CLINICAL VIGNETTE

A Classic Case of Acute Colonic Pseudo-Obstruction

(Olgilvie's Syndrome)

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Acute colonic pseudo-obstruction (ACPO), also known as Olgilvie's syndrome, is a massive dilation of the colon with obstructive symptoms, in the absence of mechanical obstruction. ACPO is a rare but possibly life-threatening condition and prompt diagnosis and management are essential to minimize complications. A wide spectrum of illnesses, including major orthopedic surgery, predisposes patients to ACPO. While the exact cause of ACPO is uncertain, it is thought to result from an imbalance of autonomic innervation to the colon. The clinical presentation of ACPO is that of an acute bowel obstruction and abdominal radiography reveals massive colonic dilation. Management includes supportive therapy, minimizing narcotics and anti-cholinergic agents, use of neostigmine, and colonoscopic decompression. Surgery is reserved as a last resort for refractory cases or if serious complications such as ischemia or perforation occur. Early recognition and appropriate management are essential to reduce morbidity and mortality and the occurrence of life-threatening complications. We present a classic case of ACPO that developed in a patient after knee replacement surgery.

Case Report

A 54-year-old male underwent a revision total knee replacement for worsening right knee pain and radiological evidence of a failed right knee arthroplasty. Past medical history was significant for osteoarthritis of the bilateral knees and hips, hypertension, and gout. Postoperatively, the patient was treated with an intravenous hydromorphone PCA (patient controlled analgesia) for pain control for two days and then transitioned to oral opioid analgesics (oxycodone/acetaminophen) as needed. The patient was also treated with oxycodone ER 10 mg morning and night. On postoperative day #3, the patient developed abdominal distension, abdominal discomfort and nausea. There was no diarrhea. On physical examination, the patient was afebrile and had a normal blood pressure and heart rate. Abdominal examination revealed a markedly distended abdomen that was non-tender and tympanic on percussion. High-pitched bowel sounds were audible on auscultation. Laboratory tests including a complete blood count and metabolic panel were normal except for mild postoperative anemia that was expected from surgical blood loss. Abdominal radiographs showed no free air, but colonic dilation with cecum diameter measuring 8 cm and transverse colon measuring 10.5 cm. The symptoms did not improve and a repeat abdominal radiograph the following day showed a cecal diameter of 10.5 cm and transverse colon diameter of 11 cm.

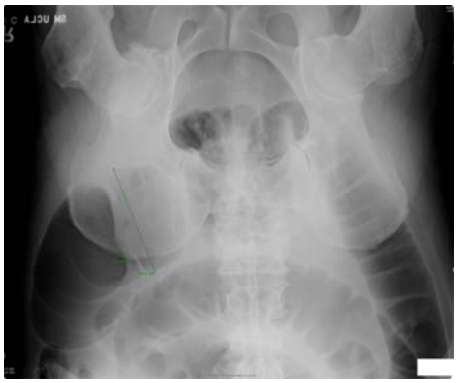

Figure 1

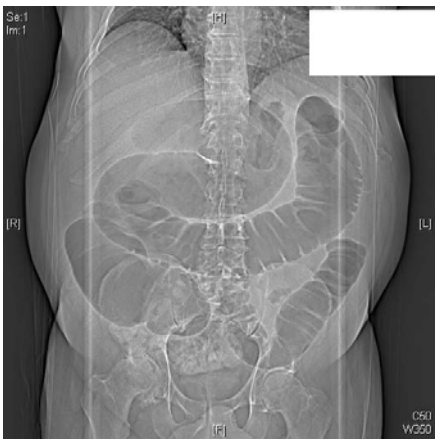

Figure 2

Subsequently, a computed tomography (CT) scan of the abdomen and pelvis showed colonic dilation but no evidence of obstruction.

The patient was diagnosed with acute colonic pseudo-obstruction and was made NPO (nothing by mouth), a nasogastric tube (NGT) was placed to low-intermittent suction and intravenous fluids were started. Gastroenterology consultation was obtained and conservative management was recommended that included NPO, NGT decompression, intravenous fluids, minimizing opiate pain medications, and a rectal tube for decompression. The patient slowly improved over the next two days. He began passing flatus and his abdominal distension improved. Serial abdominal radiographs showed resolution of the previously seen dilated colon.

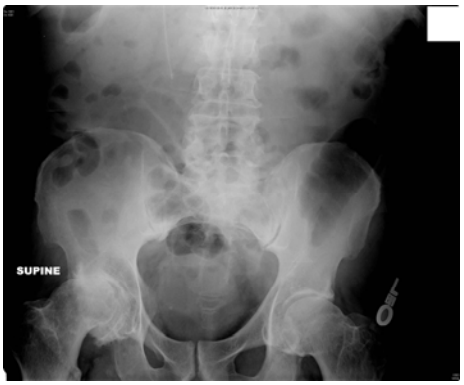

Figure 3

Intravenous neostigmine was considered, but was not necessary as the patient improved rapidly with conservative management alone. The patient was discharged home. Five months later the patient subsequently underwent a left total hip replacement for severe osteoathritis. In an effort to avoid a recurrence of ACPO, polyethylene glycol was started on postoperative day #1 and narcotic use was minimized. The patient had no recurrence of abdominal distension and was discharged on post-operative day #3.

Discussion

Acute colonic pseudo-obstruction (ACPO), or Olgilvie's syndrome, was first described in 1948 by Sir William Heneage Olgilvie, who reported two patients with the sudden onset of abdominal pain, constipation, and colonic dilation associated with malignant infiltration of the celiac plexus destroying prevertebral ganglia[1]. ACPO is characterized by massive colonic dilation with signs and symptoms of colonic obstruction without evidence of mechanic blockage on imaging. ACPO often develops in elderly hospitalized patients with serious underlying medical and surgical conditions. Ischemia and perforation are the feared complications of ACPO and spontaneous perforation has been reported in 3 to 15% of patients with a mortality rate of 50%[2, 3]. The exact prevalence of ACPO is unknown, but has been reported to occur in about 1% of hospitalized patients undergoing orthopedic procedures, including lower limb joint replacement and spinal operations[4]. ACPO is a true gastrointestinal emergency and prompt recognition and appropriate treatment are key to reducing poor outcomes.

The precise mechanisms underlying ACPO remain unknown although it is thought to result from an imbalance of autonomic regulation of colonic motor function[3]. In his initial description of the syndrome, Olgilvie attributed ACPO to sympathetic depravation because he found malignant infiltration of the celiac plexus[1]. Since that time, a better understanding of the autonomic nervous system of the gut has modified this hypothesis. It is now known that the para-sympathetic nervous system increases contractility, whereas the sympathetic nervous system decreases motility. An imbalance of autonomic innervation, produced by a variety of factors, is believed to lead to excessive parasympathetic suppression or sympathetic overstimulation[2,3]. An imbalance in either could lead to ACPO. This hypothesis is supported by the fact that neostigmine, an acetylcholinesterase inhibitor which acutely increases parasympathetic stimulation, produces rapid colonic decompression in most patients[5].

ACPO typically develops in elderly hospitalized patient with a wide spectrum significant underlying medical or surgical conditions (**Table 1**) [6].

Table 1.

Selected conditions associated with ACPO*

A. Trauma (non-operative)
    a. Mechanical ventilation
    b. Spinal cord or pelvic trauma
B. Infection
    a. Sepsis
    b. Pneumonia
    c. Acute cholecystitis
C. Cardiac
    a. Myocardial infarcion or stroke
    b. CHF
D. Postsurgical
    a. Ceasarean section
    b. Gynecological or pelvic surgery
E. Neurologic
    a. Parkinson's disease
    b. Alzheimer's disease
    c. Multiple sclerosis
F. Orthopedic Surgery
    a. Hip replacement or knee replacement
    b. Spinal surgery
G. Drug Induced
    a. Opiates
    b. Antidepressants
    c. Parkinson's medications
H. Other serious medical conditions

***** adapted from Vanek VW, Al-Salti M. Acute pseudo-obstruction of the colon (Olgilvie's syndrome). An analysis of 400 cases. Dis Colon Rectum. 1986;29:203-10.

Large retrospective evaluations have shown that the most common predisposing factors include operative trauma (23%), non-operative trauma (11%), infections (10%), and cardiac disease (10%). A variety of medical and surgical conditions were also associated with ACPO. Cesarean section and hip surgery are the most common surgical procedures associated with ACPO[6]. In a retrospective series of orthopedic surgical patients, those who developed ACPO compared to controls had lower sodium levels; higher urea nitrogen levels and remained in the hospital longer[4]. In another retrospective analysis, more than half of the patients were receiving narcotics and more than 66% had electrolyte abnormalities[2]. While the exact mechanism by which these conditions suppress colonic motility is unknown, it is clear that multiple metabolic, pharmacologic, or traumatic factors can alter the autonomic regulation of the colon leading to ACPO. In many patients who develop ACPO, several of these factors coexist making the risk even higher.

Clinical features of ACPO include abdominal distension, abdominal pain (80%), nausea and/or vomiting (60%) and frequently failure to pass flatus and stools[3,6]. Examination reveals a distended, tympanic, non-tender abdomen with high pitched 'tinkling' bowel sounds. Marked abdominal tenderness and systemic features such as fever or tachycardia are signs of possible complications such as perforation. Diagnosis relies on accurate clinical assessment and is confirmed by plain abdominal radiographs, which often show massive colonic dilatation. The cecum and right colon show the most marked distension[7]. The differential diagnosis for acute colonic distension in hospitalized patients includes mechanical obstruction, toxic megacolon secondary to severe *Clostridium difficile* infection, and ACPO. The appropriate urgent evaluation includes assessing for signs of peritonitis or perforation and excluding mechanical obstruction and *C. difficile*[2, 3]. Fever, marked abdominal tenderness, and leukocytosis are more common in patients with ischemia or perforation. In addition to plain abdominal radiographs, some experts recommend a water-soluble contrast enema or CT scan to differentiate mechanic obstruction from pseudo-obstruction and assess for free peritoneal air and pneumatosis of the bowel wall[3, 7]. Others suggest that these studies can be reserved for those patients in which air is not seen throughout all colonic segments on plain radiographs[2]. In all cases, close clinical observation and consultation with a gastroenterologist is warranted.

Treatment options for ACPO include supportive measures, pharmacologic therapy, colonoscopic decompression, and surgery. The dilemma is often whether to treat the patient with conservative measures and close observation verses proceeding with medical or endoscopic decompression. The severity of the clinical picture and perceived risk of imminent ischemia and perforation should guide treatment[2, 3]. The outcome of ACPO is influenced by several factors, with the severity of the underlying illness having the greatest influence on patient outcome (**Table 2**)[6].

Table 2.
Factors influencing outcome in ACPO*

A. Severity of underlying illness
B. Age
C. Cecal diameter (> 12 cm)
D. Duration of distension (> 6 days)
E. Bowel ischemia or perforation

***** adapted from Vanek VW, Al-Salti M. Acute pseudo-obstruction of the colon (Olgilvie's syndrome). An analysis of 400 cases. Dis Colon Rectum. 1986;29:203-10.

Patient age, cecal diameter (>12 cm), duration of distension (> 6 days), and whether or not bowel

ischemia or perforation are present also have been shown to have a significant effect on outcome. A review of over 400 cases of ACPO found no perforations at a cecal diameter < 12 cm, a 7% rate for diameters 12-14 cm, and a 23% rate for > 14 cm. Also, worse outcomes were seen in patients who had cecal distension for > 6 days[6]. Furthermore, the mortality is high (~40-50%) when bowel ischemia or perforation is present. All of these factors should influence the decision to intervene with medical therapy, colonoscopy or surgery[2, 3].

Conservative therapy is the preferred initial treatment of ACPO and should be started immediately in all patients. Conservative management is successful in the majority of patients (>85%) at a mean of 3 days[8, 9]. Supportive therapy includes nothing by mouth (NPO), intravenous fluids, correcting electrolyte imbalances (especially hypokalemia and hypomagnesemia), nasogastric suction, and insertion of a rectal tube attached to gravity drainage[2, 3, 9]. All drugs that delay gut motility (such as opiates, anticholinergics, and calcium-channel blockers) should be discontinued or minimized. Laxatives, especially osmotic compounds such as lactulose, should be avoided as they can increase gas production. Mobilization and ambulation should be encouraged. Close observation with serial physical exams and daily abdominal radiographs is warranted[8]. Conservative management is successful as the primary treatment in the majority of patients. The decision to intervene with medical management, colonoscopy, or surgery depends on the patient's clinical status. As stated above, prolonged distension and cecal diameter > 12 cm increase the risk of perforation. In the absence of signs of peritonitis or perforation, medical therapy with neostigmine is typically considered in patients who are not improving after 24-48 hours of supportive therapy[2, 8].

Medically therapy with neostigmine is the treatment of choice if supportive therapy is unsuccessful. Neostigmine is a reversible acetylcholinesterase inhibitor that stimulates muscarinic receptors and enhances colonic motor activity. Neostigmine has a rapid onset of action (1-20 min) and a short duration. Numerous reports have detailed the efficacy of neostigmine, with initial response rates of 60-90%. Three randomized trials have documented to effectiveness of neostigmine[5, 10, 11].

The landmark therapeutic trial for ACPO showed that intravenous (IV) neostigmine was a very effective treatment that resulted in rapid colonic decompression in the majority of patients[5]. In this trial, neostigmine was used in patients with ACPO with a cecal diameter > 10 cm and no response to 24 hours of conservative treatment. Exclusion criteria included suspected ischemia or perforation, renal failure with Cr > 3 mg/dL, baseline heart rate < 60 beats/minute or systolic blood pressure < 90 mm Hg, and severe active bronchospasm. Patients either received 2 mg of IV neostigmine or placebo (saline). A clinical response, defined as a prompt passage of gas and reduction of abdominal distension, was seen in 10 or 11 patients (91%) who received neostigmine compared to 0 of 10 receiving saline. The median time to response was 4 minutes (range, 3-30). Patients not responding within 3 hours to initial infusion were eligible for open label neostigmine. Eight patients not responding to initial infusion (7 saline, 1 neostigmine) were given open label neostigmine, and all had prompt decompression. In total, 17 of 18 patients (94%) who received neostigmine had a clinical response. Recurrence of ACPO happened in 2 patients. The most common side effects after receiving neostigmine were mild abdominal cramping, excessive salivation, sweating, nausea and vomiting, or transient bradycardia or hypotension. Symptomatic bradycardia requiring atropine occurred in 2/19 patients[5]. Other studies have also shown rapid decompression in the majority of patients and a very low recurrence rate[9, 10, 11]. A second dose of neostigmine should be considered if there is a partial or no response to the first trial as it is frequently effective[5].

Neostigmine must be administered cautiously in a monitored setting. The patient should be kept supine with continuous cardiac monitoring. Vital signs and continuous physician assessment is necessary for 15-30 minutes after administration. Atropine should be available at the bedside to be given intravenously for symptomatic bradycardia if needed. Contraindications to the use of neostigmine include mechanical obstruction, ischemia or perforation, pregnancy, recent myocardial infarction or un-controlled arrhythmias, severe active broncho-spasm, and renal insufficiency (creatinine > 3 mg/dL) [3]. The cost is low at around $3 per 2 mg ampule[2]. Neostigmine appears to be an effective, safe, and inexpensive method of decompression in ACPO making it the treatment of choice for patients not responding to conservative therapy. After initial resolution of ACPO, administration of polyethylene glycol solution (PEG) has been shown to decrease the recurrence rate in a small randomized trial and should be considered[12].

Colonoscopic decompression is the procedure of choice for those patients that have not responded to supportive treatment, and who have contraindications

to or fail neostigmine therapy. There is no well-defined standard of care regarding the use of colonoscopy in ACPO. This procedure should only be performed by experienced endoscopists with adequate equipment as it is technically difficult, has a greater risk of complications, and often requires the placement of a colonic decompression tube in the right colon to improve the therapeutic benefit[2]. The overall success rate of colonoscopic decompression in patients with ACPO in various retrospective series is around 80%[3]. Surgical management is reserved for patients with signs of colonic ischemia or perforation or who fail pharmacologic and endoscopic efforts. Patients with ACPO often have severe underlying medical conditions and surgical intervention is associated with significant morbidity and mortality (up to 30%)[6].

In conclusion, ACPO develops in patients in association with a variety of underlying predisposing medical and surgical conditions and is thought to result from an imbalance of autonomic control of the colon. Prompt evaluation to exclude mechanical obstruction and assess for signs of ischemia or perforation is warranted. Management includes supportive measures and selective use of neostigmine and colonoscopic decompression in those with severe and prolonged colonic dilation. Neostigmine remains the only therapy that has been proven to be beneficial in randomized clinical trials. Patient outcome is often determined by the severity of the predisposing illness. Other factors such as maximal cecal diameter, duration of colonic distension, and bowel viability are also important predictors of outcome. Early re-cognition and prompt management are crucial to minimizing morbidity and mortality in patents with ACPO.

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