Acute colonic pseudo-obstruction (Ogilvie's syndrome)

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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. 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 "Large bowel obstruction" and "Toxic megacolon" and "Chronic intestinal pseudo-obstruction: Etiology, clinical manifestations, and diagnosis".)

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 [2]. Acute colonic pseudo-obstruction is a rare complication of surgery, occurring in 0.06 percent of patients after cardiac surgery and 0.7 to 1.3 percent of patients with burns and after orthopedic surgery [3,4]. In surgical patients, symptoms usually present at an average of five days postoperatively. From a national hospital admissions

database, the estimated incidence of acute colonic pseudo-obstruction is approximately 100 cases out of 100,000 inpatient admissions per year [5].

ETIOLOGY AND PATHOGENESIS

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) [1,3,6-8]. 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 cesarean section for varying indications including preeclampsia, multiple pregnancy, and antepartum hemorrhage/placenta previa [9]. No specific risk factors could be identified for postpartum acute colonic pseudo-obstruction. 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 [10]. 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 [11]. The cause of the smooth muscle atrophy was unclear, and the only potential association was with prior hypothyroidism [11,12]. Other rare associations of acute colonic pseudo-obstruction are herpes zoster infection and pheochromocytoma [13,14].

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 [1,15]. However, there is no proposed mechanism to explain colonic dilation in those patients without obvious involvement of the parasympathetic nerves.

CLINICAL MANIFESTATIONS

Clinical presentation — 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 [1,16]. In rare cases, abdominal distention can cause dyspnea [1,16]. 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 [17,18].

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'.)

Complications — 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 [19]. The duration of dilation is probably more important than the absolute diameter of the colon [20,21].

DIAGNOSTIC APPROACH

Evaluation — 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 goal of the evaluation is to establish the diagnosis of acute intestinal pseudo-obstruction, to assess for complications (ischemia, perforation, peritonitis), 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 difficile* toxin. (See "*Clostridioides difficile* infection in adults: Clinical manifestations and diagnosis", section on 'Diagnostic laboratory assays' and "Diagnosis of and

screening for hypothyroidism in nonpregnant adults", section on 'Diagnosis' and 'Etiology 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 [1,16-18,22,23]. (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. While contrast enema can potentially cause an osmotically driven evacuation of the colon and relieve the pseudo-obstruction, they can increase intracolonic pressure and cause perforation [24]. Contrast enemas should not be performed in patients with possible peritonitis. (See "Toxic megacolon", section on 'Diagnosis'.)

Diagnosis — The diagnosis is established by abdominal CT scan, which demonstrates 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. Colonoscopy should not be used to make the diagnosis of acute intestinal pseudo-obstruction as insufflation of air may increase the colonic dilatation.

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, and altered sensorium, as well as 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. (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. There are few controlled trials comparing treatments of acute colonic pseudo-obstruction. Our recommendations are largely consistent with guidelines for the management of acute colonic pseudo-obstruction by the American Society for Gastrointestinal Endoscopy and the American Society of Colon and Rectal Surgeons (algorithm 1) [25-27].

Patients without ischemia, peritonitis, perforation

Cecal diameter <12 cm, mild to moderate abdominal pain — 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).

Supportive care — Supportive care with removal of precipitants is the first step in the management of patients with acute colonic pseudo-obstruction [28]. 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 and intravenous fluids to maintain normovolemia and correct electrolyte abnormalities [17,18].

- Decompression of the gastrointestinal tract by placement of a nasogastric tube attached to intermittent suction. Gentle tap water enemas can be administered, but their use should be limited given the risk of perforation [1,26].

- Encourage patients 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.

Supportive care can be continued for 72 hours in the absence of significant pain, extreme (>12 cm) colonic distension, or signs of peritonitis. Success rates of conservative management alone range from 70 to 90 percent with a mean time to resolution of three days in one series [1,21].

Monitoring — Given the risk of colonic ischemia and perforation, we monitor patients with acute colonic pseudo-obstruction with the following [20]:

- Serial physical examination every 12 to 24 hours.
- Plain abdominal radiographs every 12 to 24 hours to evaluate the colonic diameter.
- Laboratory tests every 12 to 24 hours, including a complete blood count and electrolytes.

Cecal diameter > 12 cm, severe abdominal pain, or failure of conservative management

Neostigmine

Initial treatment

- **Indications and contraindications** – 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. In patients with cecal diameter >12 cm and in patients who have failed 72 hours of conservative therapy, we use pharmacologic therapy with neostigmine. 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 [29].

- **Dose and administration** – Neostigmine (2 to 5 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 [26,30]. Patients should be kept supine on a bedpan, and atropine should be available at the bedside to treat bradycardia associated with neostigmine [30].

  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 [31]. In the author's experience, lower doses (1.5 mg) may also be effective and may be associated with less 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.

  Subcutaneous administration and continuous infusion of neostigmine may be alternatives to bolus dosing, but further studies are needed [32,33]. In one retrospective study of 75 patients, of whom 38 were treated with a continuous infusion of neostigmine, continuous infusion was associated with greater bowel diameter reduction at 24 hours, but was also associated with an increased number of bradycardic events [32].

- **Complications** – Adverse effects of neostigmine include bradycardia, hypotension, asystole, seizures, restlessness, tremor, bronchoconstriction, nausea, vomiting, salivation, diarrhea, sweating, and abdominal cramps.

- **Efficacy** – In a meta-analysis of four randomized trials that included 127 patients with acute colonic pseudo-obstruction, of which 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) [34,35]. Recurrence rate of acute colonic pseudo-obstruction after an initial response was 0 to 31 percent, with an overall long-term response of 69 to 100 percent.

  Most patients respond quickly to therapy. 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) [36]. Recurrence of colonic distension occurred in two patients (11 percent). Male sex, younger age, postsurgical status, and having electrolyte imbalance are risk factors for nonresponse to neostigmine [37].

Additional management based on response — Response to neostigmine can be assessed clinically and radiographically with a reduction in discomfort, abdominal girth, and colon diameter on abdominal radiographs.

- **Resolution** – In patients with successful colonic decompression, we administer oral polyethylene glycol daily to decrease the risk of recurrence in accordance with the literature [26,38]; the dose tested in a randomized placebo-controlled clinical trial was 29.5 g polyethylene glycol (PEG) 3350 (PEG3350); the commercially available PEG3350 preparation provides 17 g in a capful dissolved in 240 mL water; therefore, one can administer just under 2 capfuls in 500 mL water orally if tolerated, or by nasogastric tube.

- **Non-response, partial response, or recurrence** – In patients who fail an initial dose of neostigmine, or have a partial response, we administer a second dose of neostigmine after 24 hours before proceeding to colonic decompression. A second dose of neostigmine has been associated with clinical response in 40 to 100 percent of patients [27,30,38].

Colonoscopic decompression

- **Indications and contraindications** – We perform colonic decompression in patients with acute colonic pseudo-obstruction who have failed neostigmine or who have contraindications to neostigmine [26,30]. The role of adjunctive colonoscopic decompression with supportive therapy and neostigmine in symptomatic patients and patients with proximal colonic dilation has not been established [39].

  Colonic ischemia, perforation, or peritonitis are contraindications to colonoscopic decompression and require surgical management. (See 'Surgical management' below.)

- **Technique**

  - Colonoscopic decompression should be performed by an experienced endoscopist using water infusion and minimal to no insufflation of the colon. Carbon dioxide should be used rather than air.

  - Use of narcotics for procedural sedation should be avoided as they can potentiate colonic atony.

  - The colon should be unprepped. 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 they are often ineffective because of the dilatation and lack of propulsion in the colon and because they carry a risk of colonic perforation.

- An attempt should be made to reach at least the distal transverse colon, after which extensive suctioning of air is recommended.

- 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 and allows for colonic administration of polyethylene glycol (PEG) laxative (PEG3350 17 g in 240 mL water) [38]. In order to place a decompression tube, a 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 decompression tube can be retained for 24 hours after significant reduction of abdominal distension or consistent passage of flatus in order to ensure resolution rather than having to replace the tube if there is recurrence of the acute colonic pseudo-obstruction.

- **Complications** – Colonoscopic decompression is a technically difficult procedure and has a perforation rate of approximately 2 percent and a 1 percent risk of mortality [40].

- **Efficacy** – The use of colonoscopic decompression has only been evaluated in patients who have failed conservative treatment and has not been directly compared with neostigmine in randomized trials [41,42]. 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 [16,21,43,44].

Patients with refractory symptoms, ischemia, perforation, or peritonitis

Surgical management — We reserve surgical management for patients with colonic ischemia, perforation, or peritonitis and those that are refractory to medical management [26]. While one study proposed a colon diameter ≥11 cm to be predictive of a lack of therapeutic response to endoscopic treatment and an indication for surgery, these results have not been replicated [45,46].

For patients with acute colonic pseudo-obstruction who fail colonoscopic decompression and pharmacologic therapy with neostigmine, surgical management consists of stoma creation with or without resection [27]. Patients with perforation or peritonitis require urgent exploration, lavage, or drainage of the peritoneal cavity. The need for and extent of bowel resection, as well

as the type of stoma, are dictated by patient anatomy and clinical conditions. (See "Overview of surgical ostomy for fecal diversion".)

Other approaches — Other approaches have been evaluated in patients with acute colonic pseudo-obstruction, but evidence to support their use is limited.

- Erythromycin – In case reports, patients with acute intestinal pseudo-obstruction have been treated with erythromycin (250 mg intravenously 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 [47,48].

- **Percutaneous colostomy of the cecum** – 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) [19,26]. 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 [49-52]. In addition, percutaneous decompression has not been directly compared with colonoscopic decompression or surgery.

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 [1,30,53].

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

- **Epidemiology** – 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 'Epidemiology' above.)

- **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 medications (table 1). (See 'Etiology and pathogenesis' above.)

- **Clinical manifestations** – Patients usually present with abdominal distension that 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. Complications include 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. (See 'Clinical manifestations' above and 'Complications' above.)

- **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 is established by abdominal computed tomography [CT] scan, which demonstrates 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. Colonoscopy should not be used to make the diagnosis of acute intestinal pseudo-obstruction as insufflation of air may increase the colonic dilatation. (See 'Diagnostic approach' above.)

- **Monitoring** – 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. (See 'Monitoring' above.)

- **Cecal diameter <12 cm and mild to moderate abdominal pain** – Initial management of acute colonic pseudo-obstruction consists of conservative therapy in patients without

significant abdominal pain or signs of peritonitis. Conservative therapy can be continued for approximately 72 hours in the absence of significant abdominal pain or extreme colonic dilatation (>12 cm) (algorithm 1). (See 'Supportive care' above.)

- **Cecal diameter >12 cm, severe abdominal pain, or failure of conservative management**

  - In patients at risk for perforation (cecal diameter >12 cm) and those who have failed conservative therapy, we suggest pharmacologic therapy with neostigmine (**Grade 2B**). We administer an initial neostigmine dose (2 mg intravenously over three to five minutes) with cardiac monitoring and administer a second dose of neostigmine (2 mg intravenously over three to five minutes) after 24 hours in patients with a partial response or recurrence after initial resolution. (See 'Neostigmine' above.)

  - We reserve colonoscopic decompression in patients who fail or who have contraindications to neostigmine. (See 'Colonoscopic decompression' above.)

- **Refractory symptoms, ischemia, perforation, or peritonitis** – We perform surgical decompression (stoma creation with or without resection) for patients with acute colonic pseudo-obstruction who fail colonoscopic decompression and pharmacologic therapy with neostigmine. Patients with perforation or peritonitis require urgent exploration, lavage, or drainage of the peritoneal cavity. (See 'Patients with refractory symptoms, ischemia, perforation, or peritonitis' above.)

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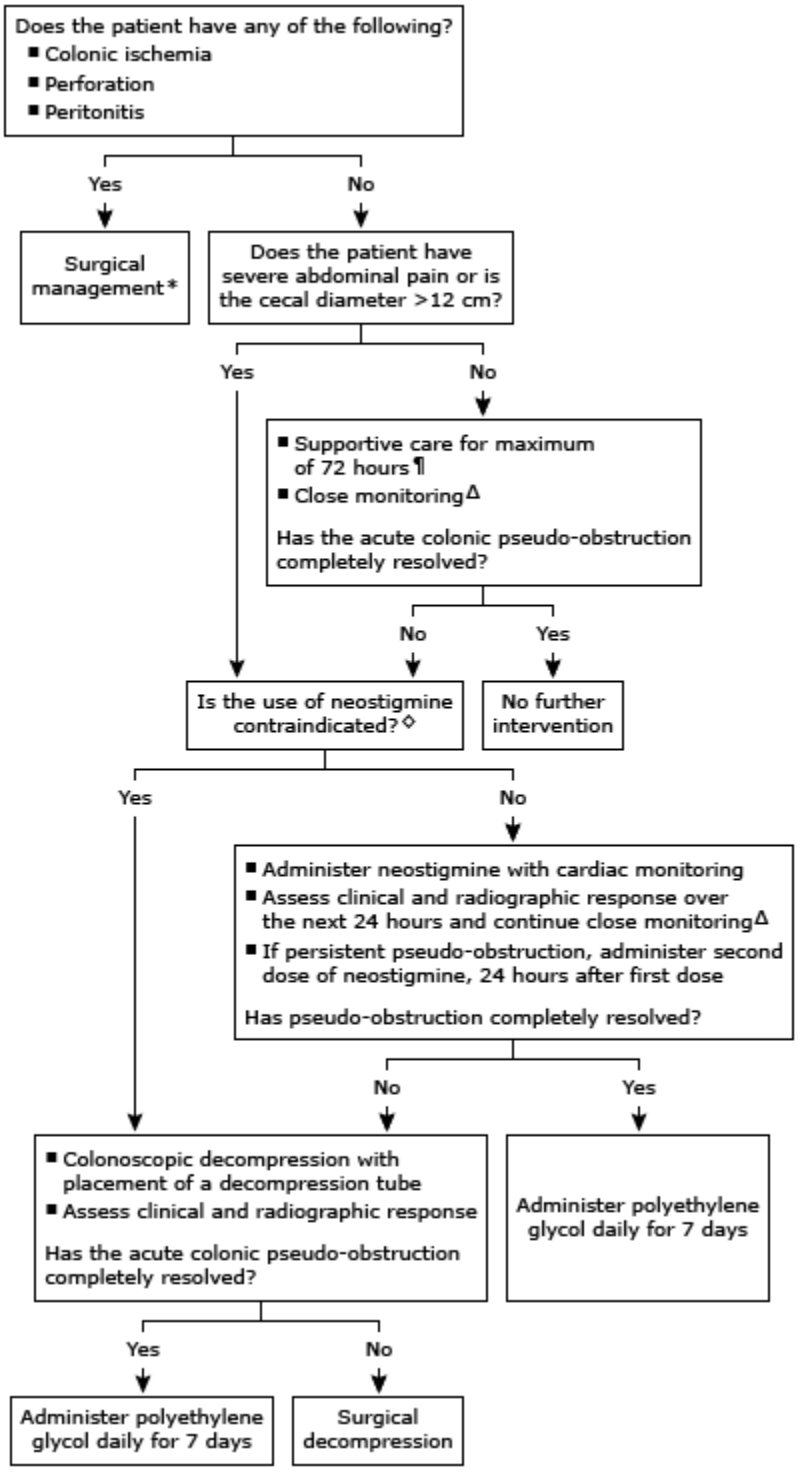
GRAPHICS

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

Approach to the management of the adult patient with acute colonic pseudo-obstruction (Ogilvie syndrome)



* Patients with colonic ischemia, perforation, or peritonitis require urgent exploration, lavage, or drainage of the peritoneal cavity. The need for and extent of bowel resection, as well as the type of stoma, are dictated by patient anatomy and clinical presentation.

¶ Supportive care includes administering nothing by mouth, nasogastric decompression, intravenous fluid and electrolyte replacement, and discontinuation of medications that can adversely affect colonic motility (eg, narcotics and anticholinergic agents).

Δ Monitoring includes serial physical examination, plain abdominal radiographs to evaluate the colonic diameter, and laboratory tests for complete blood count and electrolytes every 12 to 24 hours.

◇ Relative contraindications to the use of neostigmine include recent myocardial infarction, acidosis, asthma, bradycardia, peptic ulcer disease, and therapy with beta-blockers.

Graphic 138695 Version 1.0

Contributor Disclosures

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