

CASE REPORT

Ogilvie's syndrome after rectal prolapse repair and total hemorrhoidectomy: Case report and Discussion

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Key Clinical Message

This case highlights the rare diagnosis of Ogilvie's syndrome after minor surgery in a private hospital where facilities and expertise are generally sparse. It shows the importance of knowledge of the subject, proper assessment, accurate diagnosis, and early input from seniors is crucial to prevent ischemia and perforation of colon that carries high mortality.

Keywords

Colonic pseudoobstruction, hemicolectomy, neostigmine, Ogilvie's syndrome, parasympathetic, rectal prolapse repair.

Introduction

Ogilvie's syndrome is rare type of acute intestinal pseudoobstruction associated with spontaneous massive dilation of cecum and proximal colon in the absence of mechanical obstruction. It was first described by Sir William Ogilvie in 1948. It commonly occurs in number of different settings in hospitalized patients. If inappropriately managed, may cause ischemic necrosis and spontaneous perforation with dire consequences. Early detection and management is the key to reduce morbidity and mortality. Colonic pseudoobstruction is most often seen in postsurgical patients and in medical patients with multiple comorbidities. Exact etiology of this syndrome is still unknown but it is widely believed that an imbalance in the autonomic innervations, either an increase in sympathetic activity or decrease in parasympathetic activity of distal colon is a main contributor of colonic motility impairment. This article describes the case of a 46-year-old White British man with no significant previous medical and surgical history, who developed Ogilvie syndrome after rectal prolapse repair and total hemorrhoidectomy. The clinical characteristics, diagnostic methods, and management of Ogilvie's syndrome are reviewed.

Case Report

A 46-year-old male without any previous significant medical history was initially admitted for elective surgical repair of recta prolapse and total hemorrhoidectomy as a day case. He was doing well postsurgery and was safely discharged from the hospital later that evening with regular analgesics, that is, paracetamol and ibuprofen with lactulose.

He reported back to the hospital next morning with complaints of severe spasmodic pain in the lower abdomen and urinary retention for few hours, he opened his bowels twice after he was discharged from the hospital. On examination lower abdomen was extremely tender on palpation and vital signs were normal except sinus tachycardia. Bladder scan revealed 225 mL of residual urine. Initial impression was Bladder Spasms, therefore he was catheterized and was given tramadol 100 mg IV, buscopan 40 mg IV, and cyclizine 50 mg IV. His symptoms were slightly relieved but still complaining of severe abdominal pain, on reassessment there was a tender palpable mass in right iliac fossa, which was thought to be distended cecum due to air or fecal impaction. To rule out acute intestinal obstruction, urgent erect abdominal X-ray film was requested and routine blood tests were

done. Plain radiograph revealed massive dilatation of large intestine with few air fluid levels but there were no signs of intestinal perforation (Fig. 1) and a provisional diagnosis of Acute colonic pseudoobstruction (Ogilvie's syndrome) was made. Blood tests were unremarkable except borderline raised white cell count.

Treatment was started with nasogastric tube suction, intravenous fluids, regular enemas, and periodic change in position. Over postoperative day 2, the patient's condition did not improve and few doses of intravenous neostigmine were given prior to attempted colonic decompression. A rectal tube was inserted after endoscopic decompression of the colon. Despite these interventions, his condition deteriorated and his lower abdominal pain worsened. Computed tomography (CT) imaging showed stigmata of recent hemorrhoid surgery and gas filled distension of cecum and proximal large bowel with localized pneumatosis coli and small volume of adjacent free fluid, the appearances point toward OS and some degree of colonic ischemia (Fig. 2).

The patient proceeded to emergency laparotomy where a significantly distended colon with evidence of cecal ischemia was found. No mechanical obstruction was found. A right hemicolectomy was performed with seromuscular anastomosis. Postoperative recovery was unremarkable.

Gross examination of specimen showed hemorrhages and ulceration, histopathology revealed 'early necrotizing phlebitis and thrombi formation, ulceration, and

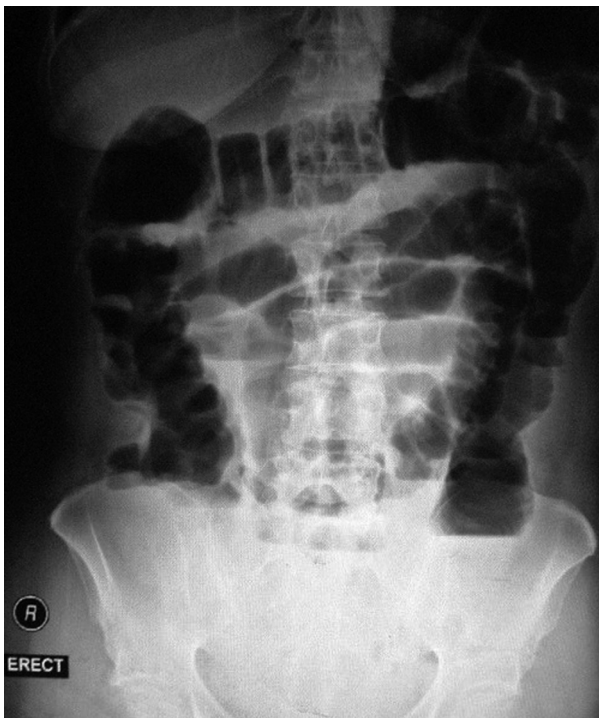


Figure 1. Diffuse colonic dilatation.

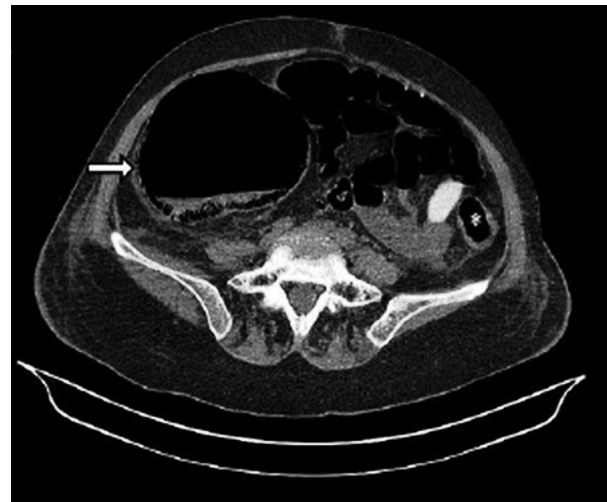


Figure 2. Abdominal CT image showing marked cecal dilatation.

granulation tissue involving lamina propria and submucosa. Few crypt abscesses are seen. Mucosa is spared'.

Discussion

Though uncommon, OS is still a potentially dangerous condition with life threatening complications like colonic ischemia, perforation, and peritonitis. The incidence of cecal perforation is 3–40% and associated mortality rate is as high as 40% [1–3].

The pathophysiology of OS remains to be construed. Several theories exist that suggest malfunction of autonomic nervous system of the large gut. These propositions focus on the increased sympathetic activity or decreased parasympathetic activity or combination of both can cause the phenomenon of OS.

This condition is most commonly associated with recent surgical intervention of any kind, trauma to spinal cord or brain, old age, respiratory failure, metabolic imbalance, electrolytes disorders, malignancy, myocardial infarction, congestive heart failure, pancreatitis, recent neurological event like stroke or subarachnoid hemorrhage, renal insufficiency, hypothyroidism, and medications, for example, narcotics, opioids, anticholinergics, and antidepressants. The studies have documented that 95% of the cases of OS are associated with medical or surgical conditions, rest are idiopathic [1,4,5].

The main clinical features are severe abdominal pain and distension, nausea and vomiting, constipation, and fever. Abdominal tenderness with some degree of guarding and rebound tenderness may be detected. Hypoactive bowel sounds, mass in right iliac fossa, and empty rectum are some other signs that are frequently picked up on physical examination.

Laboratory findings reflect the underlying medical or surgical problems. Significant fever or leukocytosis raises concern for colonic ischemia or perforation. Radiology demonstrates colonic dilation, usually confined to cecum and proximal colon. The maximum upper limit for normal cecum is 9 cm; a diameter of more than 10–12 cm increases the risk of perforation. Varying amounts of small intestinal dilation and air fluid levels can be seen.

Colonic pseudoobstruction should be distinguished from distal mechanical obstruction and toxic megacolon. Other differentials would include acute or chronic megacolon, diverticulitis, Hirschprung's disease, and carcinoma.

Conservative treatment is the first step management with detection and correction of underlying cause. A nasogastric or rectal tube should be placed. Patients should be mobilized or periodically rolled from side to side and to the knee-chest position in an effort to promote expulsion of colonic gas. Enemas may be administered repeatedly if evidence of fecal impaction. Oral laxatives are not recommended and may cause perforation and electrolytes abnormalities. Conservative treatment is successful in over 80% of the cases within 1–2 days. Patients should be continuously monitored and observed for worsening signs and symptoms. Cecal size should be assessed regularly with help of radiographs. Intervention should be considered if there is no improvement or cecal dilation of more than 12 cm. Neostigmine is main pharmacological agent used in treatment of OS, it results in rapid decompression in 75–90% of the patients. Cardiac monitoring during neostigmine infusion is indicated for possible bradycardia that may require atropine administration. Contraindications of neostigmine include bradycardia of less than 60/min, systolic blood pressure lower than 90 mmHg and bronchospasm [6]. Several new cytokinetic agents have also been reported to use in OS like 5-hydroxytryptamin-4 receptor antagonist (e.g., Cisapride) and motilin receptor agonists [7]. Methylnaltrexone (opioid receptor antagonist) and erythromycin (prokinetic agent) have also been documented as therapeutic options.

Decompression by colonoscopy is indicated in patients who fail to respond to neostigmine, the success rate is up to 70% with morbidity only 0.2–2% [8–11]. The recurrence rates after colonoscopic decompression varies from 18% to 65%. Recurrence can be reduced by placing long indwelling compression tubes; still some patients require serial colonoscopic decompressions.

Surgical interventions are the last resort which can be either cecostomy or hemicolectomy with end to end anastomosis. In cases of acute dilatation without ischemia or perforation, tube cecostomy is the preferred option [1,2,4,8,12,13]. This procedure can be performed via open, percutaneous or laproscopic approach. Hemicolectomy is reserved for those patients who develop ischemia

or perforation. All efforts should be made postoperatively to improve splanchnic circulation and prevent complications with fluid resuscitation and prophylactic antibiotics. Complications of surgery include intestinal fistula, abdominal compartment syndrome, wound infection, dehiscence, abdominal sepsis, and incisional hernia.

Prognosis is related to the underlying condition but with aggressive therapy development of perforation is unusual. Mortality rate has been reported to be 14% in medically treated patients and 30% in surgically treated patients [8].

Conclusion

Acute colonic pseudoobstruction (Ogilvie's syndrome) remains uncommon condition with serious complications. The underlying defect in motility of the colon is believed to be due to problem in enteric autonomic nervous system. The key to diagnosis is early recognition of clinical features and requesting radiological images like X-rays and CT scan to aid the diagnosis. Conservative management with NG tube insertion, rectal tubes and intravenous neostigmine (2–2.5 mg) is the mainstay of treatment. If conservative therapy fails or cecal distension on radiology is >12 cm then surgical intervention is required to prevent perforation. Decompression with colonoscopy may be effective.

It is extremely important to anticipate OS in postsurgical patients with acute abdomen. In private hospital where immediate facility of advanced radiology scan and senior input may not be readily available, it is crucial for junior doctors to recognize the condition in initial assessment and involve surgical consultant early.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of written consent is available for review by Editor-in-chief of this journal.

Conflict of Interest

None declared.

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