INTERNAL HERNIA: ISOLATED SMALL INTESTINAL PERITONEAL ENCAPSULATION, A RARE CAUSE OF ACUTE INTESTINAL OBSTRUCTION IN ADOLESCENT: A CASE REPORT

[1]Abiahu J A, [2]Mbaeri T U

[1].Surgical Unit, St. Charles Borromeo Specialist Hospital Onitsha,
[2].Royal Care Specialist Hospital Awka; both in Anambra State, Nigeria

ABSTRACT

Intestinal peritoneal encapsulation as the sole cause of acute intestinal obstruction in adolescent is rare. It poses a diagnostic dilemma where there is lack of high technological equipment to aid diagnosis, usually confirmed at surgery. A good knowledge of this condition will raise the index of suspicion and guide prompt surgical intervention, thus this report.

KEYWORDS: acute intestinal obstruction, adolescent, peritoneal encapsulation.

NigerJmed2018: 374-376
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INTRODUCTION

Acute intestinal obstruction is a surgical emergency with variable etiological factors in all age groups. Peritoneal encapsulation, which defines the encasement of intestine in an accessory peritoneal sac, is one of the rare causes, usually confirmed at laparotomy.[1,2] Delay in diagnosis may pose risk to the patient with attendant morbidity and mortality.

To my knowledge, isolated small intestinal peritoneal encapsulation in adolescents has not been reported in the literature. We therefore report a case of acute small bowel obstruction in an adolescent caused by only peritoneal encapsulation. A good knowledge of this clinical condition is important for increased index of suspicion, prompt diagnosis and treatment.

CASE REPORT

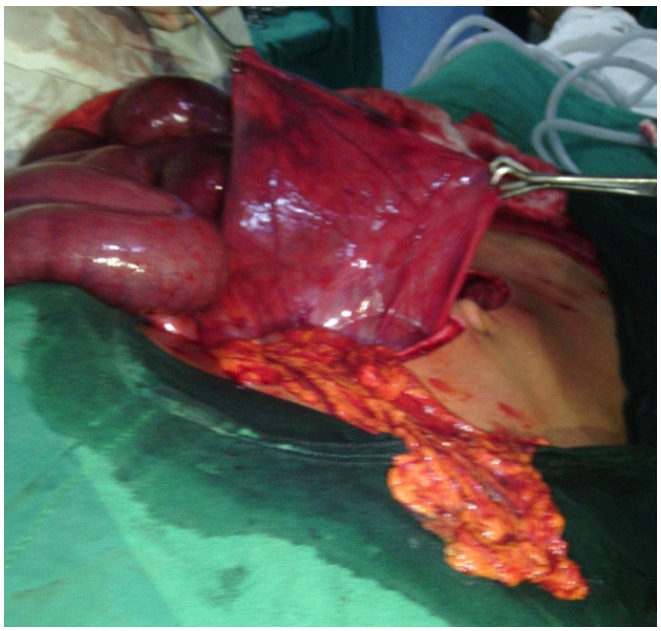
Mr I.W is a 17 year old boy who presented with 2 day history of abdominal pain and vomiting and a day history of abdominal distension. Abdominal pain was insidious in onset, rapidly progressive, centrally located, severe, colicky and relieved mildly by vomiting. Two days into the illness, the pain became constant with onset of generalized abdominal distension. No identified precipitating factor. Patient vomited about 9 times, each episode measuring about 200mls which was non-projectile and bilious. There was associated passage of frequent non-bloody, non-mucous loose stool with last episode about 18 hours prior to presentation. No associated fever. No history of dietary indiscretion or drug abuse preceding onset of symptoms. No history of prior similar episodes of abdominal pains or abdominal surgeries. No known drug or food allergy. His genotype is AA.

Clinical examination revealed a young boy in acute painful distress, afebrile but dehydrated. Pulse rate was 102/minute. Other vital signs were normal. Abdomen was distended with generalized tenderness and guarding. Bowel sound was reduced. Plain abdominal Xray showed no obvious multiple air-fluid levels, but dilated bowel loops. Abdominal Ultrasound suggested large bowel obstruction with mild ascites but no obvious intraabdominal mass or organomegaly. Serum electrolytes revealed marginal hyponatremia and hypochloremia. Bicarbonate, Urea and Creatinine were within normal range. Full blood count and differential count showed mild leucocytosis with

Correspondence: Abiahu Joseph. A,
Department of Surgery, Nnamdi Azikiwe University,
Nnewi Campus, Anambra State
Email: jabiahu2000@yahoo.com,
Tel: +234 806 231 8058.

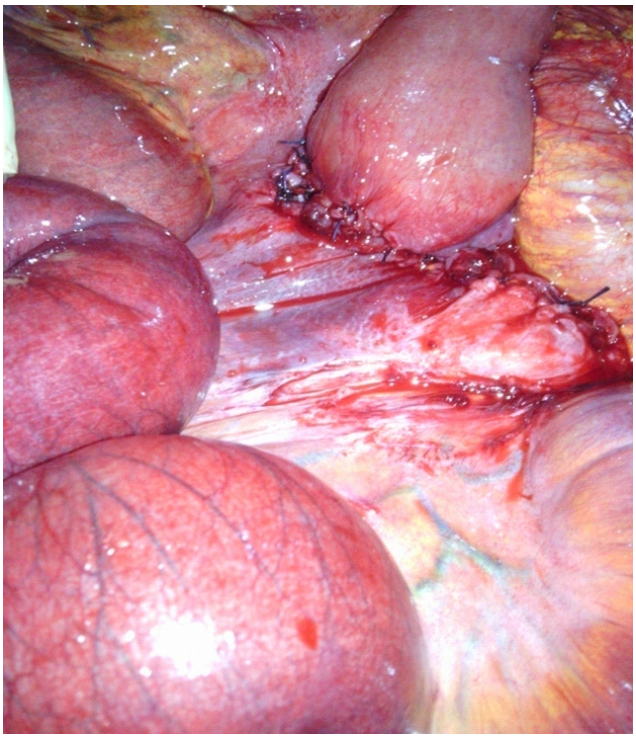
neutrophilia. Haemoglobin level was 12g/dl. A clinical diagnosis of acute abdomen was made to query cause. He was given intravenous fluids and antibiotics. He had exploratory laparotomy within 36 hours of presentation. Intraoperative findings included duodeno-jejunal mesenteric pouch with poor vascularity extending superiorly and inferiorly from duodeno-jejunal junction to about 6cm medial to the mid-portion of ascending colon, and laterally from the ascending colon to the mid-portion of posterior parietal peritoneum medially with only one opening at the duodeno-jejunal junction (fig 1) through which 48cm ileum herniated into the pouch with marked dilatation of proximal segments outside the pouch. The encased segment of gut was hyperaemic. All portions of both the small and large guts were viable with no mass lesion. The encapsulated segment was reduced

Figure 1: Jejuno-duodenal mesenteric pouch from which the herniated gut was reduced.



The redundant pouch excised near its neck with subsequent reconstruction of parietal peritoneum (fig 2). Commencement of abdominal closure was judged to be under tension thus an ileotomy and gut decompression was done. A 2-layer extramucosal repair of ileotomy was done and laparotomy wound closed in layers. Histology of segment of excised pouch showed variable sized dilated and engorged blood vessels supported by loose stromal connective tissues.

Figure 2: Excision of Pouch and parietal peritoneal reconstruction.



Postoperative period was uneventful and patient discharged home on the 9th day. One week, one month, and 3 month follow up visits were unremarkable.

DISCUSSION

Intestinal Peritoneal encapsulation, abdominal cocoon and sclerosing encapsulating peritonitis (SEP) are close differential diagnosis with similar pattern of presentation, but dissimilar pathologies.[1,2] Occurrence of more than one pathological entity in a clinical scenario has also been described.[2,3,4] Peritoneal encapsulation was first described by Cleland in 1868.[2] It is a rare developmental abnormality in which the intestine is encased in an accessory peritoneal sac.[1, 2, 5] The accessory peritoneal sac is believed to be derived from the yolk sac at 12th embryological week when the peritoneum of the physiological umbilical hernia is drawn into the abdominal cavity along with the mid gut.[1] Variable anatomical descriptions of this accessory peritoneal sac have been reported in the literature.[1,2,5] In this case report, we observed an accessory peritoneal sac with only one opening into the sac adjacent to the duodeno-jejunal junction with attachment spanning superiorly and inferiorly from the duodeno-jejunal junction to about 6cm medial to

the midportion of the ascending colon and latero-medially from medial aspect of ascending colon to mid portion of posterior parietal peritoneum. The encapsulated small intestine was also not attached to the inner surface of the accessory peritoneal sac with the greater omentum lying freely on top of it. These features were also highlighted in some reports. [2,6]

Peritoneal encapsulation may be asymptomatic with incidental finding at laparotomy for other disease conditions.[1,2,3] Features of bowel obstruction are common patterns of presentation. However, preoperative diagnosis is usually difficult. Naraynsingh et al[1] described two clinical signs helpful in preoperative diagnosis. These include a fixed, asymmetrical distension of the abdomen which does not vary with peristaltic activity due to unvarying position of the fibrous capsule, and the difference in the consistency of the abdominal wall to palpation with the flat, firm area due to dense fibrous capsule while the distended soft area is due to the thin walled distended small intestine with no overlying fibrous layer. However, these signs may also be present in abdominal cocoon and SEP.[2] These abdominal signs described by Naraynsingh et al[1] were not noted in this reported case probably masked by signs of peritoneal irritation.

The goal standard diagnostic tool is contrast enhance computerized tomographic (CECT) scan with multiplaner reformatted and maximum intensity projection images.[2,7] This can show the peritoneal capsule, bowel thickening or calcification, loculated ascites and cauliflower sign (cluster of bowel loops restricted in an area). CECT scan can also give information on the degree of obstruction and the type of bowel loops invoved. Abdominal ultrasound may also be useful but detailed information may be limited by excess bowel gas associated with this condition. Plain abdominal X-ray is usually not helpful, with non-specific features of intestinal obstruction as observed in this report and other reports.[1,2,9] The histology of accessory peritoneal sac is predorminantly connective tissues with or without inflammation or fibrosis[2,6] as also observed in our report.

Exploratory laporotomy is diagnostic and therapeutic in majority of cases. The extent of surgery depends on intraoperative findings which may include complete excision of the accessory sac. In our patient, an additional decompressive ileotomy and subsequent repair was done to achieve a tension-free abdominal wound closure.

The unpredictability of acute attacks of this condition with its sequalae emphasize the need for awareness, prompt diagnosis and treatment. Since considered a developmental abnormality, identification of fetal and maternal risk factors and need for prenatal diagnosis and treatment calls for further studies.

Financial support/funding: None
The authors have no conflict of interest.
Patients contact could not be reached for consent at the time of this report.

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