ABDOMINAL COCOON SYNDROME: A CASE REPORT AND REVIEW OF RELATED LITERATURES

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ABSTRACT:

Significance: Despite several cases reported worldwide, Abdominal Cocoon Syndrome still is a rare condition. No published reports have been found locally. This reminds us to always be prudent despite being presented with a common problem. The early correct diagnosis avoids the risk of complications. This aims to keep us abreast of the current practices and recommendations; to be better equipped in handling perioperative incidental findings of this entity prior to end of surgery.

Clinical Presentation: We present a case of a 30 year old man with subacute gastric outlet obstruction, malnutrition and delayed physical development.

Management: Initial imaging revealed dilated stomach and duodenum, concomitant distal jejunal and proximal ileal obstruction. Initially managed as SMA syndrome, but two weeks of conservative management failed. Due to incompatible clinical course, a small bowel series was requested showing delayed barium transit from stomach to ileum. Then a diagnostic laparotomy followed. A surprising intraoperative findings revealed a thick membrane covering the entire small intestines sparing the duodenum. Adhesiolysis and membrane dissection was done, releasing the trapped intestines. Histopathology confirmed the diagnosis. Patient was discharged 2 weeks post-operatively.

Recommendation: Always remain prudent in handling common problem and have a high index of suspicion. Maximize preoperative condition, decide on the timing of the definitive treatment, careful surgery, judicious postoperative care, and surveillance of possible complications are a must. Etiology remains elusive; future research should focus on relationship of prematurity at birth, smoking on the development of adhesions and membrane formation, and possible perinatal risk factors.

Keywords: Abdominal Cocoon Syndrome, Primary Sclerosing Encapsulating Peritonitis, Idiopathic Sclerosing Encapsulating Peritonitis, Primary SEP, Idiopathic SEP, Gastric Outlet Obstruction, Intestinal Obstruction.

INTRODUCTION:

It is not every day that we encounter unexpected cases. But these are the days that bring forth great challenge. And despite the best efforts placed in diagnosing such cases, it may still give us a "surprise" in the end. This is a case of Primary/Idiopathic Sclerosing Encapsulating Peritonitis (SEP) or "Abdominal Cocoon Syndrome" presenting as Gastric Outlet Obstruction. Due to limited knowledge regarding this disease entity during the time of the operation, intraoperative maneuvers to prevent future risk of complications were not made.

In the Philippines, no published reports have been found. Incidence of the disease locally is relatively unknown. Despite several cases reported worldwide, primary SEP still is considered a rare condition by almost all papers published.

This case highlights a rare clinical entity in a commonly encountered problem. Review of related literatures were made to keep us abreast of the current practices and recommendations. If similar perioperative findings were to be encountered; a "surprised" moment can be avoided and the recommended treatment can be initiated in anticipation of a possible problem prior to end of surgery.

CASE PRESENTATION

This is a case of 30 year old male with a 3 month history of epigastric pain and fullness aggravated by intake of meals. There was progression of symptoms to include post prandial vomiting, abdominal distention, frequent watery stools and increasing abdominal pain 1 month PTA. He was seen at the OPD with plates of X-rays showing dilated bowels, and stomach with minimal air fluid levels. *(Figure 1A)* Abdominal CT scan done showed dilated stomach and duodenum; to consider SMA syndrome and to consider concomitant distal jejunal and proximal ileal obstruction probably internal hernia. *(Figure 2)*

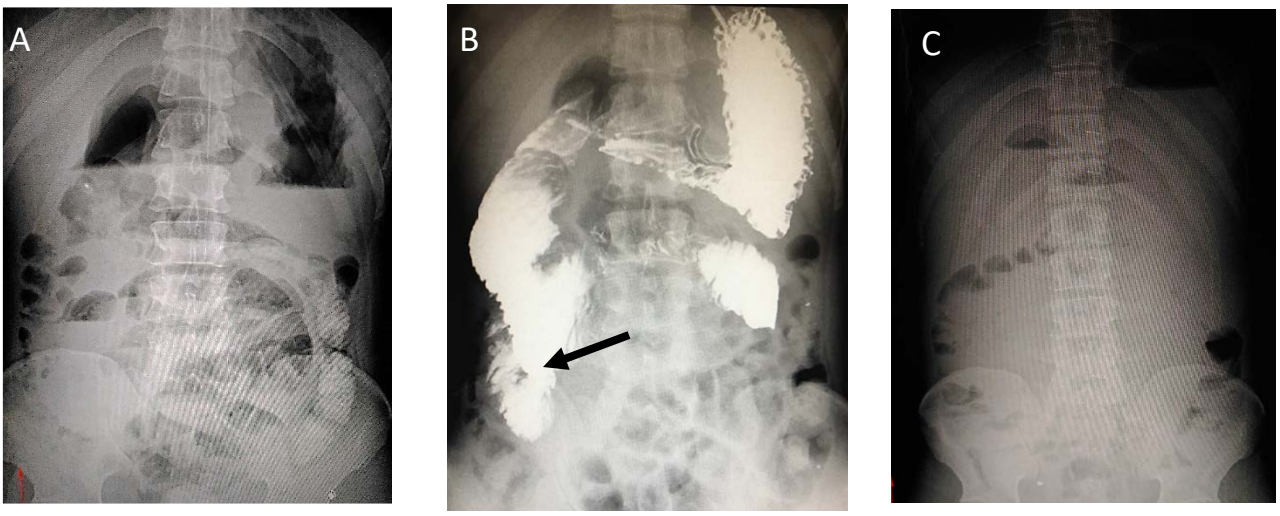

Figure 1: Roentgen Films: A. Plain Abdominal X-ray before admission.
B: Small Bowel Series, arrow shows possible diverticle
C: Plain abdominal X-ray 2 weeks post operation

Birth history showed that he was delivered prematurely. A 21 pack year smoker with bronchial asthma, who had his last attack 10 months prior to admission. Patient is physically underdeveloped relative to his age. Abdominal examination revealed a distended abdomen with normoactive bowel sound, and tympanitic all over. Epigastric fullness was noted.

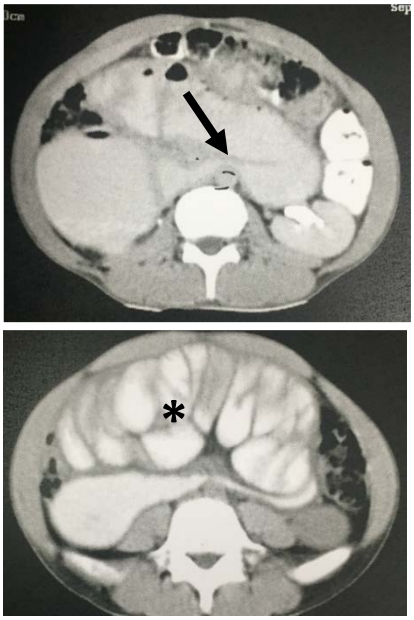

Figure2: CT scan of the abdomen with Contrast
Black arrow points to the stricture area of the duodenum.
Asterisk shows dilated small intestines.

Patient was admitted as Gastric Outlet Obstruction secondary to SMA syndrome. Review of the CT scan plates done and Surgery service referral made. Blood workups were unremarkable. Conservative management was done with TPN, IVF hydration, bowel rest and NGT decompression. Attempt to remove the NGT and start oral feeding, failed twice.

Initial diagnosis was not compatible with patient's course. A small bowel series was requested and showed partial obstruction at 3rd segment of duodenum suggestive diverticle at distal end of duodenum. Enteroenteric fistula considered at 2nd portion of duodenum towards a segment of ileum. *(Figure 1B*) Upper GI endoscopy was done, but showed only pangastritis. After 2 weeks of conservative management, a final decision to have patient undergo a diagnostic laparotomy was made.

Intraoperative findings were whitish thick membrane approximately 0.5cms in width covering the whole small intestines, sparing the duodenum. *(Figure 3A-C)* Upon opening the membrane revealed normal looking intestines with inter loop adhesions that can be easily lysed. Also the omentum was small. The membrane was biopsied revealing connective tissue stroma demonstrating fibrosis and vascular congestion. *(Figure 3D)*

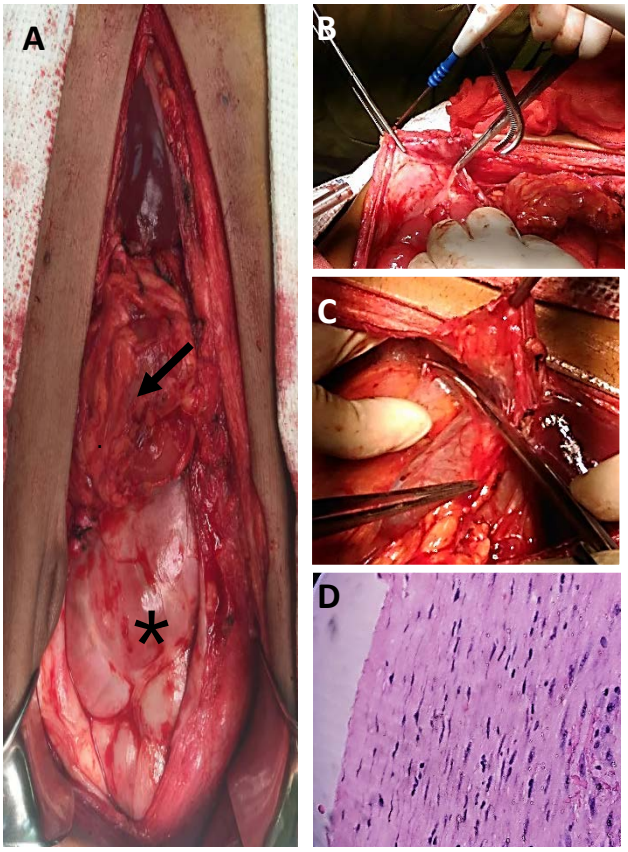

Figure 3: Intraoperative and histopathology findings
A: Thick membrane (*) with omental hypoplasia (arrow)
B: Membrane dissection revealing normal intestines.
C: Thin interloop adhesions easily lysed.
D: Histopathology showing fibrosis. (10x magnification)

Post operatively, the course was unremarkable. Patient was gradually introduced to per orem feeding. Repeat X-ray showed decreasing air fluid levels. *(Figure 1C)* After 2 weeks post-operation and patient able to tolerate regular diet, he was discharged. OPD follow up showed no recurrence of obstruction, and patient gained weight after 1 month.

DISCUSSION

It was in the beginning of the 19th century that Owtschinnikow coined the term *peritonitis chronica fibrosa incapsulata* for a disease entity that presented as intestines encased in a membrane.[1] In 1978 the term *" Abdominal Cocoon" Syndrome* was coined by Foo et al for the young girls with intestinal obstruction, upon operation with characteristic dense fibro collagenous cocoon-like membrane encasing their intestines.[2,5] This term is ascribed to the idiopathic form of Sclerosing Encapsulating Peritonitis (SEP).[3] There are two forms of SEP; the secondary SEP which an etiology is known. Primary SEP or the abdominal cocoon syndrome which the cause of the intraperitoneal membrane formation and inflammation is not identified.[3, 4, 5]

According to the extent of covered intra-abdominal organ, the abdominal cocoon syndrome can be categorized into three types. When the membrane encloses only a part of the small intestines it is type 1 cocoon syndrome. A complete encasement of the small intestines by the membrane is type 2 abdominal cocoon syndrome. Type 3 cocoon syndrome refers to the membrane covering the whole small intestines, with inclusion of other intra-abdominal organs, such as the appendix, cecum, ascending colon, and ovaries. [4,5] The clinical significance of this category maybe relevant to what will be the appropriate treatment, the clinical presentation and severity of symptoms of the patient. The broad involvement of Category 2 and 3 may require open surgery compared to category 1, which possibly can be managed conservatively or laparoscopically. Intraoperative findings in our case revealed a type 2 abdominal cocoon.

The exact etiology of this disease is yet to be elucidated. It may be a form of chronic irritation and inflammation or an exaggerated inflammatory reaction to an unidentified stimulus intraperitonealy.[6,7] Reports hypothesized the association of retrograde menstruation with concomitant viral infection and cell mediated inflammation incited by gynecological infection, intra-abdominal or retroperitoneal bleeding.[2,8]

There is a consideration for congenital abnormality, since there were reports that embryologic abnormalities accompany the disease such as omental hypoplasia, occurring in 41.7% in one report.[6,9-11] It was suggested that during embryogenesis, the membranous greater omentum descends along the transverse colon and encases the intestine.[11] One case report showed concomitant cryptochordism.[12] Thus when a primary SEP is suspected, examine the scrotal sac in males.

The presence of omental hypoplasia makes congenital anomaly highly probable as the cause of the primary SEP in our patient. This coupled with the fact that since infancy patient has failure to gain weight and has physical growth retardation relative to

his age group. Being delivered prematurely might be a risk factor for disease development, but no reports were found determining the relationship. Environmental factors such as smoking might be a risk factor in sustaining the formation of the adhesions and membrane formation.[13] One case reported a 20-pack year smoking history.[14]

Clinical manifestations are nonspecific. Suspicion should be raised when a patient presents with a prolonged intermittent obstructive symptoms with associated malnutrition.[10] But it should be membered that these patients can present with acute abdomen necessitating emergency surgery.[14,38]

In a case series involving 24 patients, intestinal obstruction and abdominal mass were the main presenting complaints. Eleven patients showed intestinal obstruction (45.8%) alone, with either acute intestinal obstruction (20.8%) or subacute obstruction (25%). Only three cases (12.5%) presented with asymptomatic abdominal mass. The rest of the 10 cases (41.7%) showed both intestinal obstruction and abdominal mass.[11]

The symptoms presented are due intestinal imprisonment by an encasing membrane, interloop adhesions, and enterokinesia. The partial small gut obstruction is a result of the kinking and compression of the intestine within the encasing membrane.[4] Due to prolonged narrowing of the intestinal lumen, maladaptive changes occur. Despite the adequate food intake, chronic subclinical obstruction resulted in both structural adaptation leading to duodenal dilatation and most likely functional alteration as well (motility and absorption), resulting in physical underdevelopment. Food aversion due to fear gastrointestinal symptoms and decreased food intake due to early satiety is also contributory to malnutrition.

Concern for intestinal perforation is present. But perforation in primary SEP is very rare, only one reported case which there was a 1cm ileal perforation due to the complete obstruction secondary to the adhesions.[15] There were other 2 cases reported that presented with ileal perforation, but did not specify if they were primary SEP; since 55% of the case studied were tuberculous in origin.[16]

Plain X-ray of the abdomen may only show features of intestinal obstruction.[6] Ultrasonography can demonstrate thick-walled mass containing bowel loops, loculated ascites and fibrous adhesions.[17] Small bowel series will show ileal loops clumped together within a sack, giving a cauliflower-like appearance on sequential films. But a delayed transit may be more diagnostic.[18] In our patient, the small bowel series did not demonstrate the cauliflower-like appearance, but there was marked alteration in the path of the barium swallowed which was read as fistula and delayed transit of barium.[19]

The characteristic CT findings show small bowel loops congregated to the center of the abdomen, encased by a soft-tissue density mantle that is not enhanced by contrast, and interloop collection. [18] It is not unusual for a CT scan of such patients to be read as internal hernia as what has been made in our patient. Most of the time the CT scan characteristics are not present. Repeated review of our patient's CT scan did not demonstrate the above characteristic. It is most likely that the dilated intestines obscured the visualization of the membrane or the membrane itself is thin.

Recommended is the combination of history, imaging, physical examination and high index of suspicion in order to diagnose pre operatively. Correct preoperative diagnosis were reported using this approach.[20] Histopathology is important in ruling out other causes especially tuberculosis.[7, 21]Biopsy of the membrane will show dense fibrocollagenic tissue along with or without inflammatory infiltrate.

Early preoperative diagnosis is very important to initiate early treatment and to prevent strangulation of the encaged segments especially in those who already presented with frequent obstruction. [22, 23] But preoperative diagnosis remains difficult. Only 3-16% of cases were correctly diagnosed preoperatively.[11, 24] Most of the cases were diagnosed during operation with the finding of a thick membrane. High index of suspicion is needed especially in patients who presents with intermittent intestinal obstruction.

The diagnosis of sclerosing encapsulating peritonitis needs to be separated from peritoneal encapsulation, a congenital malformation.[25] Peritoneal encapsulation also has encasing membrane in the intestines, but the membrane is thin and is covered by mesothelium resembling a normal peritoneum. The underlying intestines has no interloop adhesions, no associated omental hypoplasia, and most of the time, they are asymptomatic.[4]

Tuberculous peritonitis must be considered, especially in countries where the prevalence of abdominal TB is high. Manifestations of abdominal tuberculosis such as mesenteric abscesses, enlarged and caseating mesenteric lymph nodes, and tubercles over the bowel serosa should be sought during imaging and surgery.[21,26] In a series of 18 patients initially considered as abdominal cocoon syndrome, 55% were ultimately diagnosed as having intraabdominal TB. Features of TB were found in 5/9 patients diagnosed to have TB. The remaining 4 cases was diagnosed to have tubercular in etiology through histopathology.[16] Our case did not show any features of tuberculous peritonitis in imaging, surgery and histopathology.

Surgery remains the standard in the management of abdominal cocoon; simple membrane dissection and extensive adhesiolysis.[6,7,10] However in asymptomatic cases, conservative management can be made.[27]

Use of immunomodulators have been reported, including steroids, tamoxifen, mycophenolate and colchicine. [28,29] But the available data were not enough to support the practice. Besides, majority of the cases involved in these studies were secondary SEP. Decision should be weighed on the potential risk of the treatment. Applicability of this treatment approach might be useful in patients who had recurrent bouts of adhesions despite surgical treatment.

It is important to address malnutrition pre operatively. This has been shown to have lower post-operative complication, more postoperative satisfaction and lower complication risk.[28] Prophylactic appendectomy with or without appendix lesions, to prevent re-operation for possible acute appendicitis and the difficulty of the possible reoperation is recommended.[10] But this is still controversial. Especially in our patient who is already 30 year old, since the incidence of appendicitis is highest at 10-30 years old.[37] In this case the diagnosis was made perioperatively; no consent for the appendectomy was sought, and that the reason for performing the procedure will be for a theoretical difficulty in doing reoperation if appendicitis will develop.

Post-operative adhesion is a concern for SEP due to its inflammatory nature. It carries a morbidity risk of 12.5 %. [11] A retrospective analysis of 203 patients noted 55 (27%) had postoperative recurrent obstruction.[24] The use of medical sodium hyaluronate gel before closing the abdominal wall to prevent postoperative recurrence of adhesions in the abdominal cavity was reported; others advocate hydrocortisone wash. [10, 11, 14, 24, 30] Reduction in the incidence of adhesions were noted [OR 0.15 (95% CI: 0.05,0.43);p=0.0005] among those who were given hyaluronic acid intra operatively on patients who underwent non gynecologic laparotomy.[31] But the efficacy of this method for primary SEP is questionable due lack of well-set trials, because of the rarity of the disease. And besides the risk for abscess and wound complications are increased in these treatments.[32]

Laparoscopy can be used in the treatment of idiopathic SEP with complete recovery of patients. [30] But total laparoscopic surgery as a sole treatment, may delay the postoperative intestinal recovery, which is attributed to increased operation time and a large number of pneumoperitoneum stimulation. Instead it was recommended to combine laparoscopy with open surgery, which laparoscopy provide the rapid diagnosis then followed by laparotomy for the complete removal of the membrane and adhesions. [5, 10]

Post-operative care is very important in the management. Maneuvers to prevent infection, post-operative ileus and DVT should be initiated. Gradual progression of feeding must be made, from liquid diet to general diet. This is necessary to allow time for the body to adjust for the repaired gastrointestinal tract that was in prolonged maladapted state. A Report showed that this may take up to 14 days.[12] Our patient was able to tolerate regular diet after 10 days post operatively.

Prognosis for these cases are good. Once diagnosis has been made and laparotomy done, mortality from the syndrome is unusual, since surgical treatment provides definitive cure.[34] Although, there were reports of mortalities post operatively. These were related to advance age, poor preoperative condition, concomitant comorbidities (hepatitis C, liver cirrhosis, malignancy), and inadvertent intraoperative intestinal perforations leading postoperative leaks, anastomotic leaks and persistent obstruction which ultimately lead death from sepsis and cardiac decompensation. [16, 25, 27, 35, 36]

CONCLUSION:

Abdominal Cocoon Syndrome is still a rare disease entity, but must be considered in patients who presented with gastric outlet obstruction and with radiologic signs of small intestinal obstruction in a background of failure to gain weight, malnutrition and a delayed physical development. Due to lack of properly set trials for the disease recommendations such as prophylactic appendectomy, application of intraperitoneal anti adhesion agents, and immunosuppressive agents, the decisions are left at the discretion of the attending physician.

Correct preoperative diagnosis remains a challenge. It is recommended to always remain prudent in handling common problem and have a high index of suspicion. Always check for other concomitant congenital anomaly when faced with this diagnosis. To achieve an uneventful course, it is suggested to maximize preoperative condition of the patient, decide appropriately on when to do the definitive treatment, being cautious in doing surgery, judicious postoperative care, and surveillance of possible complications. Etiology remains elusive for the disease, thus for future studies it is suggested to focus on the relationship of prematurity at birth, smoking on the development of adhesions and membrane formation, perinatal risk factors and other possible triggers.

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