abnormally positioned cecum or lack of a horizontal duodenum can be present[2]. Bowel wall thickening might be an indicator of ischemia. Traditionally PDH have been repaired and often diagnosed by laparotomy[5,7,18]. The surgical principles are reduction and closure of the hernial defect without mesh, or widening of the hernial neck if the contents are not reducible. With left PDH, bowel can often be easily reduced and the peritoneum of the descending mesentry adjacent to
the IMV can be easily approximated with the posteri-
or abdominal wall lateral to the fourth portion of the
duodenum. Most authors advocate to spare the IMV
but instances haven been reported in the literature
where the IMV was sacrificed without ill-effect[19].
Right PDH often poses more of a problem, as the her-
nial contents can be fixed to the retroperitoneum. In
principle the operation tries to recreate the embryolo-
gical situation prior to rotation (or better malrotation)
of the prearterial midgut. Therefore the lateral attach-
ments of the ascending colon in the paracolic gutter
are hernia should not be approached from medially, as
the SMA, ileocolic artery and right colic vein are in
danger of being injured[18]. With the growing laparos-
copic experience of many surgeons minimally invasive
diagnosis and treatment of PDH has become a feasib-
le and safe option[12,20]. Since in PDH a very proximal
obstruction without extensive bowel distension is
present, there is usually sufficient working space for
laparoscopy. To date 15 cases of laparoscopic PDH
repair have been reported in 11 publications[9]. Of
these 73% were left sided and in 77% of cases the her-
nia was primarily closed. In the remainder of the cases
the hernial orifice was widened. Both morbidity and
recurrence were cited at 6.7%. There is no consensus
about how to close the hernial defect, both continuous
and interrupted suturing techniques seem acceptable.
All patients had resumed oral diet by post-operative
day three and the mean duration of hospitalization
was four days[9]. Most authors consider apparent
bowel ischemia or gangrene a contraindication to
laparoscopy or an indication to convert to an open
procedure[3,4,9]. In conclusion, PDH is a difficult diag-
nosis that requires a high index of suspicion from the
surgeon and appropriate imaging. Our case report
demonstrates a typical history of a patient who is labe-
lled with chronic abdominal pain and even undergoes
multiple surgical procedures for a presumed etiology
without any relief of her symptoms. Once the correct
diagnosis is made surgery is the preferred treatment
with an excellent prognosis, eliminating the morbidity
and mortality of small bowel obstruction due to PDH.

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