ABSTRACT

An 83-year-old lady with a genital prolapse had undergone elective transvaginal hysterectomy with pelvic floor repair uneventfully. She developed features of intestinal obstruction after initial recovery. Plain abdominal radiograph and ultrasound suggested low small bowel obstruction which was confirmed by CT scan and gastrograffin follow through study. She underwent exploratory laparotomy at which a congenital mesenteric defect was found in the ileocecal region with internal herniation of the terminal ileal loops through it. The defect was enlarged and hernial contents were reduced. The constriction rings at entry and exit sites were over sewn and bowel was viable without any need for resection. Paracecal internal hernia with a congenital defect in the mesentery presenting in the post-operative period is rare, interesting and challenging. Preoperative diagnosis is usually difficult. Gastrointestinal studies with contrast media and abdominal CT are helpful. Early diagnosis and prompt intervention and appropriate treatment reduce morbidity and mortality.

INTRODUCTION

Congenital lesions leading to intestinal obstructions secondary to internal herniation are generally seen in neonatal and pediatric age group and its presentation in the post-operative period has been reported earlier but mainly in the paraduodenal or paramesocolic region rather than paracecal region [1-2]. Internal hernias are rare causes of small bowel obstruction and diagnosis can be easily missed due to vague symptoms. Paracecal hernia is one of the internal hernias which accounts for 13% of all internal hernias. Symptoms are usually nonspecific and old female patients are particularly vulnerable. Computed tomography plays an important role in early diagnosis. We report a case of small bowel obstruction due to paracecal hernia after vaginal surgery.

Case report

An 83-year-old female patient suffering from a genital uterovaginal prolapse and with a background of hypertension and systemic lupus erythematosis (SLE) was admitted electively. She never had any abdominal surgery previously.

She underwent elective transvaginal hysterectomy with pelvic floor repair and sacrospinous fixation of vaginal vault uneventfully. She opened bowels on second post-operative day. However, she suddenly developed colicky abdominal pain, pallor, increasing vomiting despite antiemetic injections. Vomiting turned bilious followed by feculent associated with abdominal distension and absolute constipation.

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Abdomen was markedly distended but non tender and bowel sounds were high pitched and exacerbated. Urine dipstick, microscopy and culture were normal. Her electrolytes, renal and liver functions were within normal limits. Complete blood count showed haemoglobin 128 G/L, white cell count 21.3 X109/L, neutrophils 18.3 X109/L and C-reactive protein 94 mg/L.

Upright chest radiograph showed normal lungs and heart with a dilated loop of bowel in the upper abdomen (Figure 1). Plain abdominal radiograph showed multiple small bowel loops with vene conniventes and step ladder pattern suggesting a mechanical cause of bowel obstruction rather than a paralytic ileus or functional obstruction due to electrolyte imbalance and hypokalemia (Figure 2).

Abdominal ultrasound demonstrated dilated fluid filled bowel loops with strong peristalsis waves down to terminal ileum which was collapsed. Abdominal CT scan with 100 ml of Omnipaque 350 showed diffuse dilatation of small bowel loops down to terminal ileum which was collapsed and possibility of Meckel’s diverticulum, congenital band or internal hernia was considered (Figure 4,5).

Passage of nasogastric tube drained 1.5 litre of bilious and feculent fluid. Gastrograffin follow through study via nasogastric tube showed dilated loops of small bowel with contrast and faint contrast has reached the large bowel, features were suggestive of partial small bowel obstruction with luminal integrity.

Clinically delayed ileus due to electrolyte imbalance or infection and a remote possibility of post-operative intussusception were considered. After initial partial septic screen and imaging; low mechanical small bowel obstruction with differential diagnosis of Meckel’s diverticulum, congenital band or internal hernia was considered.

Examination under anesthesia revealed a soft 6 cm size mass. She underwent exploratory laparotomy at which a congenital mesenteric defect of 10 cm diameter was found in the ileocecal region with internal herniation of the terminal ileal loops through the mesenteric defect causing mechanical obstruction. The herniated bowel loops were dusky and cyanosed and the defect was compressing the vascular pedicle of the herniated small bowel. The defect was enlarged and hernial contents were reduced. The constriction rings at entry and exit sites were over sewn and bowel was viable and there was no need for resection.

The post-operative period was uneventful; she made smooth recovery and discharged home on 5th post-operative day. At 3 year follow up she is asymptomatic and well.
Figure 3. CT Scan of abdomen showing dilated small bowel loops in the upper abdomen

Figure 4. CT Scan of pelvis- note the collapsed small bowel loops with trickle of contrast and no gas in the rectum and large bowel
DISCUSSION

Small bowel obstruction is one of the leading causes of acute surgical admission. Early diagnosis and timely intervention is important as delayed diagnosis can lead to fatal consequence of strangulation which is associated with tenfold increase in mortality. Hernias are the most common causes of strangulation in patients presenting with small bowel obstruction [1]. Internal hernias are the result of alteration in the normal process of intestinal rotation during embryological development and are the infrequent causes of small bowel obstruction [2]. Although internal hernias account for less than 1% of all abdominal hernias, they are responsible for up to 5.8% of all small bowel obstructions [3]. We here report a case of small bowel obstruction due to paracecal hernia. Paracecal hernia is one of the internal hernias that accounts for 13% of all internal hernias [4]. Internal hernias involve protrusion of viscer a through the peritoneum or mesentery and into a compartment in the abdominal cavity [5]. The most common presentation is an acute intestinal obstruction of small bowel loops that develops through normal or abnormal apertures [6,7]. The responsible hernial orifices are usually pre-existing anatomical structures such as foramina, recesses or fossae. Pathological defects of the mesentery and visceral peritoneum, which are caused by the congenital mechanisms, surgery, trauma, inflammation, and circulation, are also potential hernial orifices [8].

In review of the embryological development of paracecal hernia, it is important to understand the rotation of midgut as those hernias are as a result of alteration of normal embryological development. The development of the cecum started from small budding and then subsequently exteriorised through anterior abdominal wall into umbilicus. The anatomy of the cecal and pericecal peritoneum is not determined until the 5th fetal month when the migration of the midgut is complete. After 5th month of fetal development, migrated midgut subsequently retracted into abdominal wall and cecum was fixed into right iliac fossa and peritoneal surfaces resolved. Alteration in this normal process of intestinal rotation predisposes to development of number of recesses or pockets [9]. Cecum is entirely retroperitoneal and cecal fossa is formed by two peritoneal folds: lateral fold is continuation of the white line of Toldt and medial fold is formed by peritoneal reflection of medial part of cecum. Superior and inferior ileocecal recesses are formed by a peritoneal fold originating from terminal ileum to cecum. Paracecal hernia commonly develop from a retrocecal recess which is formed by cecum anteriorly, the iliac fossa posteriorly, the colic gutter laterally and mesentery medially [10]. In our case, distal ileum was herniated through 10 cm mesocolic defect and symptoms were vague due to a large defect. Preoperative diagnosis of internal hernias is usually difficult due to rarity of the condition, slow development of the symptoms which may take up to months or years depending on the size and type of defects.

It may suggest that internal hernias get spontaneously reduced [11]. However; challenging diagnosis of internal hernias may lead into devastating consequences such as strangulation which is associated with high mortality (Ihedioha et al. 2006a). Paracecal hernia is rarest type of internal hernias and diagnosis can be easily missed. Presenting symptoms are epigastric discomfort, periumbilical pain, recurrent episodes of intestinal obstruction associated with some abnormal findings in the right lower abdomen, such as spontaneous pain, tenderness, or palpation of a mass lesion [12,13]. Ileal entrapment with incarcerated paracecal hernia can mimic acute appendicitis and high index of suspicion should be born in mind in the management of patients with localized peritonism and mass in right iliac fossa even in the absence of obstructive symptoms and presence of normal inflammatory markers. Paracecal hernias are usually associated with old female preponderance [11]. Computed tomography usually plays an important role in diagnosis of early or partial obstruction of all internal hernias. CT has superiority in revealing site, level and cause of obstruction and demonstrating threatening sign of bowel vitality. In addition, CT is also useful in revealing both intrinsic and extrinsic causes of small bowel obstruction such as masses, adenopathy, soft tissue infiltration, fluid collection and abscesses [14]. Combination of gastrointestinal studies enhanced with intraluminal contrast material presenting with postoperative intestinal obstruction and abdominal CT enables the accurate diagnosis of any type of internal hernia [15,16]. (Blachar, Federle 2002, Blachar et al. 2001) In our case, diagnosis was made by gastrograftin follow through study. The two characteristic features of CT findings suggestive of strangulation are sac like masses or clusters of dilated bowel loops and changes of mesenteric vascular pedicle which is engorged, stretched and displaced. These warning signs warrant immediate surgical intervention [17].

Management of small bowel obstruction is laparotomy. However, evidence indicates that small bowel obstruction due to internal hernias can be successfully managed laparoscopically. In low risk patients, laparoscopy offers combined advantages of immediate diagnosis and definitive treatment of acute pathology [18-20]. Internal hernias are rare causes of intestinal obstruction. Transmesenteric hernia is commonest type and usually associated with prior abdominal surgery. Paracecal hernia is rarest type of internal hernia and may or may not be preceded by prior surgery. (Blachar et al. 2001, Sciaccia et al. 1997) Late diagnosis is associated with high morbidity and mortality. Knowledge of normal anatomy of the peritoneal cavity and characteristic anatomical location of each internal hernia helps to get early diagnosis and timely intervention. Computed tomography plays an important role in early diagnosis, identification of intrinsic and extrinsic causes and demonstration of threatening sign of bowel vitality. Most of the small bowel obstructions due
to paracecal hernias are managed by laparotomy without need for further bowel surgery. In low risk patients with early diagnosis, laparoscopic release of obstruction can lead to satisfactory outcome.

CONCLUSION
Paracecal internal hernia with a congenital defect in the mesentery is the rarest form of lesion in an octogenarian lady who had no previous abdominal surgery. The occurrence in the post-operative period of transvaginal surgery makes it more challenging cause of small bowel obstruction. Preoperative diagnosis is usually difficult due to vague symptoms and rarity of the condition. Gastrointestinal studies with contrast media and abdominal CT can diagnose most of the internal hernias with high accuracy. Early diagnosis and prompt intervention and appropriate treatment reduce morbidity and mortality.

REFERENCES