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A Case Report on Oligvie's Syndrome

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Abstract: *Acute colonic pseudo-obstruction first described by Sir HeneageOgilivie in 1948 is a poorly understood syndrome and is characterized by signs of large bowel obstruction without a mechanical cause. This condition usually develops in hoapitalized patients and is associated with a range of medical and surgical conditions. If inappropriately managed, it may cause ischemic necrosis and colonic perforation. The main underlying cause is an imbalance in the autonomic innervations (sympathetic over activity and parasympathetic suppression) has been thought to be the patho-physiological factor in the causation of this condition. A healthy, 33 years old female presented abdominal distension and tachycardia on post op day#4 after an uncomplicated elective caeserean section and was diagnosed with acute colonic pseudo-obstruction also known as Ogilivie's syndrome. She underwent Emergency Exploratory Laparotomy And Underwent Limited Resection Of The Ascending Colon And Caecum With Ileotransverse Anastomosis.*

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I. Introduction

Acute colonic pseudo-obstruction (Ogilvie's syndrome) is a disorder characterized by acute dilatation of the colon in the absence of an anatomic lesion that obstructs the flow of intestinal contents.

Etiology

Acute colonic pseudo-obstruction usually occurs in hospitalized or instpatients in association with a severe illness or after surgery[1-5]. Most common predisposing conditions were nonoperative trauma, infection, and cardiac disease, each of which were associated with 10 percent of cases[1]. Acute colonic pseudo-obstruction is also well-documented after kidney transplantation, and possible contributing factors include obesity,cumulative dose of prednisone and mycophenolate mofetil[6]

Epidemiology

Acute colonic pseudo-obstruction usually involves the cecum and right hemicolon, although occasionally colonic dilation extends to the rectum. Acute colonic pseudo-obstruction appears to be more common in men and in patients over the age of 60 years[1] Acute colonic pseudo-obstruction is a rare complication of surgery, occurring in 0.06 percent of patients after cardiac surgery, 0.29 percent of burn patients and 0.7 to 1.3 percent of patients after orthopedic surgery[4,7] In surgical patients, symptoms usually present at an average of five days postoperatively.

Pathogenesis

The precise mechanism by which colonic dilation occurs in patients with acute colonic pseudo-obstruction is unknown. The association with trauma, spinal anesthesia and pharmacologic agents suggests an impairment of the autonomic nervous system. Interruption of the parasympathetic fibers from S2 to S4 leaves an atonic distal colon and a functional proximal obstruction[1,8].In patients with acute colonic pseudo-obstruction, increasing colonic diameter accelerates the rise in tension on the colonic wall, increasing the risk of colonic ischemia and perforation. The risk of colonic perforation increases when cecal diameter exceeds 10 to 12 cm and when the distention has been present for greater than six days[9] The duration of dilation is probably more important than the absolute diameter of the colon[10,11]

Rare cases have been reported in association with atrophic visceral myopathy with an extremely thin colonic wall, atrophic circular and longitudinal muscularispropria without inflammation or fibrosis and unaffected ganglion cells and myentericplexus[12]

II. Clinical manifestations

The main clinical feature in patients with acute colonic pseudo-obstruction is abdominal distension. Abdominal distension usually occurs gradually over three to seven days but may develop rapidly within 24 to 48

hours and associated with abdominal pain,nausea and vomiting may be seen in up to 60 percent of patients. Constipation and paradoxically, diarrhea have also been reported in approximately 50 and 40 percent of patients, respectively[1,13] In rare cases, abdominal distention can cause dyspnea[1,13] On physical examination, the abdomen is tympanitic, but bowel sounds are present in almost 90 percent of patients[1]

Diagnosis

The diagnosis of acute intestinal pseudo-obstruction should be suspected in patients with abdominal distension or pain and a physical examination that reveals a distended and tympanitic abdomen. The diagnosis of acute intestinal pseudo-obstruction is established by abdominal imaging.

Laboratory tests

Complete blood count, electrolytes, and serum lactate levels. In patients with a suspected perforation and diffuse peritonitis, serum aminotransferases, alkaline phosphatase, bilirubin, amylase, and lipase levels should be obtained to rule out other causes of acute abdominal pain. In patients with diarrhea, we also perform stool cultures and stool evaluation for *C. difficile* toxin.

Imaging:

Abdominal CT scan

Contrast enema

Abdominal radiographs

Differential Diagnosis

The differential diagnosis of acute colonic pseudo-obstruction includes other causes of acute colonic dilation. Mechanical obstruction Toxic megacolon.

III. Management

- Due to the risk of colonic ischemia and perforation, patients with acute colonic pseudo-obstruction should be carefully monitored with serial physical examinations and plain abdominal radiographs every 12 to 24 hours to evaluate the colonic diameter. In addition, we perform laboratory tests every 12 to 24 hours, including a complete blood count and electrolytes[10]
- Initial management of acute colonic pseudo-obstruction is usually conservative in patients without significant abdominal pain, extreme (>12 cm) colonic distension or signs of peritonitis and those who have one or more potential factors that are reversible.

In patients with cecal diameter >12 cm and in patients who have failed 24 to 48 hours of conservative therapy, pharmacologic therapy with neostigmine.[14,15]

In patients who fail or who have contraindications to neostigmine, should advised for colonoscopic decompression. In patients whose acute colonic pseudo-obstruction may be precipitated by opiates,should be administered with subcutaneous methylnaltrexone, prior to percutaneous or surgical decompression[16] Because of the possibility of recurrence after the colonoscopic decompression, some authors recommend use of a multiperforated rectal tube and oral or colonic administration of polyethylene glycol laxative[17]

Neostigmine:

Neostigmine, anacetylcholinesterase inhibitor is indicated in patients with acute colonic pseudo-obstruction and cecaldiameter>12 cm or in patients who fail 24 to 48 hours of conservative therapy.

- **Dose and administration** – Neostigmine (2 mg) should be delivered by slow intravenous injection over five minutes, with continuous monitoring of vital signs and electrocardiograph for 30 minutes and continuous clinical assessment for 15 to 30 minutes[18] Patients should be kept supine on a bedpan and atropine should be available at the bedside to treat bradycardia associated with neostigmine[18] In patients with successful colonic decompression, should be administered polyethylene glycol electrolyte balanced solution to decrease the risk of recurrence[19]

X.Other pharmacological approaches:

Nonsurgical decompression:

Nonsurgical methods of colonic decompression in patients with acute colonic pseudo-obstruction consist of colonoscopic decompression with or without placement of a decompression tube and percutaneous decompression.

Colonoscopic decompression:
Colonoscopic decompression is a technically difficult procedure and has a perforation rate of approximately 3 percent[20] It is contraindicated in patients with colonic perforation or peritonitis.

Percutaneous decompression:
Percutaneous cecostomy may be effective for treatment of acute colonic pseudo-obstruction, it is invasive and can be complicated by local infection and bleeding[21-26] In addition, percutaneous decompression has not been directly compared with colonoscopic decompression or surgery. Percutaneous decompression can be accomplished by endoscopically guided insertion of a plastic tube into the left colon or cecum allowing decompression and irrigation. Alternatively, tube placement in the right colon requires a combined endoscopic and radiologic approach (fluoroscopic guidance) [9]

Surgery:
In the absence of a colonic perforation, a surgically placed cecostomy tube or a segmental or subtotal resection with primary anastomosis can be performed to decompress the colon[25]

IV. Case report

A 33 years old female admitted for elective ceaserean section. She developed gaseous abdominal distension with distended bowel loop immediate post op, general surgeon's opinion was sought and advised for x-ray erect abdomen which was found to be normal. She was discharged at request on post op day#2. On post op day#4 she was presented withabdominal distension,breathlessness and tachycardia since one day. Examination showed distended and tense abdomen.On palpation with mild right lower quadrant tenderness. Auscultation of abdomen diminished bowel sounds. There was no evidence of any rigidity and rebound tenderness. Laboratory results showed haemoglobin-12.2g/dl, leukocyte count 15,400 and serum electrocyte within normal range (sodium-143 meq/l, chloride-106 meq/l, potassium-3.5meq/l, bicarbonate-22meq/l,urea-12mg/dl,creatinine-0.6 mg/dl). After obtaining general surgeon's opinion:

USG ABDOMEN showed multiple air fluid level suggestive of dilated bowel loops.
FIG 1. X-RAY erect abdomen showed multiple air fluid level suggestive of small bowel obstruction.

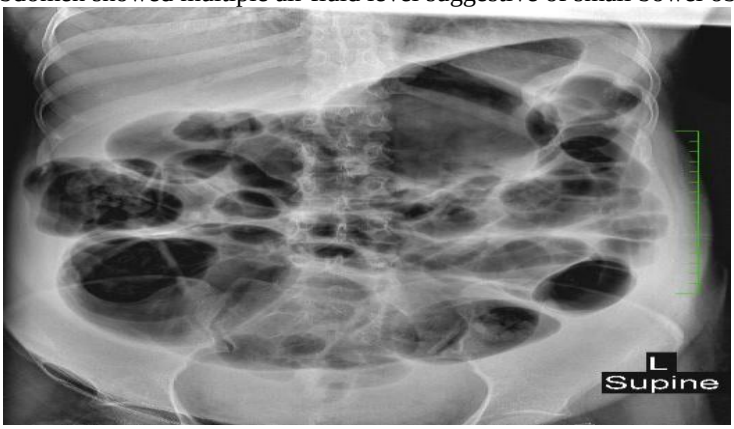
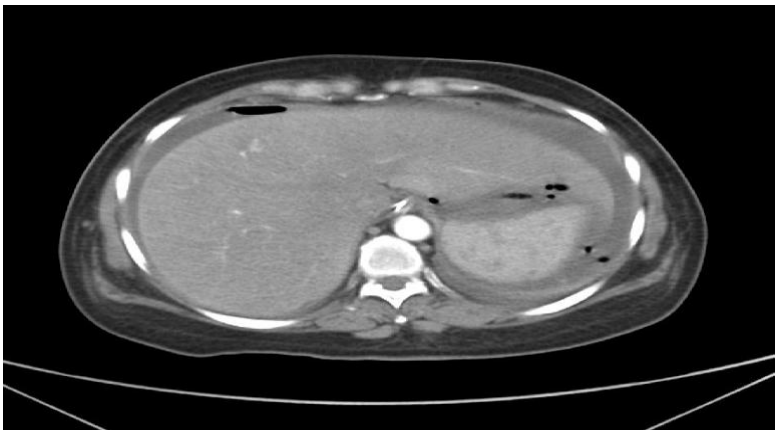
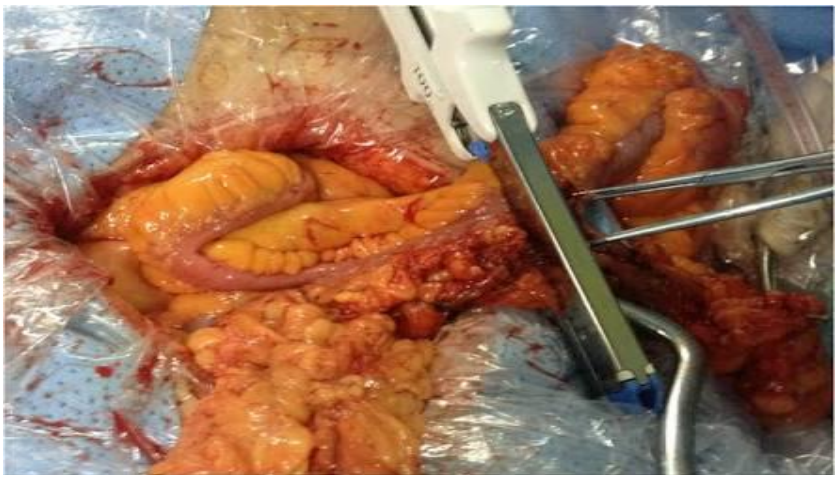
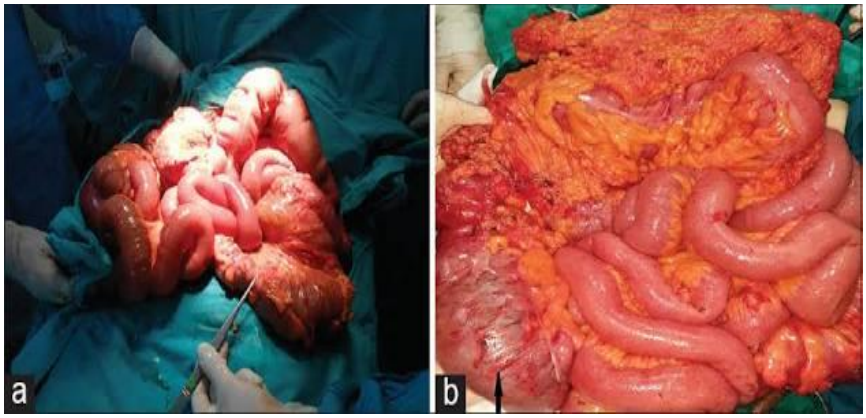


FIG2.Before shifting for CECT-whole abdomen she developed breathlessness, hence intubated and proceeded with CECT-Whole abdomen it showed suspicion of perforation in the hepatic flexure.



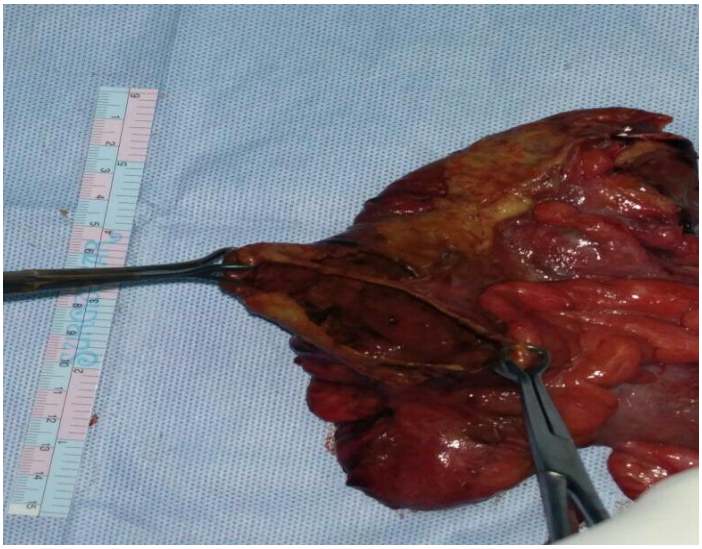
Hence patient was planned for emergency exploratory laparotomy and underwent limited resection of the ascending colon and caecum with ileotransverse anastomosis.





Intra-operative findings:

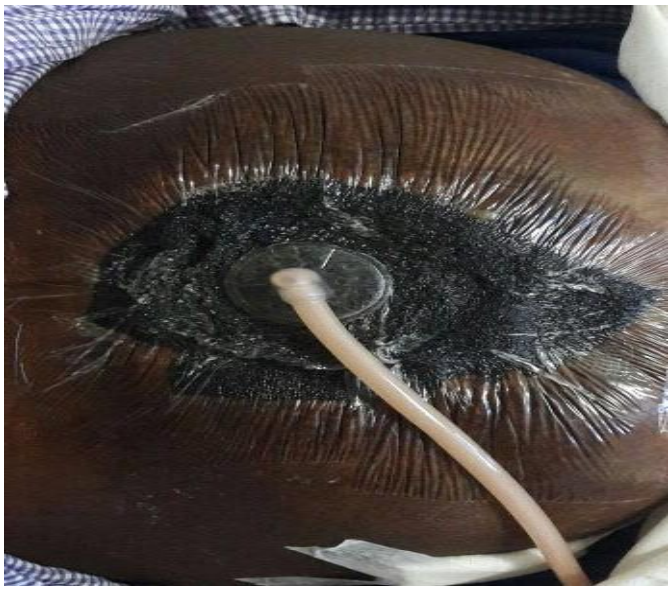
Massive pneumoperitoneum noted. Ceacal perforation of size 6 x 7 cm noted in anti-mesenteric border. There was fecalperitonitis(involving entire peritoneal cavity). Omental caking seen entire abdomen and minimal pus flakes seen adherent to the small bowel loops.Other structures like appendix, liver, gallbladder, duodenum, pancreas, stomach appears to be normal.



Patient was shifted to Intensive care unit.

On POD#0 patient had a drop in saturation and decreased urine output, there was an electrolyte imbalance same corrected. Patient wasextubatedon POD#3.USG thorax showed bilateral minimal pleural effusion,hence pulmonology opinion was sought. One cycle of TPN was given.2-D ECHO was done same showed minimal pulmonary hypertension. Hence cardiology opinion was sought and advised for CT pulmonary angiogram which showed upper lobe sub-segmental pulmonary thrombosis with mild pulmonary artery hypertension. Bilateral lower limb venous Doppler showed normal study.

She was started on anti-coagulants as per cardiologist opinion. She was started on normal diet. In view of extensive discharge with pus from the wound site, all sutures were let out subsequently she developed burst abdomen which was managed conservatively with daily saline dressings and culture specific antibiotics. Surgical gastro opinion was sought and advised for proper wound management with USG abdomen. USG abdomen showed minimal collection in the Morrison's pouch. Wound care manager(Laprostomy bag) dressing was done and wound management with dressing and wash done thrice a day. She was symptomatically improved, hence I.V Antibiotics were stopped and changed to oral antibiotics,she was mobilised and laparotomy wound site was applied with vaccum suction dressingand repeat vaccum dressing was done four days later which showed healthy granulation tissue.Patient was symptomatically improved and hence being discharged with the vaccum suction dressing on POD-30.



XI. Conclusion

Ogilvie's syndrome or acute colonic pseudo-obstruction is a clinical syndrome arising with marked abdominal distension without evidence of mechanical obstruction. Diagnosis is confirmed by abdominal radiology. Prompt treatment is important to avoid the complication of perforated cecum. Treatment should include an initial trial of conservative measures with nasogastric decompression, bowel rest, and correction of electrolytes. Cessation of medications with the potential to exacerbate the condition, such as opioids, is also important.

After a 24- to 48-hour period, if there is no improvement, the patient should have a trial of neostigmine provided there are no contraindications. Use of colonoscopy, decompression tube placement in the ascending colon, and cecostomy should be reserved for patients who do not respond to neostigmine administration. In the presence of peritoneal signs or perforation, surgery is the appropriate first intervention.

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