Acute colonic pseudo-obstruction (Ogilvie's syndrome)

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INTRODUCTION

Pseudo-obstruction is characterized by signs and symptoms of a mechanical obstruction of the small or large bowel in the absence of a mechanical cause. Pseudo-obstruction may be acute or chronic and is characterized by the presence of dilation of the bowel on imaging. Other causes of colonic distension including toxic megacolon, mechanical obstruction, and chronic intestinal pseudo-obstruction are discussed in detail, separately. (See "Overview of mechanical colorectal obstruction" and "Toxic megacolon" and "Chronic intestinal pseudo-obstruction".)

DEFINITION

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 institutionalized patients in association with a severe illness or after surgery and in conjunction with a metabolic imbalance or administration of culprit medication (table 1) [1-5]. In a large, retrospective series that included 400 patients with acute colonic pseudo-obstruction, the most common predisposing conditions were nonoperative trauma, infection, and cardiac disease, each of which were associated with 10 percent of cases [1]. In this series, cesarean section and hip surgery were the most common surgical procedures associated with acute colonic pseudo-obstruction. In a systematic review of 125, postpartum cases of acute colonic pseudo-obstruction, 62 (92 percent) occurred following caesarian section for varying indications including preeclampsia, multiple pregnancy, and antepartum hemorrhage/placenta previa [6]. No specific risk factors could be identified for postpartum acute colonic pseudo-obstruction. In this review, 43 percent of patients had intestinal perforation or impending perforation, and 47 percent of patients required laparotomy, whereas conservative management was successful in 50 percent of cases. All patients with a cecal diameter of >12 cm perforated as compared to 3 of 17 patients with a diameter of <9 cm. Most perforations were diagnosed between day 3 and day 5 following the caesarian section.

Acute colonic pseudo-obstruction is also well-documented after kidney transplantation, and possible contributing factors include obesity, cumulative dose of prednisone received, and mycophenolate mofetil [7].

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]. However, cases have been reported in children [8]. 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 [4,9]. In surgical patients, symptoms usually present at an average of five days postoperatively. From a national hospital admissions database, the calculated incidence of acute colonic pseudo-obstruction is approximately 100 cases out of 100,000 inpatient admissions per year [10].

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 [1,11]. However, there is no proposed mechanism to explain colonic dilation in those patients without obvious involvement of the parasympathetic nerves.

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 [12]. The duration of dilation is probably more important than the absolute diameter of the colon [13,14].

Rare cases have been reported in association with atrophic visceral myopathy with an extremely thin colonic wall, atrophic circular, and longitudinal muscularis propria without inflammation or fibrosis, and unaffected ganglion cells and myenteric plexus [15]. The cause of the smooth muscle atrophy was unclear and the only potential association was with prior hypothyroidism [15,16]. Other rare associations of acute colonic pseudo-obstruction are herpes zoster infection and phaeochromocytoma [17,18].

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. Approximately 80 percent of patients have associated 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 [1,19]. In rare cases, abdominal distention can cause dyspnea [1,19].

On physical examination, the abdomen is tympanitic to percussion, but bowel sounds are present in almost 90 percent of patients [1]. Approximately 65 percent of patients with a viable colon have mild abdominal tenderness on physical examination. However, the presence of fever, marked abdominal tenderness, and the presence of peritoneal signs (eg, guarding, rigidity, rebound tenderness) are suggestive of colonic ischemia or perforation or their impending development. (See "Colonic ischemia", section on 'Acute colonic ischemia' and "Overview of gastrointestinal tract perforation", section on 'Presentations'.)

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. Colonoscopy should not be used to make the diagnosis of acute intestinal pseudo-obstruction, as insufflation of air may increase the colonic dilatation.

Evaluation — The goal of the evaluation is to establish the diagnosis of acute intestinal pseudo-obstruction, to assess for complications, and to rule out other causes of colonic distension.

Laboratory tests — We perform a complete blood count, electrolytes, and serum lactate levels. Given the rare cases associated with hypothyroidism and the fact it is eminently treatable, we also screen for hypothyroidism with a serum thyroid stimulating hormone. 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 *Clostridioides* (formerly *Clostridium*) *difficile* toxin. (See "Clostridioides (formerly Clostridium) difficile infection in adults: Clinical manifestations and diagnosis", section on 'Laboratory assays' and "Diagnosis of and screening for hypothyroidism in nonpregnant adults", section on 'Diagnosis' and 'Pathogenesis' above.)

There are no pathognomonic laboratory findings in patients with acute pseudo-obstruction. Laboratory evaluation may reveal leukocytosis and metabolic

abnormalities. If present, leukocytosis is usually due to the patient's underlying disease or impending perforation and is not associated with an uncomplicated pseudo-obstruction. Metabolic abnormalities, especially hypokalemia, hypocalcemia, and hypomagnesemia, are common, occurring in more than 50 percent of patients [1,19-23]. It is recognized that a variant of recurrent acute or chronic colonic pseudo-obstruction with megacolon may be associated with a secretory diarrhea that results in fecal potassium loss and severe hypokalemia and is less likely to respond to usual treatments such as decompression or neostigmine [22,23]. Correction of hypokalemia is essential [23]. (See 'Supportive care' below.)

Imaging — We perform an abdominal computed tomography (CT) scan to establish the diagnosis of acute intestinal pseudo-obstruction and to rule out other causes of intestinal obstruction. In the absence of access to a CT scan, a contrast enema using a water-soluble contrast can be used to establish the diagnosis, provided that there is no evidence of peritonitis on physical examination.

- **Abdominal CT scan** – On abdominal CT scan in patients with acute colonic pseudo-obstruction, there is proximal colonic dilatation, often with an intermediate transitional zone at or adjacent to the splenic flexure and a characteristically absent structural cause of colonic obstruction. Occasionally, dilation may extend to the rectum. In addition to differentiating acute colonic pseudo-obstruction from a mechanical obstruction, abdominal CT also has the advantage of demonstrating other intra-abdominal pathology that may have precipitated its development (eg, retroperitoneal hemorrhage).

- **Contrast enema** – Contrast enema with a water-soluble contrast demonstrates colonic dilation in the absence of a mechanical obstruction. In addition, it can potentially cause an osmotically driven evacuation of the colon and relieve the pseudo-obstruction [24]. However, contrast enemas can increase intracolonic pressure and cause perforation. Contrast enemas should not be performed in patients with possible peritonitis. (See "Toxic megacolon", section on 'Diagnosis'.)

- **Abdominal radiographs** – Plain and upright abdominal radiographs show a dilated colon, often from the cecum to the splenic flexure and occasionally to the rectum; haustral markings are normal (image 1). As abdominal radiographic findings are not specific for acute colonic pseudo-obstruction, the role of

abdominal radiographs is limited to monitoring the colonic diameter once the diagnosis of acute colonic pseudo-obstruction has been established by abdominal CT or contrast enema. (See 'Approach to management' below.)

DIFFERENTIAL DIAGNOSIS

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

Mechanical obstruction — Patients with mechanical obstruction frequently complain of crampy abdominal pain; however, lack of pain, especially in the older adult or postoperative patient receiving opiates, does not exclude that diagnosis. A "cut-off sign" (lack of gas in the distal colon or rectum) or small bowel air-fluid levels on abdominal radiographs can also be seen in patients with acute colonic pseudo-obstruction. However, a mechanical obstruction can be differentiated from acute colonic pseudo-obstruction by visualization of an obstructing lesion on abdominal CT or contrast enema. (See 'Imaging' above.)

Toxic megacolon — Unlike patients with acute intestinal pseudo-obstruction, patients with toxic megacolon have evidence of significant systemic toxicity with fever, tachycardia, altered sensorium, and abdominal pain. Patients with toxic megacolon often have a history of severe bloody diarrhea or other signs or symptoms of chronic inflammatory bowel disease. On abdominal upright and plain films, there is evidence of colonic distension, but the normal colonic haustral pattern is either absent or severely disturbed with "thumbprinting" due to the presence of submucosal edema, or thickening of the colonic wall (image 2). (See "Toxic megacolon", section on 'Clinical manifestations'.)

MANAGEMENT

The goal of management is to decompress the colon in order to minimize the risk of colonic perforation and ischemia, which are associated with a high mortality [1]. There are few controlled trials comparing treatments of acute colonic pseudo-obstruction. Our recommendations are largely consistent with a 2020 guideline for the management of acute colonic pseudo-obstruction by the American Society for

Gastrointestinal Endoscopy and Clinical Practice Guideline from the American Society of Colon and Rectal Surgeons (algorithm 1) [25,26].

Approach to management

- Given 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 [13].

- 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 (algorithm 1).

  In patients with cecal diameter >12 cm and in patients who have failed 48 to 72 hours of conservative therapy, we use pharmacologic therapy with neostigmine. A meta-analysis of four studies that included 127 patients revealed that 65 were treated with neostigmine. Resolution of acute colonic pseudo-obstruction was significantly higher with only one dose of neostigmine as compared with placebo (89 versus 15 percent, number needed to treat 1, 95% CI 1-2) [27,28]. A comparative study showed that bolus dosing or continuous infusion of neostigmine were equally safe and effective [29]. Although continuous infusion neostigmine was associated with greater bowel diameter reduction at 24 hours in one retrospective study, these findings require validation [30].

  In patients who fail or who have contraindications to neostigmine, we perform colonoscopic decompression. In patients whose acute colonic pseudo-obstruction may be precipitated by opiates, we administer subcutaneous methylnaltrexone, prior to percutaneous or surgical decompression [31]. 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 [32].

  There is some evidence that performing colonoscopic decompression before neostigmine treatment may actually shorten hospital stay, though this advantage

of decompression first may pertain mostly to younger patients without cardiac disease [33].

Percutaneous colostomy should be reserved for patients who fail endoscopic decompression and are not surgical candidates. We reserve surgical decompression for patients who fail endoscopic and pharmacologic therapy or have evidence of perforation or peritonitis [26].

Supportive care — Supportive care with removal of precipitants is the first step in the management of patients with acute colonic pseudo-obstruction [34]. Supportive care can be continued for 48 to 72 hours in the absence of significant pain, extreme (>12 cm) colonic distension, or signs of peritonitis. Success rates of conservative management range from 20 to 92 percent with a median time to resolution of 1.6 days in one series [14]. Supportive care includes the following:

- Treatment of the underlying disease (eg, infection, congestive heart failure).
- Discontinuation of medications that can decrease colonic motility (eg, opiates, calcium channel blockers, medications with anticholinergic side effects) and avoidance of laxatives [1].
- Patients should be given nothing by mouth. Intravenous fluids should be administered to maintain normovolemia and electrolyte abnormalities corrected [22,23].
- Decompression of the gastrointestinal tract by placement of a nasogastric tube attached to intermittent suction and a rectal tube attached to gravity drainage. While gentle tap water enemas can be administered, their use should be limited given the risk of perforation [1,26].
- Patients should be encouraged to ambulate, if possible. Patients should be placed in a prone position with the hips elevated on a pillow or the knee chest position with the hips held high. These positions should be alternated with right and left lateral decubitus positions each hour.

Neostigmine — Neostigmine, an acetylcholinesterase inhibitor, is indicated in patients with acute colonic pseudo-obstruction and cecal diameter >12 cm or in patients who fail 48 to 72 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 [26,35]. Patients should be kept supine on a bedpan, and atropine should be available at the bedside to treat bradycardia associated with neostigmine [35]. In patients with successful colonic decompression, we administer polyethylene glycol electrolyte balanced solution to decrease the risk of recurrence [26,36]. Patients with a partial response or recurrence after initial resolution should be treated with repeat administration of neostigmine [35]. It is the author's practice not to repeat dosing within 24 hours.

  In the author's experience, lower doses (1.5 mg) may also be effective and may possibly decrease abdominal cramping, nausea, and vomiting. A further reduction in dosage to 0.5 or 1 mg is indicated in patients with new-onset heart block, a history of second-degree heart block, or following bowel resection with primary anastomosis. Alternative dosing strategies reported in the literature are subcutaneous and continuous infusions [30,37]. However, additional studies are needed to directly compare their efficacy.

  Some side effects (particularly bradycardia and bronchoconstriction) might be reduced by coadministration of glycopyrrolate, an anticholinergic agent that has limited activity on the muscarinic receptors of the colon [38].

- **Adverse effects and contraindications** – Adverse effects of neostigmine include bradycardia, hypotension, asystole, seizures, restlessness, tremor, bronchoconstriction, nausea, vomiting, salivation, diarrhea, sweating, and abdominal cramps. Relative contraindications to the use of neostigmine include recent myocardial infarction, acidosis, asthma, bradycardia, peptic ulcer disease, and therapy with beta-blockers. The use of neostigmine in pregnancy, although reported, has not been well studied [39].

- **Efficacy** – Approximately 88 to 94 percent of patients have decompression of colonic distension with neostigmine. In a randomized trial, 21 patients with acute colonic pseudo-obstruction were assigned to treatment with neostigmine or placebo. Prompt decompression was observed in 11 patients (91 percent) who received neostigmine compared with none receiving placebo. In patients treated

with neostigmine, the median time to response was four minutes (range 3 to 30 minutes) [40]. Recurrence of colonic distension occurred in two patients (11 percent).

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 [41]. It is contraindicated in patients with colonic perforation or peritonitis.

An oral bowel preparation should not be administered prior to colonoscopy, due to the risk of aspiration in the presence of pseudo-obstruction. We do not administer enemas prior to the colonoscopy, as there is little stool passed following their administration because of the dilatation and lack of propulsion in the colon and the risk of colonic perforation. We place a decompression tube with the aid of a guidewire at the time of colonoscopy as this may reduce the need for repeated colonoscopic decompression. The guidewire is passed through the channel of the colonoscope after reaching the distal transverse colon. Air is suctioned from the colon and the wire left in place as the colonoscope is gently removed. The decompression tube (customized with several extra side holes, if necessary) is then passed over the guidewire and left in the transverse colon. To minimize air inflation, the whole colon should not be examined and the guidewire should not be delivered into the cecum. The decompression tube should be placed to gravity drainage and flushed every four to six hours.

The efficacy of colonic decompression has not been established in randomized trials. In case series, colonoscopic decompression alone is definitive therapy in less than 50 percent of patients, but, with concurrent placement of a decompression tube, success rates of 88 percent have been reported [14,19,42,43].

Transanal decompression — Colonic decompression may also be achieved via fluoroscopy-guided trans-anal tube placement and was successful in one center study in 70 percent of 22 cases with clinical benefit in 50 percent [44]. The availability of local

expertise should be considered when deciding on colonoscopic or transanal colonic decompression.

Percutaneous decompression — While percutaneous cecostomy may be effective for treatment of acute colonic pseudo-obstruction, it is invasive and can be complicated by local infection and bleeding [45-48]. 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) [12].

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 [49]. In the patients with a colonic perforation, a total colectomy, ileostomy, and Hartmann procedure are performed in order to retain the option of future ileorectal anastomosis. The Hartmann procedure involves resection of the diseased colon, an end-colostomy, and creation of a rectal stump; this is followed by colostomy closure three months later.

Other — Other agents have been evaluated in patients with acute intestinal pseudo-obstruction, but evidence to support their use is limited. In case reports, patients with acute intestinal pseudo-obstruction have been treated with erythromycin (250 mg IV every eight hours for three days or orally 250 mg four times daily for 10 days), but the responses to treatment have been inconsistent, with only gradual improvement over 12 to 24 hours of therapy [50,51].

In one case report of acute colonic pseudo-obstruction in the setting of opioid use that was refractory to neostigmine, administration of subcutaneous methylnaltrexone (12 mg dose) resulted in successful decompression [31]. However, large, prospective studies are needed to determine the role of methylnaltrexone in the treatment of patients with opioid-associated acute colonic pseudo-obstruction.

Decompression of the colon with percutaneous endoscopic colostomy of the cecum (PEC-cecum) is an alternative to surgical decompression [26]. PEC-cecum tubes can be placed by a combined endoscopic and radiologic approach. However, the procedure is

invasive, has a high risk of complications, and has not been compared to other methods of decompression.

PROGNOSIS

The prognosis in patients with acute intestinal pseudo-obstruction depends on the presence of complications. Colonic ischemia and perforation are the two main complications of acute intestinal pseudo-obstruction, which develop in approximately 3 to 15 percent of patients. The mortality rate in acute intestinal pseudo-obstruction in the absence of complications is approximately 15 percent with early appropriate management as compared with 36 to 44 percent in patients with a perforated or ischemic bowel [1,35,52].

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Acute colonic pseudo-obstruction (Ogilvie's syndrome)".)

SUMMARY AND RECOMMENDATIONS

- 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. Acute colonic pseudo-obstruction usually involves the cecum and right hemicolon, although occasionally colonic dilation extends to the rectum. (See 'Definition' above.)
- Acute colonic pseudo-obstruction usually occurs in hospitalized or institutionalized patients in association with a severe illness or after surgery and in conjunction with a metabolic imbalance or medication administration. (See 'Etiology' above.)
- 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. Patients may also

have nausea, vomiting, abdominal pain, constipation, and, paradoxically, diarrhea. Plain and upright abdominal radiographs show a dilated colon, often from the cecum to the splenic flexure and occasionally to the rectum; haustral markings are normal. (See 'Clinical manifestations' above.)

- 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 (eg, abdominal computed tomography [CT] scan or contrast enema) that helps to exclude a mechanical obstruction. Colonoscopy should not be used to make the diagnosis of acute intestinal pseudo-obstruction, as insufflation of air may increase the colonic dilatation. (See 'Diagnosis' above and 'Differential diagnosis' above.)

- Given 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.

- Our recommendations for management are largely consistent with a guideline by the American Society for Gastrointestinal Endoscopy and the Clinical Practice Guideline from the American Society of Colon and Rectal Surgeons (algorithm 1). (See 'Approach to management' above.)

    - Initial management of acute colonic pseudo-obstruction consists of conservative therapy in patients without significant abdominal pain or signs of peritonitis and those who have one or more potential factors that are reversible. Conservative therapy can be continued for approximately 48 to 72 hours, in the absence of significant abdominal pain or extreme colonic dilatation (>12 cm). (See 'Supportive care' above.)

    - We suggest pharmacologic therapy with neostigmine in patients at risk for perforation and those who failed conservative therapy (**Grade 2B**). In patients with cecal diameter >12 cm or failure of 48 to 72 hours of conservative therapy, we administer neostigmine (2 mg IV). We administer a repeat dose of

neostigmine in patients with a partial response or recurrence after initial resolution. (See 'Neostigmine' above.)

- We suggest colonoscopic decompression in patients who fail or who have contraindications to neostigmine (**Grade 2C**).
- We suggest surgical decompression (with cecostomy if possible or colectomy) for patients who fail endoscopic and pharmacologic therapy and for those in whom exploration, lavage, or drainage of the peritoneal cavity is indicated for management of a colonic perforation (**Grade 2C**). (See 'Approach to management' above.)

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Topic 2531 Version 16.0

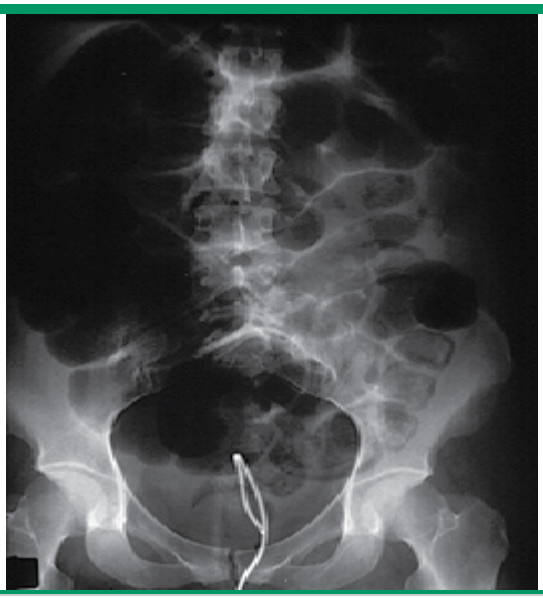
GRAPHICS

Common clinical conditions associated with Ogilvie's syndrome

Category	Examples
Medications	Opioids, anti-cholinergics, alpha-2-adrenergic agonists, anti-psychotics, Ca^{++} channel blockers, cytotoxics, dopaminergics, epidural anesthesia
Trauma and orthopedic surgery	Fractures, hip and spine surgery
Obstetric and gynecological	Pelvic surgery especially involving spinal anesthesia; cesarian section; vaginal (normal or instrumental) delivery
Cardiothoracic surgery or disease	Cardiac surgery including transplantation; myocardial infarction, heart failure, pneumonia
Neurological diseases	Parkinsonism, stroke, dementia
Retroperitoneal diseases	Malignancy, hemorrhage
Metabolic imbalance	K^{+}, Ca^{++}, Mg^{++} imbalance; hypothyrodism
Infection	Herpes zoster

Graphic 66068 Version 3.0

Acute colonic pseudo-obstruction

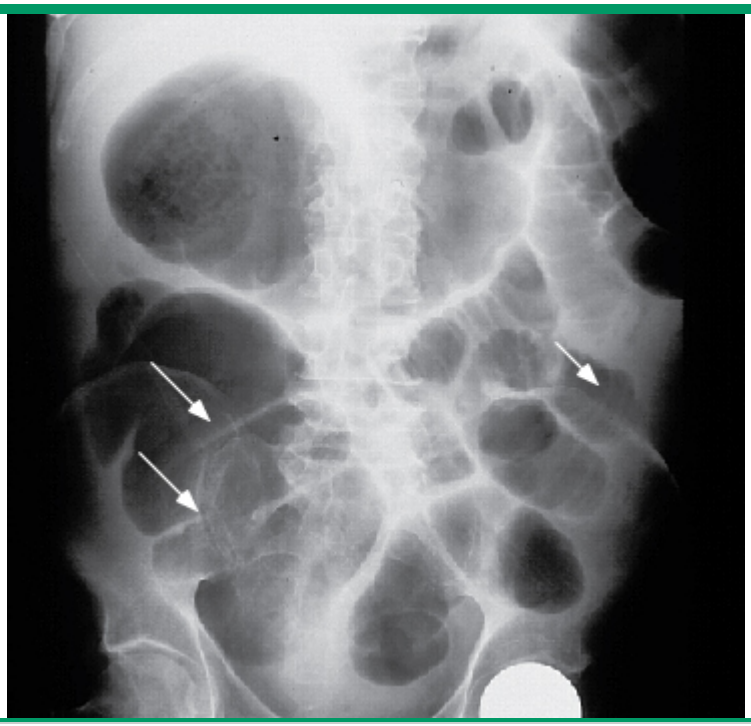


Plain abdominal radiograph in a patient with septic shock and leukemia reveals a massively dilated and air filled right and transverse colon. Haustral markings are normal.

Courtesy of Michael Camilleri, MD.

Graphic 78963 Version 3.0

Toxic megacolon in *Clostridioides* (formerly *Clostridium*) *difficile*

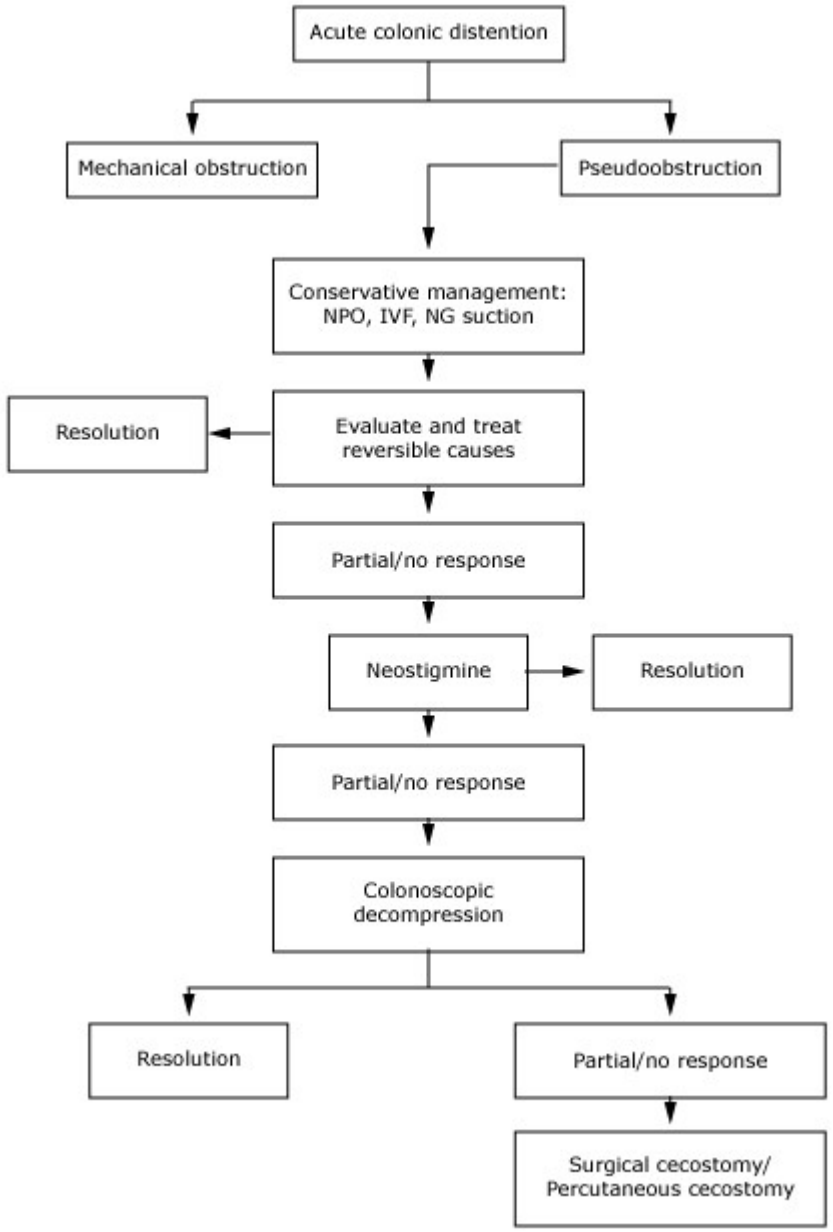


Plain film of the abdomen from a patient with toxic megacolon associated with *Clostridioides* (formerly *Clostridium*) *difficile* infection. The large and small intestines are grossly dilated. Dilatation of the small bowel, which has the thin transverse folds of the valvulae conniventes (short arrow), is seen best in the left lower quadrant. Large bowel dilatation occupies most of the right lower quadrant and has characteristic thick haustral markings that do not extend across the entire lumen (long arrows).

Courtesy of J Thomas Lamont, MD.

Graphic 59217 Version 7.0

Acute colonic dilation



NPO: nothing given by mouth; IVF: intravenous fluids; NG: nasogastric.

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Contributor Disclosures

Michael Camilleri, MD Equity Ownership/Stock Options: Dignify Therapeutics [Spinal cord injury]; Enterin [Constipation in Parkinson disease]. Patent Holder: Capsule for colonic transit by scintigraphy; 13C-mannitol for permeability measurement; Obesity-metabolomics to identify different phenotypes. Grant/Research/Clinical Trial Support: Takeda [Gastroparesis]; Novartis

[Bile acid diarrhea]; Allergan [Bile acid malabsorption]; Vanda [Gastroparesis]; ILSI North America [Permeability]; Arena [Visceral pain in GI disorders]. Consultant/Advisory Boards: Ironwood [Gastroparesis]; Allergan [Diabetic gastroparesis]; AstraZeneca [Naloxegol and OIC]; ElobixAB [Bile acid inhibitor for constipation]; Takeda [Clinical trials]; AlfaSigma [Gastroparesis]; Bio-kier [Obesity, diabetes]; Shionogi [Opioid-induced constipation]; AEON Pharma [Gastroparesis]; Check-Cap [Capsule to measure GI and colonic motility]; Protagonist Therapeutics [IBS-diarrhea]; Arena [Visceral pain in GI disorders]; Kallyope [Obesity]; QED Therapeutics [Diarrhea]; Biokier [Diabetes]; OrbiMed [IBS-constipation]; Salix [Over-the-counter therapy for GI disorders]; Rose Pharma [IBS]; Allakos [Eosinophilic and mast cells GI disorders]; Ironwood [IBS]; EA Pharma [Gastroparesis and IBS]. **Nicholas J Talley, MD, PhD** Employment: University of Newcastle [PVC Global Research]; John Hunter Hospital [Senior Staff Specialist in gastroenterology]; Medical Journal of Australia [Editor-In-Chief]. Patent Holder: Biomarkers of irritable bowel syndrome [Irritable bowel syndrome]; Licensing Questionnaires [Mayo Clinic Talley Bowel Disease Questionnaire - Mayo Dysphagia Questionnaire (Nepean Dyspepsia Index)]; Nestec European Patent [Application 127353589; Nanotechnology]; Singapore 'Provisional' Patent [NTU Ref TD/129/17 "Microbiota Modulation Of BDNF Tissue Repair Pathway"]. Grant/Research/Clinical Trial Support: Commonwealth Labs [IBS (diagnostic blood test]); VN National Science Challenge [Research funding for IBS]. Consultant/Advisory Boards: Adelphi Values [Functional dyspepsia (Patient-reported outcome measures)]; Viscera Labs [Bile acid sequestrant]; twoXAR [IBS]; Anatara Lifesciences [IBS, IBD (Dietary supplement)]; Avrio [Gastroenterology (OTC drugs)]; Censa [Diabetic gastroparesis epidemiology]; Sanofi [Probiotic product (Bacillus clausii)]; Pfizer [IBS (OTC drugs in gastroenterology)]; Planet Innovation [Gastroenterology (Gas capsule)]; Takeda [Gastroparesis (TAK906)]; Allakos [Gastric eosinophilic disease (AK002)]; Progenity Inc, San Diego [SIBO (Intestinal capsule device)]; IM HealthScience [IBS, FD (Peppermint oil)]. Other Financial Interest: IM Health Science [Medicine, clinical skills]. **Shilpa Grover, MD, MPH, AGAF** Nothing to disclose

Contributor disclosures are reviewed for conflicts of interest by the editorial group. When found, these are addressed by vetting through a multi-level review process, and through requirements for references to be provided to support the content. Appropriately referenced content is required of all authors and must conform to UpToDate standards of evidence.

Conflict of interest policy