Case Report

INTERNAL HERNIA WITH ABDOMINAL COCOON - A CASE REPORT AND REVIEW OF LITERATURE

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ABSTRACT

A 19-year-old mentally challenged male presented with features suggestive of Acute Abdomen. A globular mass was palpable in epigastrium and left hypochondrium region. A provisional diagnosis of intestinal obstruction made on the basis of erect X-ray abdomen and Ultrasonographic findings for which the patient was taken up for exploratory laparotomy. IntraOperatively, a well defined thick capsule surrounding the whole small bowel was seen. The capsule was opened to release the bowel which revealed features suggestive of internal hernia with an Ileal stricture (passable) and enlarged mesenteric lymph nodes. Histopathology of the capsule wall and stricture site showed chronic non-specific inflammatory reaction and mature fibrous tissue. Serum ADA and histopathology of mesenteric lymph node confirmed the tuberculosis. Finally a diagnosis of Internal Hernia with Abdominal Cocoon secondary to Abdominal Tuberculosis was made. **Kevwords:**

INTERNAL HERNIA, ABDOMINAL COCOON, MESENTERIC LYMPHADENOPATHY, SERUM ADA

INTRODUCTION

Internal hernia may present with signs and symptoms of obstruction with varied intraoperative findings, but internal hernia presenting with abdominal cocoon leading to intestinal obstruction is very uncommon association. Majority of abdominal cocoons cases are either primary or idiopathic. There are reports of abdominal cocoon cases secondary to tuberculosis in literature and are mainly from developing countries like India.

CASE REPORT

We report a case of 17 year old male patient who presented with complaints of abdominal pain, recurrent bilious vomiting and abdominal distension since 5 days. He had history of similar pain episode 5 months before which was treated conservatively. He was not having

past history of any major medical or surgical illness. The patient was mentally challenged since childhood, hence the history was given by his father.

The patient was thin built with signs of dehydration and less cooperative because of being mentally challenged. Pulse was 116/minute, temperature 37.6°C, blood pressure 110/70 mm Hg. General and systemic Examinations were normal. The abdomen was distended and moderately tender with hyper dynamic bowel sounds. We found a tender palpable lump with mild guarding in supra umbilical, epigastric and left hypochondriac region. There was no organomegaly and digital rectal examination was normal.

Blood investigation revealed a total leukocyte count of 17400 cells/ml, with predominantly neutrophils [94%], hemoglobin of 15.9 g %, normal serum chemistry and normal urine analysis. Chest X- ray was normal but erect X-ray abdomen revealed multiple air-fluid levels which were centrally located, with no free gas under the diaphragm. USG findings suggested dilated bowel loops with typical to and fro movements and minimal inter bowel free fluids suggesting the intestinal obstruction.

Emergency exploratory laparotomy was done for clinical diagnosis of mechanical small bowel obstruction. Intra Operatively, an encapsulated cystic lump with multiple overlying peritoneal layers was noticed in the left hypochondrium posteroinferior to stomach and in supracolic compartment. The whole small bowel was covered by a dense whitish and grayish 5 mm thick membrane resembling a large cocoon (Figure no.1).On dissecting the layers of the cocoon, small bowel loops were found hugely distended and herniated (Figure no. 2) On gradual separation of the loops, the intermingled small bowel loops found to be coming from the very narrow defect in mesocolon of transverse colon, which were then gradually reduced (Figure no.3). The herniated small intestine was viable and the large intestine was normal. The small bowel on thorough inspection from ligament of Treitz to ileocaecal junction revealed one stricture in ileum at around 15 cm proximal to ileocaecal junction. The stricture was found to be passable, hence only the biopsy was taken from it. Multiple mesenteric lymph nodes were found enlarged and the biopsy was taken from one of them(Figure no.4). The excised cocoon wall, stricture site tissue and mesenteric lymph node were sent for histopathology examination. The defect in the transverse mesocolon was closed.

The histopathology examination revealed chronic non specific inflammatory reaction in cocoon wall and in the stricture site while tuberculous mesenteric lymphadenitis was reported in mesenteric lymph node. The serum ADA level was 57 in post operative period, strongly suggesting the tuberculous etiology.

The recovery in the post-operative period was uneventful; the nasogastric tube was withdrawn on 3^{rd} postoperative day and liquid diet started. The patient was discharged from the hospital on the 10th post-operative day after starting on anti-tuberculosis treatment. He has completed full course of Anti Tubercular Therapy, and he is in our regular follow up and has remained symptom free for the last 1 year.

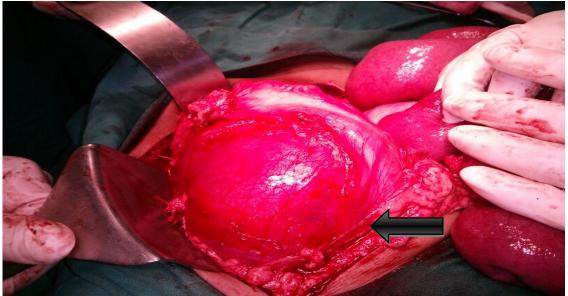


Figure no.1: Intra-operative findings with an arrow showing the abdominal cocoon occupying the upper half of abdomen.

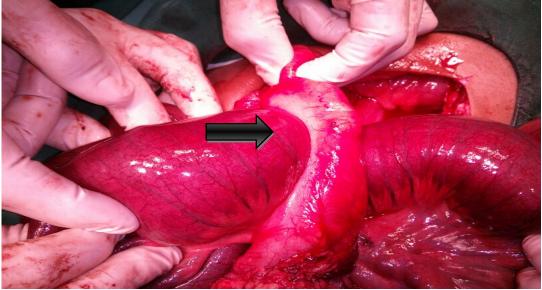


Figure no.2: Internal hernia seen after sac dissection. Arrow showing herniated bowel.

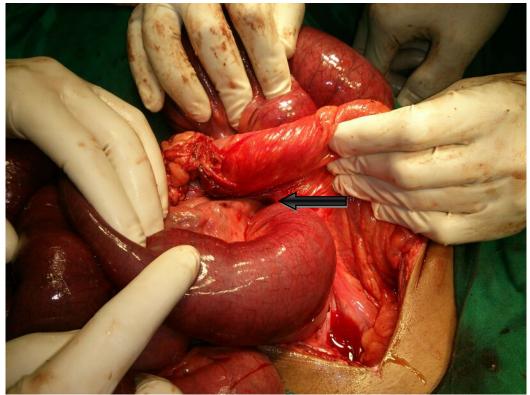


Figure no.3: Reduced internal hernia with defect.

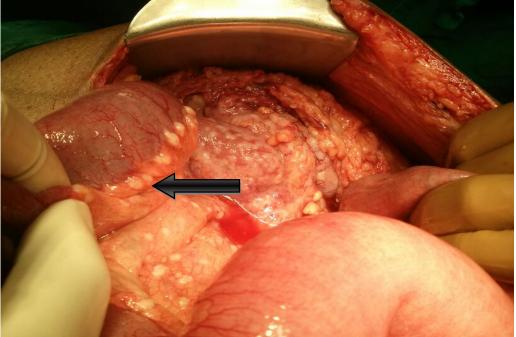
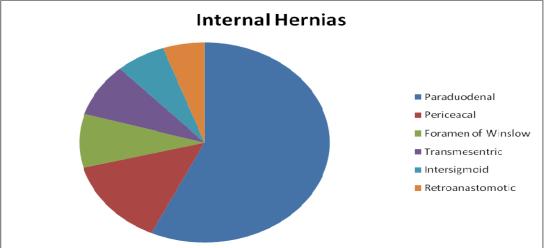


Figure no.4: Mesenteric Lymphadenitis DISCUSSION

Acute intestinal obstruction is one of the important and common cause of acute abdomen presenting as surgical emergency. The basic etiopathology usually are peritoneal bands, intestinal adhesion or obstructed hernia, or at times the rare causes may be encountered such as the internal herniation or 'abdominal cocoon', Though the combination of above two is very rare but still possible and even rare case reports are published in literature.

Internal hernias are defined by the protrusion of viscera through a normal or abnormal peritoneal or mesenteric aperture within the confines of the peritoneal cavity. The reported incidence of internal hernia is less than 1% and they constitute about 5.8% of all cases of intestinal obstruction with more than 50% mortality rate.[1]

Meyers has sub classified internal hernias according to their location. [2]



Transmesentric hernias have a bimodal peak of distribution with no sex predilection. The first peak occurs in children and adolescence with etiology being congenital defect in the smallbowel mesentery, near the ileocecal region or ligament of Treitz, may be due to intrauterine intestinal ischemia [3]. The second peak which occurs in adult age group has mostly iatrogenic etiology such as prior abdominal surgery, especially with Roux-en-Y anastomosis, liver transplant etc. [4]. Other causes are trauma and inflammation.

The transmesentric internal hernias are further classified into three types. First is transmesocolonic which is more common after laparoscopic roux en y bypass surgeries. In the second type the small bowel passes through a defect in small bowel mesentry and the third type (which is also called as Peterson's type) the small bowel herniates behind the Roux loop before the small bowel eventually passes through the defect in the transverse mesocolon.[5]

In our case we found the transmesocolonic type of internal hernia but there was no history of any previous surgery.

Most transmesentric hernias present with sign and symptoms of acute intestinal obstruction i.e. peri umbilical abdominal cramps, nausea and abdominal distension. Frank vomiting is usually not there due to lower down level of obstruction. A tender abdominal mass is often palpable (Gordian knot of herniated intestines).[5]

Volvulus and strangulation is more common in transmesentric hernias as compared to other internal hernias due to smaller aperture of defect and lack of sac. The mortality is 50% in treated group and 100% in non treated group. [4]

Imaging studies are not very helpful in the diagnosis of transmesentric hernias due to confining sac. In x-ray abdomen, sign of gut obstruction with significant air in the gastric remnant may be seen.

The barium studies and the CT scans give the variable outcomes which depend upon the type of mesenetric hernia. In mesocolonic type the picture is often confused with paraduodenal hernia, small bowel volvulous or a closed loop hernia.[2] In differentiating with paraduodenal henia presence or absence of sac should be noted. In type 2 mesentric hernia there are typical findings described by Blachar et al. like presence of clustered bowel loops in the periphery of the peritoneal cavity, lateral to the colon (a reversal of the normal pattern),

with central, inferior, and posterior displacement of the transverse colon and displacement of overlying omental fat. [6]

The abdominal cocoon or 'sclerosing encapsulating peritonitis' (SEP) is a rare condition which is characterized by a fibrocollagenic cocoon-like sac enveloping the small bowel.

The first description of abdominal cocoon was given by Owtschinnikow in 1907 who termed it as "peritonitis chronica fibrosa incapsulata". Various other authors termed it differently, but the first description with term "abdominal cocoon" was given in 1978 by Foo et al. [7]. SEP is a rare disorder with reported incidence varying between 0.4% to 5.5% and a higher incidence 19.2% in the patients maintained on the peritoneal dialysis for 5-8 yrs.[8,9, 10] SEP is of two types – primary or idiopathic and secondary.

SEP and Internal Herniation are both infrequent entities and their coexistence is a rare finding. The transmesocolonic type of internal hernia is usually iatrogenic but in the present case, the transmesocolonic internal hernia was not associated with any prior history of abdominal surgery. Although tubercular infection might have played a role in etiopathogenesis of both condition but the association is still debated. The indications of surgery in this case were features of acute abdomen with recurrent type of history. An ADA level in ascitic fluid analysis is needed for TB diagnosis, which was 57 in our case. During the surgery of internal hernia, lysis of adhesions, reduction of internal hernia and closure of mesenteric defects is usually sufficient and resection and anastomosis is rarely required which is a poor prognostic factor in SEP and internal herniation.

In our case, only sac dissection followed by reduction of hernia and closure of the mesenteric defect was sufficient and resection & anastomosis was not required.

CONCLUSION

Internal herniation secondary to abdominal cocoon is a rare cause of intestinal obstruction and its possibility must be considered in cases of intestinal obstruction with palpable abdominal mass. The treatment of choice is careful dissection of the sac with gradual separation of the bowel loops. Antitubercular therapy should be started postoperatively if histopathology and ADA levels are suggestive of tubercular etiology.

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