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Ogilvie's Syndrome – A Post-Operative Emergency in a Case of Carcinoma Rectum

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ABSTRACT

Acute colonic pseudo obstruction or Ogilvie's syndrome is a rare entity that is characterized by acute dilatation of the colon without any mechanical obstruction. It is usually associated with medical disease or surgery and rarely occurs spontaneously. If not diagnosed early, Ogilvie's syndrome may cause bowel ischemia and perforation. Its early diagnosis and prompt treatment are the keystones to avoid any subsequent fatal complications. We report a case of acute intestinal pseudo obstruction causing post-operative wound dehiscence in a patient who underwent colostomy reversal following a Hartman's procedure for a rectal carcinoma. We also review the clinical characteristics, diagnostic methods, and management of Ogilvie's syndrome with reference to literature.

Keywords: Ogilvie's Syndrome, Intestinal Obstruction, Colon cancer.

INTRODUCTION

Acute Colonic Pseudoobstruction (ACPO), Nonobstructive Colonic Dilatation, or Ogilvie's syndrome is an entity that is characterized by acute dilatation of the colon, usually involving caecum and right hemicolon in the absence of any mechanical obstruction (80–90%), abdominal pain (80%), abdominal tenderness (62%), nausea and/or vomiting (60%), constipation (40%), and fever (37%). It is usually associated with an underlying illness, infection, or surgery and rarely occurs spontaneously. Identification of this condition is important due to the increased risk of subsequent bowel ischemia and perforation, particularly with caecal diameter >9 cm, with high mortality rate up to 50%. Here, we report a case of burst abdomen in a patient who underwent colostomy reversal following Hartmans procedure for rectal carcinoma, where exploratory laparotomy was done which showed massively distended large bowel without any mechanical cause of obstruction.

CASE REPORT

A 60-year-old male presented with constipation and he was diagnosed a case of Carcinoma Rectum. He underwent Hartmans procedure with end descending colostomy. He completed 6 cycles adjuvant chemotherapy following the procedure (FOLFOX 4). Patient was doing well in followup and clinical examination and imaging showed no evidence of residual or recurrent disease. The patient was willing to undergo colostomy reversal. He was planned for colostomy closure and anastomosis with the rectal stump under general anaesthesia. The surgery proceeded without any intraoperative complications. There were dense adhesions of bowel with anterior abdominal wall as well as between the bowels. There was no evidence or recurrence of malignant disease.

On the first and second postoperative day [POD], the patient looked well with stable vital signs. Systems review was within normal, and physical examination showed soft and lax abdomen with audible bowel sounds. The third day the patient passed flatus and was started on the liquid diet. The same night, he developed mild abdominal distension but bowel sounds were still audible with stable vital signs.

On the fourth day patient had abdominal distension with hiccups. On examination bowel sounds were present and vitals were stable. We kept the patient on nil per oral with continous ryles tube aspiration. Serum electrolytes were requested and showed mild hypokalemia, which was corrected with potassium infusion and was advised to mobilize the patient.

On the fifth day the patient had progressive abdominal distension on auscultation bowel sounds heard. X ray abdomen shows dilated large bowel. He initially was diagnosed to have paralytic ileus, but his general condition eventually deteriorated

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dramatically. He developed fever, tachycardia and other signs of septicaemia. Serous discharge was draining from the laparotomy site. She was diagnosed as burst abdomen. Patient was immediately taken up for emergency laparotomy.

We found that bowels were massively dilated from caecum to descending colon.

Rectal anastomosis was healthy. There was no hematoma or abscess. We could not find any mechanical cause. Needle puncture of transverse colon was done and air let out. Flatus tube was passed upto 40 cm and fixed. Thorough wash was given and drains kept. Laparotomy wound edges freshened and closed with tension sutures.

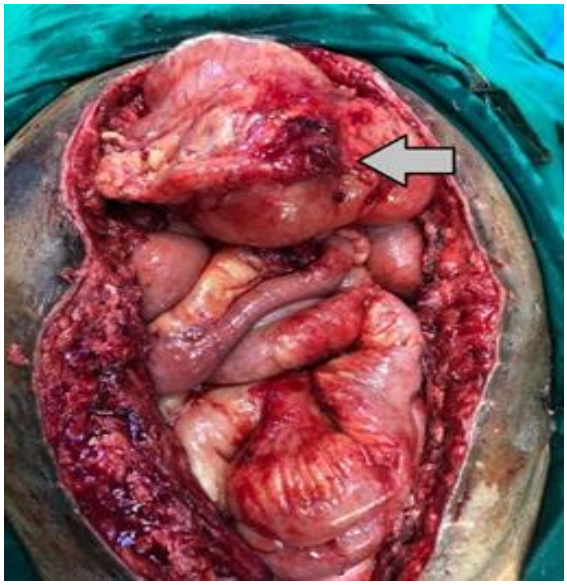

Figure 1: showing the dilated transverse colon with transverse diameter approximately 15 cm

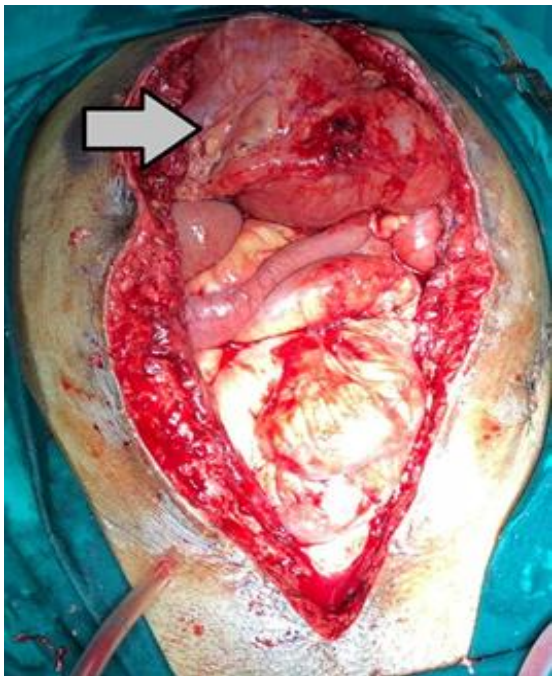

Figure 2: showing collapsed transverse colon after tube decompression of the descending colon

The patient was properly hydrated all through the surgery. The patient received broad-spectrum antimicrobial agents. He was given supportive therapy. Patient passed stools and flatus tube was removed. He was started on oral diet. Drains removed on 7 th day and 9 th day. Patient was discharged on 10 th day. Tension sutures removed on 21 st POD.

DISCUSSION

Table 1: Compares between Ogilvie's syndrome and paralytic ileus.

	Ogilvie's syndrome	Paralytic ileus
Impaired area	Limited to colon	Throughout the gut
Bowel sounds	Hyperactive/high-pitched/absent	Always absent
Nausea & vomiting	Mild and inconstantly present	More common
Passing flatus	Present	Always ceased
Passing stool	Present/diarrhea/obstipation	Always ceased

Ogilvie's syndrome or ACPO was first reported by Sir Ogilvie in 1948.[1] It is described as acute dilatation of the colon usually involving Caecum and right Hemicolon without any existing mechanical obstruction.[2,3] It is a rare condition, yet it can result in dangerous complications with subsequent high mortality rate beyond 50%.[4] It can occur at any age with higher frequency in the sixth decade of life.[5] Its incidence in males is higher than that in females (1.5:1). It has been reported after pregnancy or cesarean section.[6] The condition has been also associated with trauma, severe burns, drugs (narcotic analgesics, antidepressants, corticosteroids, antipsychotic, calcium channel blockers, narcoleptics, and syntocinon), spinal anesthesia, opioid use, alcohol, cardiac failure, respiratory failure, neurological problems (Parkinson's, Multiple Sclerosis, and Alzheimer's), electrolyte imbalance, stress that causes central secretion of corticotrophin-releasing factor (which, in turn, inhibits gut motility), and hormones affecting the smooth muscles and, in rare occasions, may occur spontaneously.[6-9] In our case the following factors might have caused the condition: (1) elderly patient, (2) post-operative surgery, (3) electrolytes imbalance. The exact pathophysiology of the disease is still unclear but it was hypothesized that either the increase in the sympathetic tone or the decrease in the sacral parasympathetic innervations to the colon results in decreased colon motility with subsequent proximal colon dilation which will eventually increase the intraluminal pressure in the proximal colon and caecum, obstructing the caecal capillary circulation and causing subsequent ischemia, gangrene, and perforation.[2,8,10] As the ACPOs have serious complications, timely diagnosis and treatment are critical. Clinical and radiological findings are both needed to confirm the diagnosis of

the syndrome. Typically, it presents within 48 h and up to 12 days postoperatively and can be confused with mechanical obstruction of bowel-like paralytic ileus.[5,11,12]

Xray of the erect abdomen is helpful in most cases for diagnosing Ogilvie's syndrome. Plain abdominal X-ray is the most useful diagnostic modality that reveals gaseous distention in colon, mostly involving caecum and ascending colon, with or without fluid levels seen in small bowel.[10,11] Caecal diameter of 9–12 cm warrants ischemia and subsequent perforation if not managed urgently.[5] Though X-ray is a fundamental diagnostic modality, other modalities like CT scans and water soluble contrast enema are used to confirm the diagnosis and to exclude mechanical obstruction.[11] When X-ray is indeterminate and the correct diagnosis cannot be made, gastrografin enema is desirable to detect bowel distention. However, an exploratory laparotomy in some patients will remain the final option to reach a conclusive diagnosis.[13] In our case, initially we suspected paralytic ileus, yet the final diagnosis of burst abdomen due to Ogilvie's syndrome was reached only after laparotomy. In ACPO, laboratory findings are nondiagnostic. Some electrolyte imbalances like hyponatremia, hypomagnesemia and hypokalemia can be seen in ACPO, but they represent a consequence of the pathological condition rather than its etiologic factor. Similarly, leukocytosis can be present, especially with perforation or bowel ischemia. Hypokalemia and leukocytosis were present in our case. Management for uncomplicated patients is initially conservative with limiting oral intake, active mobilization, cessation of opioids, and correction of electrolytes, and underlying comorbidities should be treated.[14] Intravenous hydration, nasogastric decompression, rectal tube decompression, close clinical monitoring with serial physical examinations, laboratory studies, and abdominal radiological modalities should be done. The most effective pharmacological agent is neostigmine, given intravenously at a dose of 2 mg over 3–5 min and repeated once if required in 2-3 hours.[11] Neostigmine is a reversible anticholinesterase inhibitor that potentiates the effects of the parasympathetic system and improves colonic motility, causing effective colonic decompression up to 88%. Ganglionic blockade with guanethidine followed by cholinergic stimulation with neostigmine can be effective. Neostigmine should not be used in overly distended caecum and, due to its bradycardiac hypotensive effects, it should be given to vitally stable patient with monitored setting.[13] An alternative to neostigmine is erythromycin, amotilin receptor agonist.[13] Other pharmacological agents are naloxone and cisapride.[5] If conservative and medical management, including the second dose of neostigmine, failed, colonoscopic decompression is recommended. It is successful in 68–95% of cases and prevents any ischemia and bowel perforation, yet recurrence is common. Colonoscopic decompression is contraindicated if perforation or peritonitis exists. Surgery is recommended if colonoscopic decompression failed, or progressive clinical deterioration or signs of ischemia and perforation are present, or if caecal diameter is >12 cm. Surgical treatment can be either caecostomy or, in case of ischemic bowel, hemicolectomy with or without primary anastomosis or total abdominal colectomy. The surgical treatment has mortality rate ranging from 30% to 60%. For postoperative pain control, nonsteroidal anti-inflammatory drugs may be considered in place of opioids for high-risk patients. Having similar pain relieving effect as systemic opiates, thoraco lumbar epidural anesthesia can be used to reduce the duration of postoperative ileus. Once the case is suspected (severe abdominal distention, abdominal pain, nausea, and constipations), it is required to obtain initial proper assessment including history (surgeries, caesarian section, infection, and spinal injuries, keeping in mind the previously discussed etiological factors), clinical examination, and laboratory investigations (absence of mechanical obstruction favor the diagnosis of Ogilvie's syndrome or ACPO). Conservative management should be established for 24–48 hours in the Acute Massive Colonic Dilation form of close observation, hydration and correction of electrolyte imbalance, insertion of the nasogastric tube, keeping the patient fasting without using rectal enema or laxatives, cessation of all narcotic medications, and treating underlying etiological factors if any. Assess to rule out the presence of mechanical obstruction and to evaluate for perforation as this will terminate conservative management. In the case of no improvement, when abdominal X-ray shows caecal dilation, colonic distention 10–12 cm, likely normal small bowel, and no mechanical obstruction or no improvement for 3 days, pharmacological decompression with intravenous neostigmine could be started with caution if caecum is significantly dilated and if there is no response, endoscopic colonic decompression can be considered. If pharmacological/ endoscopic decompression failed or signs suggestive of perforation exist, then surgical intervention should be considered in the form of caecostomy, hemicolectomy, and resection of the ischemic or perforated segment of the bowel should be performed. If we have doubt of Ogilvie syndrome, better to insert a flatus type per rectally, it will reduce the intraluminal pressure thereby reducing the fatal complications like burst abdomen in our case, gangrene bowel, perforation and peritonitis.

CONCLUSION

Ogilvie's syndrome is a rare, yet very important clinical condition to surgeons to diagnose and

manage it as early as possible in post-operative patients to avoid any subsequent fatal complications. We recommend precise assessment and close monitoring with conservative management in any suspected case. Reassessment is important to assess whether the disease progresses or regresses. If we have suspect of Ogilvie syndrome or paralytic ileus, we recommend to insert a flatus type per rectally, it will reduce the intraluminal pressure immediately thereby reducing the fatal complications like burst abdomen in our case, gangrene bowel, perforation and peritonitis.

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