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CASE REPORT

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Chronic constipation and acute small bowel obstruction due to small bowel encapsulation: A case report

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Key Clinical Message

Peritoneal encapsulation is most of the time asymptomatic and is found incidentally, but when symptomatic it usually presents with bowel obstruction. CT scan is a gold standard for the diagnosis of bowel encapsulation.

Abstract

Congenital peritoneal encapsulation (CPE), abdominal cocoon syndrome (ACS), and sclerosing encapsulating peritonitis (SEP) are syndromes in which the small bowel is encapsulated. Small bowel encapsulation is usually asymptomatic and rarely presents with small bowel obstruction. In this article, we report a 65-year-old man who presented to our hospital with signs and symptoms of small bowel obstruction. He underwent an urgent operation, and small bowel encapsulation was diagnosed. One year after the surgery, all symptoms improved.

KEYWORDS

abdominal cocoon syndrome (ACS), congenital peritoneal encapsulation (CPE), sclerosing encapsulating peritonitis (SEP), small bowel encapsulation

1 | INTRODUCTION

Small bowel obstruction (SBO) is a prevalent surgical emergency responsible for about 20% of all emergency laparotomies with considerable medical costs.[1,2]

In approximately 90% of patients, small bowel obstruction arises from adhesions, hernias, and neoplasms. The adhesion band is the most common reason for SBO.[2,3] Remainder causes include small bowel tumors, Crohn disease, and others.[1] However, SBO has another unusual cause, such as a foreign body or has been occurred post-traumatic.[4–6]

Congenital peritoneal encapsulation (CPE) is a congenitally scarce condition where the accessory peritoneal membrane is encapsulated the small bowel.[7] Other syndromes with small bowel encapsulation are abdominal cocoon syndrome (ACS) and sclerosing encapsulating peritonitis (SEP). The primary etiologies of both syndromes are idiopathic, and the second one is related to chronic peritoneal dialysis.[8]

All of these conditions are usually accidentally diagnosed during surgery or autopsy. Otherwise, some cases became symptomatic. Rarely did the issues present with bowel obstruction signs and symptoms.[7] This article described a patient with small bowel obstruction and chronic constipation due to small bowel encapsulation.

2 | CASE PRESENTATION

A 60-year-old man with a history of diabetes mellitus, hypothyroidism, and appendectomy 35 years ago presented to our Emergency Department with colicky abdominal pain. His pain was spread over the abdomen without nausea and vomiting. The last defecation occurred 10 days ago, but with gas passing. He had a history of chronic constipation. He was treated with levothyroxine, metformin, and prazosin. On physical examination, the vital signs were stable, with no evidence of fever, tachycardia, or tachypnea.

His abdomen was distended, soft, without tenderness, and with decreased bowel sounds. Rectal examination demonstrated an empty ampulla. His laboratory findings were all within normal range except his hemoglobin (10.9) and platelets count (80,000) that showed anemia and thrombocytopenia. The plain X-ray of the Abdomen showed multiple air-fluid levels. For more clarity of bowel obstruction, the abdomen and pelvic computed tomography (CT) scan was done (Figure 1) and revealed the saclike appearance of jejunal and ileal loops in the central region of the abdominal cavity with twisting and engorgement of superior mesenteric vessels just left to the midline in the epigastric region.

According to all findings, a laparotomy was proposed. A sac encapsulating the small bowel from the ligament of Treitz to the 90 cm ileocecal valve was identified. The sac was excised entirely, and adhesiolysis was achieved. There was not any evidence of intestinal ischemia or perforation. Histopathology demonstrated fibro-hyalinized and adipose tissue with mixed inflammation, partly bordered with fibrinous exudate. He spent the postoperative period uneventfully and was discharged without complications. After 1 year of follow-up, he was asymptomatic.

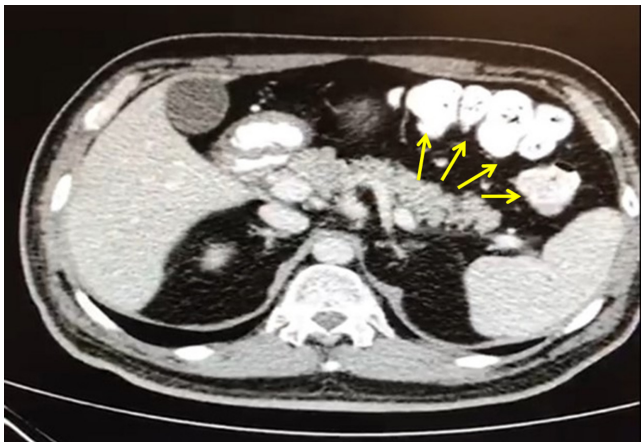

FIGURE 1 Abdominal CT scan (axial view). The saclike appearance of jejunal and ileal loops in the central region of the abdominal cavity (yellow arrows).

3 | DISCUSSION

Peritoneal encapsulation (PE) included three different conditions: congenital peritoneal encapsulation (CPE), abdominal cocoon (AC), and sclerosing encapsulating peritonitis (SEP).[7]

Congenital peritoneal encapsulation (CPE) is a malformation of the gastrointestinal tract. It is specified by the accessory peritoneal membrane, which covers a variable extent of the small bowel. CPE is a congenital condition with no clear etiology. Nonetheless, during weeks 8–10, some evidence confirms the considerable effect of fetal midgut herniation on the umbilical cord. The widely used theory of Papez illustrates CPE as an aberrant peritoneal adhesion between the linings of the physiological umbilical hernia and the caudal duodenum.[7,9] The peritoneum encapsulates the midgut within the cord, which results in a sac-like shape on the walls of the hernia. Therefore, the neck of this sac becomes close to the caudal duodenum. In case of adhesion between these peritoneal layers, the peritoneum must tolerate considerable traction forces, lining the midgut when the hernia is getting milder. Possibly this leads to peeling off and surrounding the bowel as an extra-peritoneal accessory sac.[9,10]

CPE is commonly asymptomatic and diagnosed during autopsy or operation for another cause. The most complication caused by CPE is small bowel obstruction.

Plain radiographs may be helpful but not specific. It demonstrated the dilated small bowel loops or multiple air-fluid levels. Furthermore, CT is helpful and maybe reveals the membranous capsule and the sac.[11]

The other two conditions with intestinal encapsulation included abdominal cocoon (AC) and sclerosing encapsulating peritonitis (SEP). AC is an acquired idiopathic condition with male dominance. Like CPE, the etiology and pathogenesis are poorly understood, but the theory that subclinical peritonitis developed to AC generally agrees. The underlying cause of this condition included viral infection, retrograde menstruation, and pelvic inflammatory disease (PID).[8,11] This theory cannot justify the male dominance of AC.[11]

SEP is also an acquired condition that mainly occurs in peritoneal dialysis patients. 20% of cases with peritoneal dialysis are expected to be developed into SPE after 8 years.[12] Other underlying causes included peritoneal shunts, tuberculosis, malignancy, systemic lupus erythematosus, sarcoidosis, and Mediterranean fever.[11,13]

The histopathology of CPE is normal peritoneal, but the histopathology of AC and SEP differ. In AC and SPE, the mechanism is the formation of a thick fibrotic membrane due to inflammation.[14]

Until now, no criteria or specific radiologic signs for PE diagnosis exist. But plain radiography or CT may be helpful. Plain radiographs demonstrated the dilated small bowel loops or multiple air-fluid levels. Furthermore, CT may show the membranous capsule and sacs and it is the gold standard for PE diagnosis until now.[15]

In our case, similar to Abdallah Mohamed et al. case study, CT was able to suggest intestinal encapsulation as the differential diagnosis of bowel obstruction before the operation.[7]

The most challenges are in asymptomatic cases discovered during operation with another cause, especially in minimally invasive methods, and could be the source of confusion. So, more studies about the anatomy of these anomaly types are necessary.

4 | CONCLUSION

Peritoneal encapsulation (PE) is an entity that is encapsulated in the small intestine. Cause and mechanism are divided into three categories: congenital peritoneal encapsulation (CPE), abdominal cocoon (AC), and sclerosing encapsulating peritonitis (SEP). Most of them are asymptomatic and accidentally discovered. If become symptomatic, most presentation is bowel obstruction. Sometimes CT can help to diagnose before surgery and demonstrate sac. So, more studies about the anatomy of these types of anomalies are necessary.

AUTHOR CONTRIBUTIONS

Seyed Hossein Hajimirzaei: Data curation; formal analysis; investigation; writing – review and editing. **Maryam Abbasi:** Data curation; formal analysis; investigation; writing – review and editing. **Nasser Malekpour Alamdari:** Conceptualization; investigation; project administration; supervision; writing – review and editing. **Sara Besharat:** Data curation; formal analysis; writing – review and editing. **Yeganeh Farsi:** Data curation; formal analysis; writing – original draft. **Atoosa Gharib:** Data curation; formal analysis; writing – review and editing. **Mahdiyeh Sadat Seyyedi:** Data curation; formal analysis; writing – original draft.

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CONFLICT OF INTEREST STATEMENT

Authors of this article claim that they do not have any conflict of interest.

DATA AVAILABILITY STATEMENT

All data are available on request from the corresponding author.

CONSENT

After a complete clarification of the aim of the study, written informed consent was obtained from the patient, including to use and publish the patient's history, findings of physical examination, and paraclinical studies unidentifiable and anonymously.

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