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Acute Colonic Pseudo-Obstruction (Oglivies Syndrome): Understanding Risk Factors, Diagnosis and Treatment

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Abstract

Acute colonic pseudo-obstruction (ACPO), also known as Ogilvie syndrome is a potentially fatal condition leading to an acute massive dilation of the colon without mechanical obstruction. It usually occurs in hospitalized or institutionalized patients with serious underlying medical and surgical conditions. The pathophysiology underlying ACPO is mainly related an imbalance in colonic autonomic regulation of colonic motor function, multiple risk factors have been identified. Early recognition and appropriate management are essential to reduce the occurrence of life-threatening complications like ischemia and perforation, which can result in a high mortality rate reaching 40%. In this review, the incidence and the etiology of psudoobstruction, the clinical presentation and the diagnosis is discussed. A practical approach is described to guide management of a critically ill patient who develops colonic psudoobstruction.

Introduction

Acute colonic pseudo-obstruction (ACPO) was described first by Sir Heneage in 1948 [1]. It is a syndrome characterized by massive dilation of the colon with symptoms, signs and radiographic appearance of acute large bowel obstruction without a mechanical cause [2]. A number of underlying causes have been described that predispose to ACPO [3,4], these include systemic or neurological illnesses, recent non-abdominal surgery and trauma. The clinical presentation of ACPO is nonspecific which can result in delay in the diagnosis, it is essential to rule out any mechanical obstruction or other causes of toxic megacolon before considering the diagnosis of ACPO. The goal of management in these patients is to decompress the colon in order to minimize the risk of colonic perforation and ischemia, which are associated with a high mortality [5]. Appropriate management includes supportive therapy and selective use of neostigmine, in case of failure of medical treatment, the next option is to proceed with colonoscopy for decompression. Surgical intervention is reserved for those with peritonitis or perforation.

Epidemiology

The exact prevalence of acute colonic pseudo-obstruction is unknown. The highest prevalence is observed in late middle age (mean age of 60 years), with a slight male predominance (60%) [6]. It occurs most often in hospitalized or institutionalized patients with serious underlying medical and surgical conditions. ACPO occurs in about 1 per cent of patients undergoing orthopaedic procedures like lower limb joint replacement and spinal operations [7]. It affects 0·3 per cent of patients with severe burns [6].

Acute colonic pseudo obstruction is a serious condition with considerable clinical and social impact. Published data and reviews [8] clearly show that, because of multiple co-morbidities, delayed diagnosis and inappropriate treatment, it is responsiblefor considerable morbidity, with a mortality rate of 25–31 per cent overall and 40–50 per cent when the condition complicated with ischemia or perforation [5,9-11].

Etiology

Pathogenesis

The pathogenesis of ACPO is not completely understood but likely results from an alteration in the autonomic regulation of colonic motor function [12]. The parasympathetic nervous system increases contractility, whereas the sympathetic nerves decrease motility. An imbalance in autonomic innervation, produced by a variety of factors, leads to excessive parasympathetic suppression or sympathetic stimulation. The result is colonic atony and pseudo-obstruction [3].

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Table 1: Common clinical conditions associated with Ogilvie's syndrome.

Trauma
Major surgery (orthopedic, spine, pelvic, obstetric, abdominal)
Infection (pneumonia, sepsis)
Cardiac disease (acute myocardial infarction, heart failure)
Neurological condition (Parkinson's disease, spinal cord injury, multiple sclerosis, Alzheimer's disease, dementia, stroke)
Retroperitoneal pathology (malignancy, bleeding)
Metabolic imbalance (electrolyte imbalance, renalfailure, hepatic failure)
Medication (narcotics, anti-Parkinson agents, anticholinergics, antipsychotics calcium channel blockers, , epidural analgesics)
Miscellaneous Major burns/trauma, severe sepsis, idiopathic

Predisposing factors

Multiple metabolic, pharmacologic, or traumatic factors appear to alter the autonomic regulation of colonic function resulting in pseudo-obstruction (Table 1), the vast majority of patients had multiple predisposing factors or clinical conditions [5,13-16]. In a large, retrospective series that included 400 patients with acute colonic pseudo-obstruction, the most common predisposing conditions were nonoperative trauma, infection, cardiac disease, and surgical procedure each of which were associated with approximately 10 percent of cases [5], Cesarean section and hipsurgery were the most common surgical procedures.

Clinical Presentation

Acute colonic pseudo-obstruction is characterized by abdominal distension, which is the most relevant clinical finding and present in virtually all patients [5], the distention usually develops over three to seven days but can occur as rapidly as over a 24 to 48-hour period. Other symptoms include fever, nausea and/or vomiting (60%), constipation and, paradoxically, diarrhea have also been reported in approximately 50 and 40 percent of patients. Patients may presents with dyspnea secondary to the abdominal distention can cause [1,5,7,13-23].

On examination, the abdomen is tympanic and bowel sounds are typically high-pitched 'tinkling', but may be reduced or absent. Interestingly, it is always difficult to distinguishbetween patients with perforation or ischemia and those with uncomplicated distension. With the exception of peritoneal signs and fever, there is a similar cohort of symptoms among patients who have perforated or ischemic bowel and those with Ogilvie's syndrome [24]. Therefore, a high index of suspicion and use of diagnostic studies are important early in the evaluation of massive colonic distension. In surgical patients, symptoms and signs develop at a mean of 5 days post-operatively

Diagnosis

The diagnosis of ACPO is suggested by the clinical presentation and confirmed by plain abdominal radiographs, which show varying degrees of colonic dilatation (Figure 1). The right colon and caecum show the most marked distention, and 'cutoffs' at the splenic flexure or descending colon are common.

This distribution of colonic dilatation may be caused by the different origins of the proximal and distal parasympathetic nerve supply to the colon. Air fluid levels and dilatation can also be seen in the small bowel. Abdominal radiographs should be assessed for the presence of pneumoperitoneum and pneumatosis. The differential diagnosis of acute colonic distention in hospitalized or institutionalized patients includes mechanical obstruction, toxic

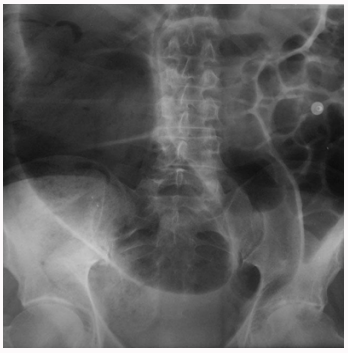

Figure 1: Abdominal radiograph showing diffuse colonic distension, most prominent in right colon.

megacolon caused by severe Clostridium difficile infection, and ACPO. A water-soluble contrast enema or computed tomography (CT) should be performed to differentiate mechanical obstruction from pseudo-obstruction. No direct comparison has been made between these two imaging modalities in terms of diagnostic accuracy, but both are excellent at differentiating mechanical obstruction from pseudo-obstruction. Contrast enema has a sensitivity of 96% and specificity of 98% [25]; CT with intravenous contrast has sensitivity and specificity both of 91% [26]. CT has the additional advantages of allowing more accurate measurement of bowel diameter and a better appraisal of the condition of the mucosa, both in terms of detection of coexisting inflammation and of viability. Ischaemic changes may show as wall thickening, submucosaloedema and, with advancing necrosis, intramural gas. Adjacent fat stranding is much less pronounced with ischaemia than with inflammation andmay help differentiate between the two. Endoscopic evaluation of the colon may also be effective in distinguishing ACPO from large-bowel obstruction but is generally not recommended for diagnostic purposes in this setting because of its invasive nature and associated risks.

Management

Keys to management of ACPO include early recognition and diagnosis, evaluation to exclude mechanical obstruction or other causes of pseudo-obstruction, assessment for signs of ischemia or perforation which would warrant urgent surgical intervention followed by initiation of appropriate treatment plan. The degree, duration of colonic distention and the status of the bowel often determines the management. A proposed algorithm for the management of ACPO is detailed in Figure 2.

The risk of spontaneous colon perforation in ACPO about 3% [27]. The two main predictors of increase risk of colonic perforation are cecal diameter greater than 12 cm and duration ofdistension

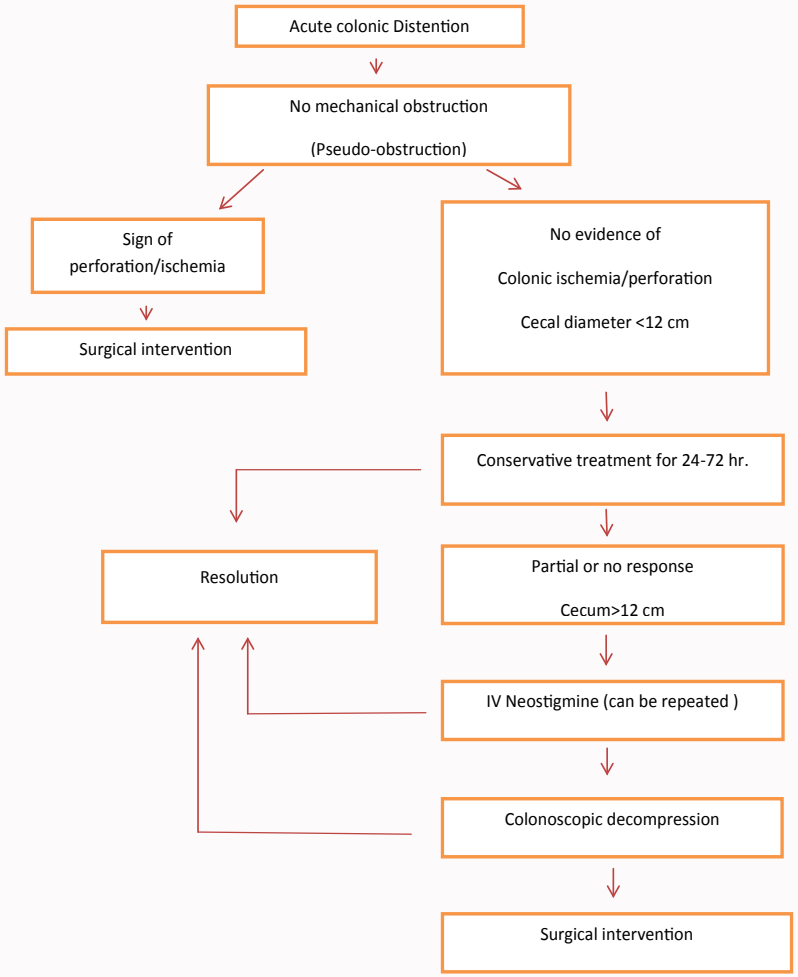

Figure 2: Algorithm for suggested management for acute colonic pseudo-obstruction.

for more than 6 days. On the basis of the limited available evidence patients with marked cecal distension (>10cm) of significant duration (>3–4days) and those not improving after 24–48 hours of supportive therapy are considered to be candidates for further intervention. In the absence of signs of ischemia or perforation, medical therapy with neostigmine should be considered the initial therapy of choice.

Conservative measures

First-line therapy for patients with ACPO without clinical or radiologic evidence of colon ischemia or perforation and cecal diameter <12cm is conservative (Box 1), which includes correction of serum electrolyte abnormalities especially hypokalemia and hypomagnesaemia, fluid resuscitation, avoidance or minimization drugs delaying gut motility (such as opiates, anticholinergics and calcium-channel blockers), identification and treatment of concomitant infection, bowel rest, ambulation, knee-chest or prone positioning to promote flatus, and the insertion of nasogastric and rectal tubes to facilitate intestinal decompression. The use of oral osmotic and stimulant laxatives is not recommended in these patients as they may worsen dilation of the colon via gas production and propulsion of gas into an already dilated colon [3,4].

- Bowel rest with discontinuing oral intake
- Intestinal decompression: nasogastric and rectal tube insertion
- Intravenous fluid replacement and correction of electrolyte imbalances
- Avoid all drugs delaying gut motility
- Avoid the use of oral osmotic and laxatives
- Increase patient mobility if possible

BOX 1: Supportive therapy for acute colonic pseudo-obstruction.

Acontinuous reassessment with serial physical examinations and repeat abdominal radiographs should be done regularly in these patients for early detection of ischemia or perforation. Clinical signs of ischemia include increased pain, fever, abdominal tenderness, and leukocytosis.

If serial examinations and abdominal radiographs do not suggest colon ischemia, perforation, or impending perforation, the conservativemeasure should generally be continued for 48-72 hour, with the expectation that it will lead toresolution of ACPO in 70% to 90% of patients [3].

Medical therapy

Neostigmine: Neostigmine is an antiacetylcholinesterase drug that transiently and reversibly increases acetylcholine levels in the synapse of muscarinic receptors of the parasympathetic nervous system. In the colon, acetylcholine promotes contractility and accelerates colon transit. In patients with ACPO, three placebo-controlled, randomized prospective trials of intravenous administration of neostigmine have

Gastrointestinal system
Salivation
Nausea
Vomiting
Abdominal pain
Cardiovascular system
Bradycardia
Hypotension
Respiratory system
Bronchospasm

Box 2: Side-effects of acetylcholinesterase inhibitors.

Gastrointestinal disease
Recent history or signs of bowel perforation or peptic ulcer
Cardiovascular disease
Recent myocardial infarction
Use of beta-blockers
Respiratory disease
Asthma
Chronic obstructive pulmonary disease
Renal insufficiency
Serum creatinine>3mg/dl

Box 3: Relative contraindications to use of acetylcholinesterase inhibitors.

shown that this drug leads to resolution of colon dilation in ≈90% of cases [28-30]. Neostigmine, administered intravenously as 2 mg over 3-5 minutes result in clinical response, which is defined as the passage of flatus or stool and decreased abdominal distension at a median interval of 4 minutes (range, 3–30 minutes). In initial nonresponders or partial responders to neostigmine, a second dose has proven effective in 40% to 100% of patients and therefore may be considered afteran interval that exceeds the normal 80-minute eliminationhalf-life of the drug [4,31-33].

Serious side effects have been reported with the use of neostigmen (Box 2), these include bronchospasm, bradycardia and hypotension, potentially leading to syncope. Therefore, the vital signs and electrocardiogram should be monitored, with medical support immediately on hand during the infusion. If bradycardia is severe, atropine should be administered promptly. Risk can be reduced by using intravenous infusion rather than bolus administration (0·4–0·8mg/h continuous intravenous administration over 24h), or starting with a dose of 1mg instead of 2mg [34].

Neostigmine should not be used in ACPO that is complicated by colon ischemia or perforation or in the setting of pregnancy, uncontrolled cardiac arrhythmias, or severe active bronchospasm. It may be used with caution in patients with bradycardia, asthma, chronic obstructive pulmonary disease, renal insufficiency, or recent myocardial infarction. Relative contraindication to neostigmine is illustrated in Box 3.

Endoscopic decompression: Colonoscopic decompression may be required in patientswith persistent, marked colonic dilatation that has failed torespond to supportive therapy and neostigmine or whenneostigmine is contraindicated.

Endoscopic decompression of the colon has been shown to result in initial colon decompression in 61% to 95% of cases and sustained decompression in the 70% to 90% range [5,27,31,35-37]. To prevent the recurrence of colon dilation, more than 1 endoscopic decompression procedure and/or endoscopic placement of a decompression tube is often required.

Surgical intervention

Surgical management is reserved for patients with signs of colonic ischemia or perforation or for those who fail endoscopic and pharmacologic treatment. Persistent colon dilation refractory to nonoperative measures can be estimated to occur in ≈10% of patients [5,28,35]. Surgical intervention is associated with significant morbidity and mortality, probably related to the severity of the underlying medical conditions in this group of patients.

Summary

Ogilvie's syndrome or acute colonic pseudo-obstruction is a clinical syndrome arising with marked abdominal distension without evidence of mechanical obstruction.Diagnosis is confirmed by abdominal radiology. Prompt treatment is important to avoid the complication of perforated cecum. Treatment should include an initial trial of conservative measures with, bowel rest, correction of electrolytes and cessation of medications with the potential to exacerbate the condition. If there is no improvement within 24- to 48-hour period, the patient should have a trial of neostigmine provided there are no contraindications. Endoscopic decompression should be reserved for patients who do not respond to neostigmine administration. In the presence of peritoneal signs or perforation, surgery is the appropriate first intervention.

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