

Internal Hernia through a Defect in the Broad Ligament

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Case Report

ABSTRACT

Intestinal obstruction is a common emergency in the daily surgical practice. Internal hernias causing intestinal obstruction are rare and internal hernias through broad ligament are extremely rare. Symptoms may be colicky abdominal pain, nausea, vomiting and absolute constipation. CT can help in diagnosis although most of the time diagnosis comes on laparotomy performed for intestinal obstruction of obscure origin. Treatment consists of reduction of the herniating intestine and repair of the defect in broad ligament. We present a case of 49 years old female without any prior surgical history, delivery trauma or pelvic pathology who presented by clinical picture of intestinal obstruction. Internal herniation through a defect in the right broad ligament was found.

Keywords: Intestinal obstruction, internal hernia, Broad ligament, constipation

INTRODUCTION

Internal hernia is a rare cause of intestinal obstruction, accounting for 1% of all intestinal obstruction [1]. An internal hernia means herniation of hollow viscus usually the small intestine through an opening within the peritoneal cavity. It may be acquired or congenital and persistent or intermittent [2]. Hernia through defects of broad ligament is even rarer, accounting for only 4% to 5% of all internal hernias [3]. We present a case of intestinal obstruction in a female patient 49 years old. Plain X-ray and CT scan revealed small bowel obstruction. On diagnostic laparoscopy, internal herniation through the right broad ligament was found.

CASE REPORT

49 year-old female patient suffered from general abdominal colicky pain, nausea and vomiting for 2 days and failure to pass motions for 3 days. Patient had regular menstrual cycles. There was no prior history of pelvic or abdominal surgery. She was on antihypertensive medications. General examination was unremarkable. Locally: there was abdominal distension and no scars. Abdomen was soft with tenderness at the right iliac fossa with no guarding. Bowel sounds were exaggerated. on PR examination, rectum was empty with no swellings, no blood, no mucus, no faecal impaction. Blood investigations revealed WBC=10.86 $10^3/\mu\text{L}$ with neutrophilia (89.95%). Serum potassium was 3.31. The other biochemical blood investigations were within normal values.

Plain X-ray abdomen showed multiple air-fluid levels in the erect view and dilated ileal loops. Supine view showed distended small bowel loop in the middle abdomen (Figure 1). Abdominal ultrasound showed multiple dilated bowel loops. The bowel loops were seen filled with debris which showed peristaltic antiperistaltic movements. Mild fluid was noted in the pelvis with a rim of fluid seen in Morrison's pouch. Abdominal computed tomography (CT), (Figure 2) showed: dilated small bowel loops all-over the abdomen and pelvis with the maximum thickness reaching about 4 cm. The abrupt change in the calibre "the transition zone" was seen in the right iliac fossa with prominent stretched mesenteric vessels noted there. Faecal matter and gases were seen in the large bowel, but it was not distended properly. Mild free fluid was seen in the perihepatic, hepatorenal recess and in the pelvis (Figure 2). Good hydration of the patient and insertion of a Ryles tube were done. Emergency exploratory laparoscopy was performed. It revealed an internal herniation of a loop of ileum about 20 cm length through a 3x4 cm defect of the right broad ligament from posterior to the anterior direction

(Figure 3). The defect in the right broad ligament was immediately below the round ligament of the uterus. The herniating loop of ileum was massively congested and expected to be friable. Other side was examined and was found normal. At first, we attempted to reduce the herniating loop laparoscopically but found hazardous. So, decision was taken towards open conversion. On laparotomy the congested herniating loop of ileum was reduced and was found little cyanosed. By application of warm saline and increasing O₂ by anaesthetist, colour came to normal. Repair of the defect was performed using 2/0 polypropylene suture. Patient passed uneventful postoperative course till discharge on the fourth day after surgery.

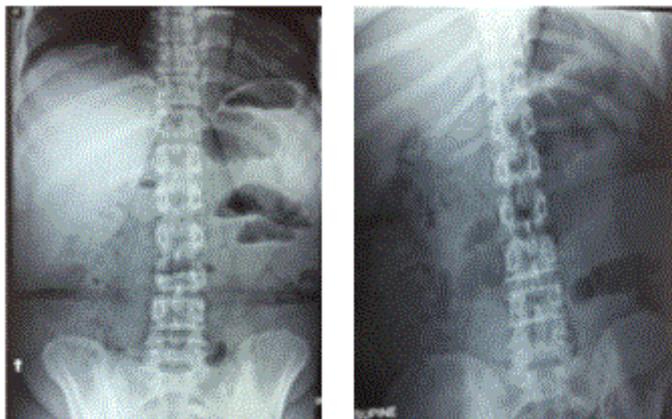


Figure 1. Plain X-ray abdomen erect and supine respectively showing multiple air- fluid levels and dilated ileal loops which is abrupt at right iliac fossa.

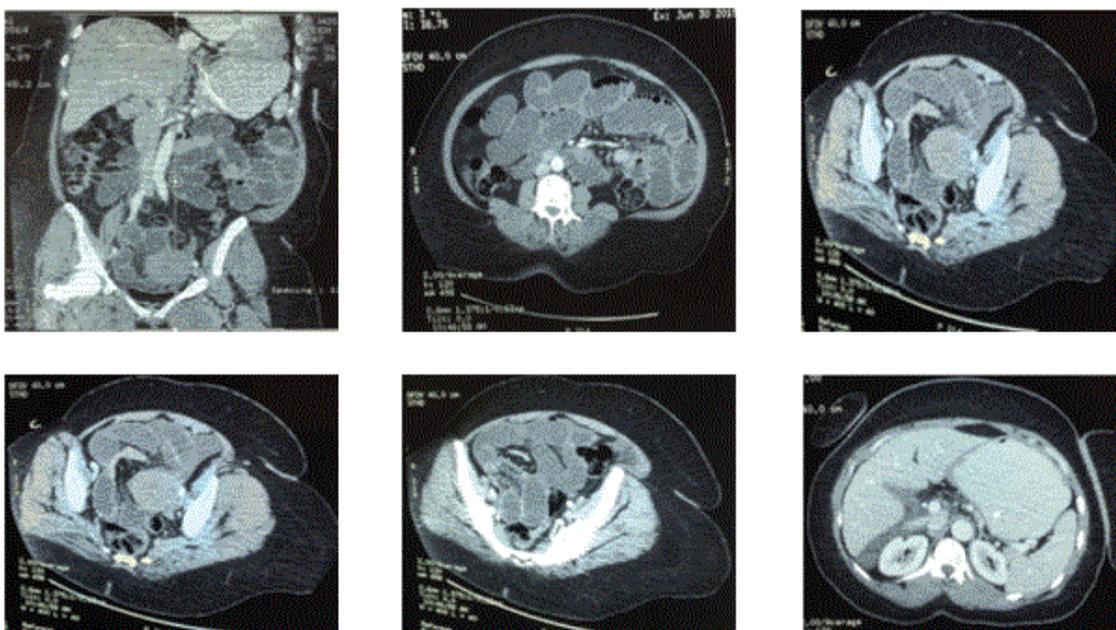


Figure 2. CT pictures showing dilated small intestinal loops all over the abdomen. The abrupt change in calibre “transition point” in the right iliac fossa with prominent stretched mesenteric vessels. Mild free fluid is seen in the perihepatic and hepatorenal recess.

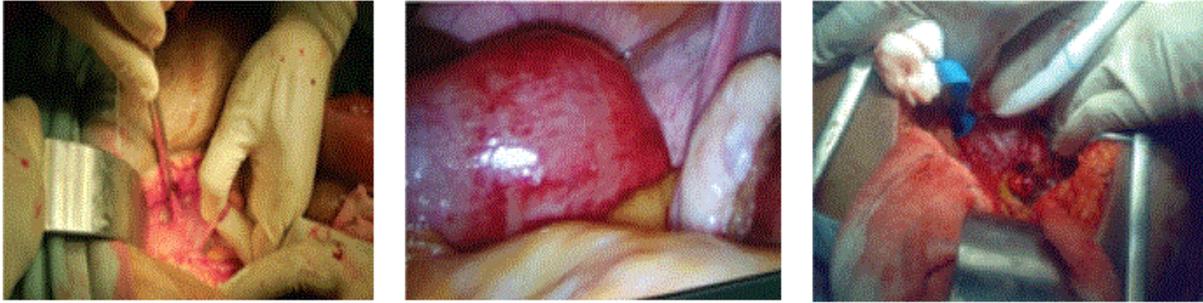


Figure 3. Showing the defect in the right broad ligament, congested entrapped ileal loop after release and picture during final reduction of the entrapped loop.

DISCUSSION

Internal hernia is a rare cause of small bowel loop obstruction and accounts for about 1% of all intestinal obstruction [1]. The various types of internal hernias include paraduodenal hernias (53%), pericecal hernias (13%), hernias through the foramen of Winslow (8%), sigmoid related hernias (6%) and transmesenteric hernias (8%) [4]. Herniation of structures through a defect in the broad ligament is even more uncommon and accounts for only 4-5% of all internal hernias [5]. The ileum is most commonly involved. Herniation of colon, ovary and ureter has also been described [6]. Three types of hernia of the broad ligament have been classified by Hunt : the fenestra type that involves a complete fenestration through a defect in the broad ligament (presence of defect in the two peritoneal layers), the pouch type that involves hernia into the pouch from an anterior or posterior aperture (presence of defect in only one of the two layers, so, the herniating visceral structure would be trapped in a pouch , and finally the heria sac type where the herniating visceral structure is lined by a double layer of attenuated peritoneum , forming a true internal hernia [7]. The defect in our case was of the fenestra type. Cilley et al in 1986 classified broad ligament defects anatomically into three types. Type I, where the defect is caudal to the round ligament. Type II, where defect is above the broad ligament. Type III, where defect is between the round ligament and remainder of the broad ligament [8]. Our case was of the type. The aetiologies may be congenital (primary) or acquired (secondary) [9]. Congenital defects are the result of a developmental abnormality of the broad ligament or from the rupture of congenital cystic structures thought to be remnants of the mesonephric or Mullerian ducts [10]. Secondary or acquired defects may be due to previous surgery, perforation following vaginal manipulation, trauma (including birth trauma) and pelvic inflammatory disease [9]. Our case was a primary or congenital type. Low body mass index may be a contributory factor because it may lead to a very thin mesoovarium and mesosalpinx and cause the patient more prone to a rupture of the broad ligament, with resultant bowel herniation through this defect [11]. Majority of the cases reported in the English literature are unilateral and secondary in nature (22 out of 28 cases). It is rare to have a unilateral defect that is primary in nature [12]. There were three cases of unilateral primary broad ligament defect in English literature, first by Hiraiwa et al., subsequently, the second case by Ngabou et al. and lastly the third case by Sue et al. [12]. The defect in our case was of the fenestra type and the included ileal loop was congested partial impairment of blood supply. After reduction, blood supply improved and loop looked normal. The direction through the defect of the broad ligament may be from anterior to posterior direction [13] or may be from posterior to anterior [14] (as in our case). (CT) showed: dilated small bowel loops seen all-over the the abdomen and pelvis with the maximum thickness reaching about 4 cm. The abrupt change in the calibre “the transition zone” was seen in the right iliac fossa with prominent stretched mesenteric vessels noted there. Hernia through a defect of the broad ligament may cause closed-loop obstruction (incarceration) or even strangulation (ischemia) [6,15].

CONCLUSION

Internal hernia through a defect of broad ligament is a very rare form of all internal hernias that may lead to severe complications. We emphasize that intestinal herniation through through a defect of the broad ligament should be considered one of the causes of internal herniation causing intestinal obstruction. It may be congenital or acquired. It may present by general abdominal colicky pain, nausea, vomiting and absolute constipation. Preoperative recognition and specific CT characteristics of this rare form of internal hernia prompt emergent operation and reduce the mortality and morbidity of the patient. Early surgical treatment is mandatory and is emphasised in these cases. It can be carried out through open or laparoscopic approaches.

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