Open Journal of
Clinical & Medical
Case Reports

Volume 4 (2018)
Issue 24

ISSN 2379-1039

Peritoneal encapsulation: One of several rare etiologies of small bowel obstruction in a virgin abdomen and a discussion of the differential diagnosis

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Abstract

A 50-year-old woman, with no history of abdominal operations, presented to the ER with severe right upper quadrant pain, nausea, and acute constipation. On admission, a CT scan demonstrated possible intestinal obstruction and she was treated conservatively. The patient did not improve with conservative management and a small bowel follow through study demonstrated limited contrast progression and dilation of the small bowel. The patient was taken to the operating room for a diagnostic laparoscopy converted to a laparotomy. Intra-operatively, a peritoneal pocket was identified which encapsulated a portion of the small intestine. The peritoneal encapsulation was lysed and intestines reduced and the patient had an uneventful recovery.

Keywords

small bowel obstruction; peritoneal encapsulation; virgin abdomen; internal hernia

Abbreviations

SBO: Small bowel obstruction; CT: Computed tomography; IH: Internal hernia; RUQ: Right upper quadrant; GERD: Gastroesophageal reflux disease

Introduction & Background

Small bowel obstruction (SBO) accounts for up to 15% of all surgical admissions. However, suspicion for the diagnosis of SBO is greatly diminished when the patient is said to have a 'virgin abdomen' (i.e. no history of abdominal procedures) and no external hernia. Key symptoms of intestinal obstruction include, severe colicky abdominal pain of sudden onset, vomiting, abdominal distension, and absolute constipation [1,2]. Causes of SBO in a virgin abdomen include: congenital and acquired adhesions, internal hernias (IH), neoplasms, gallstone ileus, and other rare conditions [1-3]. Awareness of possible etiologies is important for both diagnosis and management. This case demonstrates a rareetiology of SBO, peritoneal encapsulation, and reviews other SBO etiologies within a virgin abdomen.

Case Presentation

A 50-year-old, post-menopausal female, with a history of GERD and intermittent constipation, presented with a one-day history of increasingly severe, RUQ pain that radiated to the right flank. The

patient was nauseated and reported one episode of non-bloody emesis. Though she continued to pass flat us, her last bowel movement was 2 days prior. She denied any previous similar episodes, abdominal surgeries, or recent trauma. She had not experienced fevers/chills, chest pain, or urinary symptoms. Examination revealed a mildly distressed, a febrile, adult. Abdomen was soft and tender to palpation diffusely in the bilateral upper quadrants. No rebound tenderness or abdominal distention was appreciated.

Complete metabolic panel and complete blood count were unremarkable, urinalysis demonstrated no evidence of a urinary tract infection or hematuria, and beta-hCG was negative. Abdominal ultrasound, to investigate the RUQ pain, only demonstrated mild hepatic steatosis. A non-contrast CT abdomen, obtained for concern of nephrolithiasis, was significant for numerous dilated loops of small bowel (figure 1). There was no evidence of nephrolithiasis or additional pathology. Secondary to the inconclusive laboratory and imaging studies, it was believed that the most likely diagnosis was viral enteritis and the patient was admitted for observation. She was made NPO, a nasogastric tube placed, IV fluids started, and symptomatic management of pain and nausea continued.

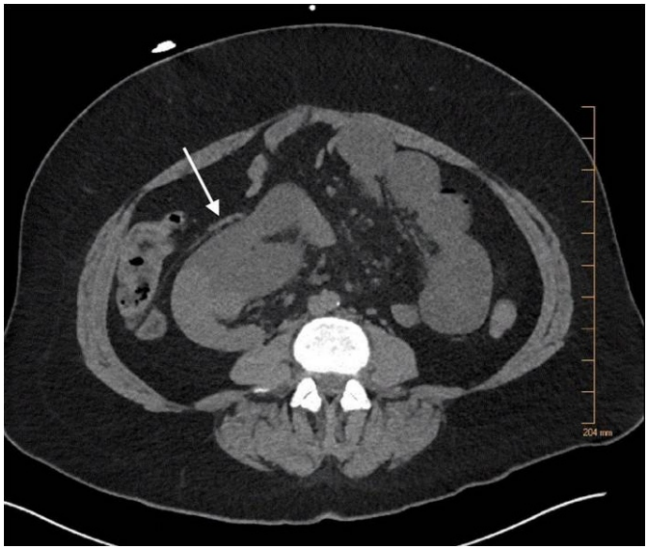

Figure 1: Non-contrast axial abdominal CT image. Demonstrating dilated loops of small bowel in the mid-abdomen, potential transition point, and peritoneal encapsulation (white arrow).

The following morning, a small bowel follow through study was obtained as the patient continued to endorse severe abdominal pain and was no longer passing flatus. Initial progression of contrast through the small bowel was considered adequate. However, after 6 hours and 45 minutes it demonstrated moderate dilation of the jejunum and limited oral contrast progression through the small bowel (figure 2A-D). It was apparent that the patient's condition progressed overnight and at that time a diagnosis of SBO of unknown cause was made. Given the patient's virgin abdomen, she was taken for an immediate diagnostic laparoscopy despite reassuring vital signs and laboratory data.

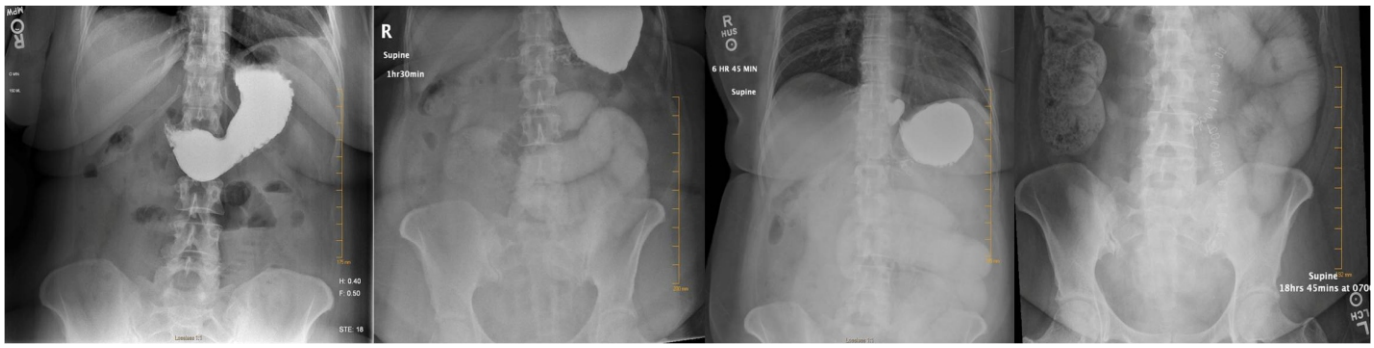

Figure 2: Small bowel follow through study at 0 minutes (A), 90 minutes (B), 6 hours and 45 minutes (C), and post-operatively at 18 hours and 45 minutes (D) after ingestion of water soluble contrast. Demonstrating adequate initial progression of contrast through the small bowel, followed by limited progression of contrast through the small bowel at 6 hours and 45 minutes, consistent with SBO. Finally, post-operative image demonstrates progression of contrast through the small bowel into the ascending colon.

Upon gaining access to the peritoneal cavity, the bowel appeared dilated and hyperemic with engorged lymphatics. A wide, flat peritoneal fold was identified and the procedure was converted to an open laparotomy. As the small bowel was run, an a vascular peritoneal fold was identified. The peritoneal fold was anchored along the cecum, ascending colon, and part of the transverse colon with an opening along the midline near the ileocecal junction, creating a pocket-like structure. Through the opening, the distal jejunum to proximal ileum herniated causing a closed loop obstruction. Doppler ultrasound confirmed the pocket was avascular. The absence of adhesions allowed for easy reduction of the small bowel. The band was lysed and there was no evidence of bowel ischemia or perforation. The small bowel was again run from the ligament of Treitz to the terminal ileum and careful inspection of abdominal anatomy revealed no additional abnormalities.

The operating physician originally believed the obstruction to stem from a hypertrophic ileocecal fold of Treves. However upon review, findings were consistent with partial peritoneal encapsulation. The patient was hospitalized through post-operative day 5 until pain was controlled and post-operative ileus had resolved. Two weeks later, the patient was doing well and had regained normal bowel function.

Discussion

Peritoneal encapsulation is a congenital malformation, described as a thin peritoneal membrane encasing some or all of the small bowel, from the ligament of Treitz to the ileocecal junction. This peritoneal membrane is attached superiorly to the transverse colon, laterally to the ascending and descending colon, and inferiorly to the parietal peritoneum. There are two potential defects within the encapsulation, at the ligament of Treitz and the ileocecal junction [4,5].During the 12th week of embryogenesis, as the herniated midgut returns to the abdominal cavity anomalous association with the yolk sac, results in peritoneal encapsulation [6]. Specific physical exam findings include: inconsistency of abdominal wall and asymmetric/fixed abdominal distension. Imaging is often normal or demonstrates non-specific findings of SBO making pre-operative diagnosis difficult [4,5,6]. Intra-operatively congenital peritoneal encapsulation is described as a thin, non-adherent peritoneal layer covering the small bowel. The bowel loops within the encapsulation are not adherent to each other or the capsule [5,6].

Two similar conditions are often confused with congenital peritoneal encapsulation: Abdominal cocoon syndrome and sclerosing encapsulating peritonitis. Abdominal cocoon syndrome is a post-inflammatory reaction with higher incidence in tropical countries. It is characterized as a fibrocollagenous membrane, with intra-capsule adhesions, encasing the intestines plus or minus solid organs [4-6]. Sclerosing peritoneal encapsulation is characterized by a thick fibrocollagenous membrane and intra-capsule adhesions without solid organ involvement. It is more common than abdominal cocoon syndrome and associated with peritoneal dialysis, practolol (a selective beta-1 adrenergic blocker, which has since been removed from the market due to serious side effects), ventriculoperitoneal shunts, systemic erythematous lupus, and various infections [4-6]. The bloodless fold of Treves, known as the inferior ileocecal fold, is a peritoneal fold that runs from the terminal ileum to the base of the appendix forming the anterior boundary of the inferior ileocecal fossa [7-9]. The ileocecal fold of Treves is by far the rarest cause of SBO, discussed here with only two previously reported cases. In the first case infarction of the inferior ileocecal fold lead to inflammation and necrosis, presenting similar to appendicitis [8]. The second case presented with SBO resulting from a hypertrophic fold of Treves which became pedunculated and self-adherent, forming an obstructive band around the ileum [9].

Intra-abdominal adhesions are highly vascularized collagen bands that connect intra-abdominal structures and are classified as congenital or acquired. At autopsy, adhesions have been found in 3.3% - 28% of virgin abdomens and account for 35% to 75% of SBO in virgin abdomens [1-3, 10-12]. Congenital adhesions vary in appearance to post-operative adhesions, being described as smooth, thin, highly vascularized fibrous tissue, occurring most frequently near the ascending colon and terminal ileum [10, 12-13].

Internal hernias account for under 6% of all SBO cases and are associated with greater than 20% mortality [15,16]. Congenital IH arise secondary to malrotation of the mid gut during the embryonic period [15,16]. Paraduodenal hernia are the most common IH subtype, accounting for more than half of congenital IH. They are sub-classified as either right or left, with left-sided occurring three times as frequently [15,17]. Left-sided hernias occur when the small bowel herniates through Landzert's fossa at the duo denojejunal flexure, the mouth of hernia pocket opens towards the right and contains the inferior mesenteric vein and left colic artery. CT imaging may demonstrate small bowel within a hernia sac around the ligament of Treitz and mesenteric vessel anomalies [7,17].Right-sided hernias occur when the leading edge of the hernia pocket opens to the left and extends from the fossa of Waldeyer to the right retroperitoneal space, resulting in anterior displacement of the ascending colon [7,17]. Paracecal hernias are the second most common subtype of congenital IH [7,18]. The hernia pocket typically extends under the cecum and/or into the right paracolic gutter. Imaging may demonstrate displacement of the cecum and abnormal anatomical relationship between the small intestines and the cecum/ascending colon [7,18,19].

In conclusion, we report a case of congenital peritoneal encapsulation, a rare cause of SBO within a virgin abdomen. Given that 10% of SBO occur within virgin abdomens, we briefly reviewed the differential diagnoses, including inferior ileocecal fold pathology, intra-abdominal adhesions, and internal hernias (table 1). Special consideration is needed to make this diagnosis and confirm the underlying etiology. Therefore, physicians should have a high index of suspicion and use the patient's

history, physical exam, laboratory, imaging, and intro-operative findings to assist in making the diagnosis of SBO in the patient with a virgin abdomen.

Table 1: Differential diagnosis and distinguishing features of SBO in a virgin abdomen.

Differential Diagnosis	Distinguishing Features
Peritoneal Encapsulation	- Congenital malformation - Thin non-adherent peritoneal membrane - Specific physical exam findings: inconsistency of abdominal wall and symmetric/fixed abdominal distension
Abdominal Cocoon Syndrome	- Acquired - Fibrocollagenous membrane with intra-capsule adhesions - Higher incidence in tropical countries - May encase solid organs
Sclerosing Encapsulating Peritonitis	- Acquired - Fibrocollagenous membrane with intra-capsule adhesions - No solid organ involvement
Intra-Abdominal Adhesions	- Congenital or acquired - Most common cause - Highly vascularized collagen bands that connect intra-abdominal structures
Congenital Internal Hernia	Paraduodenal hernia - Most common subtype of IH - Left sided: imaging may demonstrate small bowel within a hernia sac at the ligament of Treitz with mesenteric vessel anomalies - Right sided: imaging may demonstrate anterior displacement of the ascending colon Paracecal hernias - Imaging may demonstrate displacement of the cecum and abnormal anatomical relationship between the small intestines and the cecum/ascending colonb

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Manuscript Information: Received: September 05, 2018; Accepted: December 18, 2018; Published: December 31, 2018

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Citation: Roider LA, Ahmad S. Peritoneal encapsulation: One of several rare etiologies of small bowel obstruction in a virgin abdomen and a discussion of the differential diagnosis. Open J Clin Med Case Rep. 2018; 1498.

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