An update on acute colonic pseudo-obstruction (Ogilvie's Syndrome)

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Definition and prevalence

The syndrome of acute colonic pseudo-obstruction was first described by Ogilvie in 1948 (1). Ogilvie's syndrome is characterized by an acute massive dilation of the caecum and the right colon, in the absence of a distal mechanical obstruction. Since then, it has been recognized as a complication occurring in patients who underwent surgery and in patients with serious medical conditions. Ogilvie's syndrome is associated with an underlying disease in 95 percent of patients. Most often, the dilatation of the right colon is associated with a simultaneous pseudo-obstruction of the small intestine, which is less severe (2).

Ogilvie's syndrome is a relatively rare condition, but since the original description more then 500 cases were described in literature. It has been estimated that this complication occurs in approximately 0.1% of the patients undergoing surgery, in approximately 0.05% of the patients admitted for trauma and in 0.3% of critically ill patients with burns (2,3). Elderly patients have a higher risk of developing acute colonic pseudoobstruction (4).

Clinical features

Ogilvie is more common in men and in patients over the age of 60. The symptoms associated with colonic pseudo-obstruction occur as a consequence of a progressive dilatation of the right colon. Initially a painless abdominal distention may occur, followed by nausea and vomiting. Abdominal pain is generally mild and constant. Cessation of bowel movements is often present, but almost half of the patients continue to pass flatus. Occasionally, diarrhea may accompany the syndrome.

The most obvious clinical finding is abdominal distention. In case of massive dilatation of the colon, mild rebound tenderness can be present. Bowel sounds may be normal, hyperactive or clangorous. Most of the cases, the abdomen is tympanic, although bowel sounds are present in almost 90 percent op patients.

Peritoneal signs are absent in the early stages of the disease. If present, they suggest impending perforation. A low-grade fever may also be present. There are no pathognomonic physical or laboratory findings.

Complications

The most severe complication of colonic pseudo-obstruction, with a high mortality rate, is a colonic perforation and ischemic necrosis caused by progressive massive dilatation. When a perforation occurs, this is usually at the level of the caecum. A diameter of the caecum on abdominal x-rays of 12 cm or more indicates imminent perforation (5). Perforation should be suspected in case of a clinically quiet abdomen, increasing fever and progressive elevation of the leukocyte count.

Diagnosis and differential diagnosis

Laboratory tests may show increased blood leukocytes, and disturbances in electrolyte levels such as hypokalemia, hyponatremia, hypocalcemia or hypomagnesemia. The presence of leucocytosis is not attributable to uncomplicated obstruction, but suggests perforation or underlying disease.

Acute colonic pseudo-obstruction has to be differentiated from mechanical colonic obstruction caused by sigmoid volvulus, tumors, benign strictures or fecal impaction. Additional differential diagnoses include toxic megacolon, ischemic colitis, typhlitis and chronic idiopathic intestinal pseudo-obstruction.

A plain abdominal x-ray will demonstrate dilatation of the right colon (caecum > 9 cm), most often with an abrupt end at the hepatic or splenic flexure. Haustral markings are normal. A prone lateral x-ray of the rectum demonstrating gaseous distention of the rectum may add to the diagnosis. Gaseous distention of the rectum does not occur in patients with structural obstructive lesions and is suggestive for pseudo-obstruction (6). Although the colon is usually devoid of air-fluid levels, they may be present in dilated loops of small intestine.

An enema with water-soluble contrast medium may further aid in diagnosing colonic pseudo-obstruction and in excluding mechanical obstruction and toxic megacolon. Its sensitivity and specificity is higher than that of plain abdominal x-ray and history. Colonoscopy has both diagnostic and therapeutic possibilities and is

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Table 1

Conditions associated with acute colonic pseudo-obstruction
Trauma (non-surgical)
Surgery (abdominal, gynecologic, orthopedic, urologic)
Inflammatory processes (pancreatitis, cholcystitis, appendicitis,…)
Toxic megacolon (IBD, pseudomembranous colitis,…)
Malignancy
Radiation therapy
Drugs (narcotis, antidepressants, phenothiazines, clonidine, anticholinergics, cortico steroids, theophylline)
Cardiovascular disease
Neurologic disease
Respiratory failure
Metabolic disease (electrolyte and acid~base imbalance, diabetes, hypothyroidism, alcoholism, uremia, lead poisoning)
Burns
Retroperitoneal hemorrhage
Mesenteric ischemia

considered a safe procedure in the setting of acute colonic pseudo-obstruction.

Pathophysiology

The pathogenesis and pathophysiology of Ogilvie's syndrome remain incompletely elucidated. Most cases of Ogilvie's syndrome occur with a variety of underlying abdominal and extra-abdominal conditions (Table 1) (2, 3,4,7). The mechanisms through which these conditions induce colonic dilatation are unknown. In particular, it is unclear whether hypocontractility of the proximal colon or hyperactivity of the distal colon are the prime event. Some observations suggest an imbalance of the inhibitory sympathetic and the excitatory parasympathetic innervation of the distal colon, leading to a functional obstruction (8). The original description of the syndrome by Ogilvie was in patients with retroperitoneal malignancy invading the prevertebral ganglia, suggesting interruption of the sympathetic drive, which would lead to unopposed parasympathetic stimulation with a hypertonic contraction of the distal colon (1). According to current views, however, transient impairment of the sacral parasympathetic nerves causing atony of the distal large bowel, or hyperactivity of extrinsic or intrinsic inhibitory nerves cannot be ruled out (9).

Treatment

The goals of therapy are to avoid perforation and to gain time until the syndrome runs its course. Treatment consists of conservative measures, removal of precipitants, colonoscopic decompression, surgical caecostomy and laparotomy. Although numerous cases have been reported in the literature, controlled clinical trials are lacking.

A. *Conservative measures*

A trial of conservative measures alone is appropriate in patients who lack significant abdominal pain, who have no signs of peritonitis and who have underlying factors that are potentially reversible. Conservative measures include correction of fluid and electrolyte imbalance, cessation of sedative, narcotic and anticholinergic drugs, treatment of underlying systemic medical conditions, optimization of tissue oxygenation and withholding oral intake (2). Maximization of physical activity and positional changes can be useful (10).

Conservative measures also include intestinal decompression using nasogastric suction and insertion of a rectal canula through a rigid rectoscope. Gentle enemas can be used, but with much care not to cause perforation. Oral laxatives are generally not helpful, and lactulose should be avoided as it may promote intraluminal accumulation of gas. The patient should be mobilized or periodically turned from side to side and also placed prone. In the absence of signs of tenderness over the caecum, conservative treatment can be prolonged for up to 72 hours. After this time pharmacological and/or colonoscopic decompression should be considered.

B. *Pharmacotherapy*

A second step in the management is the introduction of pharmacological agents which stimulate colonic motility. Prior to using stimulants of colonic motility, organic obstruction should be ruled out.

1. *Neostigmine* is an acetylcholinesterase inhibitor acting by competing with acetylcholine for attachment to acetylcholinesterase at sites of cholinergic transmission. Administration of this drug results in increased availability of acetylcholine at the neuromuscular junction, thereby enhancing smooth muscle contractility. In Ogilvie's syndrome, neostigmine is administered intravenously or subcutaneously (11-14). A recent study (12) did show a complete clinical resolution of large bowel distention in 26 of 28 patients (93%). Time to flatus varied from 30 seconds to 10 minutes after administration of 2.5 mg IV over 3 minutes. No complications or adverse effects were noted. Other studies using neostigmine (2.5 mg IV as a slow injection or over 60 minutes) did show a rapid and satisfactory clinical and radiological decompression of the large bowel in a high percentage of patients (11-14). In most patients

the response was durable. Acontrolled study of intravenous neostigmine or placebo confirmed the efficacy of cholinesterase inhibitors in Ogilvie's syndrome (14). The most frequently observed adverse effects were transient mild to moderate crampy abdominal pain, excessive salivation and vomiting. In theory, administration of a cholinesterase inhibitor might induce symptomatic bradycardia requiring atropine, but the occurrence has been found to very low. Nevertheless, neostigmine should not be used in patients on beta-blockers or with recent myocardial infarction. In susceptible patients, cholinesterase inhibitors can also induce bronchospasm.

2. *Cisapride* is a 5-hydroxytryptamine4 receptor agonist, which acts by enhancing the release of acetylcholine in the myenteric plexus of the gut wall and thereby induces colonic contraction and shortens colonic transit time (15). There is anecdotal evidence of succesful treatment with cisapride in Ogilivie's syndrome (10 mg IV every four hours for four doses, followed by 10 mg PO TID) (16, 17). However, others have been unable to observe a clinical benefit of cisapride in acute colonic pseudo-obstruction (18). As the use of cisapride may be complicated by cardiac arrhythmias, availability of cisapride has been restricted and in most European countries the drug is no longer available. So far, the use of newer 5-HT4 agonists such as tegaserod or prucalopride in colonic pseudo-obstruction has not been reported.
3. *Erythromycin* stimulates smooth muscle contraction by binding to motilin receptors in the intestine (19). It has strong prokinetic properties, mainly in the upper gut (20, 21). Anecdotal reports exist of successful treatment of Ogilvie's syndrome with erythromycin either orally (250 mg QID for 10 days) of intravenously (250 mg in 250 ml of normal saline every eight hours for three days) (22-24).

In addition to those medications anecdotal success has been reported with ganglionic blockers (guanethidine) (8), ganglionic stimulants (nicotine patch) and epidural anesthesia (to provide sympathetic blockade) (25). Naloxone may be useful in opioid-induced acute colonic pseudo-obstruction. Conservative treatment without colonoscopy may result in a resolution of the dilatation in 53 to 96% within 3 to 6 days (2,13,26).

C. *Endoscopic decompression of the colon*

Endoscopic decompression is indicated when the colonic diameter exceeds 12 cm, when dilatation persists for more than 48 to 72 hours under supportive measures, and in the presence of progressive dilatation or clinical deterioration. But there is no exact colonic diameter that mandates decompression.

Uncontrolled studies suggest that colonoscopic decompression is a safe procedure (27-32). Standard colon cleansing before colonoscopy is omitted by patients with Ogilvie's syndrome. Stool in the distal colon can be removed by small-volume saline enemas. Successful colonoscopic decompression is achieved in 60 to 90% of the patients with Ogilvie's syndrome, but recurrence may occur in 20 to 45%. Insertion of a multifenestrated decompression tube during colonoscopy may help to avoid rapid recurrence. It is, however, a laborious procedure with a potentially higher risk when the tube is dragged along by the colonoscope. Moreover, the tube can become occluded with solid fecal material. However, the reported perforation rate is less than 2% with morbidity and mortality rates of only 3 and 1% respectively (27-32).

Colonoscopic decompression only causes a small decrease in cecal size (32). As the clinical benefit of colonoscopic decompression has been demonstrated in patients with acute pseudo-obstruction, the early clinical improvement does not necessarily correlate with radiographic changes in cecal diameter.

D. *Surgical intervention*

Surgery is reserved to patients in which medical management and endoscopic decompression were unsuccessful or by clinical signs indicating impending or actual cecal perforation. The type of surgery depends on the findings at laparotomy.

1. *Surgical caecostomy* is indicated when endoscopic decompression was unsuccessful. In the absence of ischemia or perforation, a simple surgical caecostomy can be performed under local anesthesia (33, 34). An alternative approach to achieve decompression consists of a percutaneous puncture guided by laparoscopy, ultrasonography or radiography (35, 36). Catheter caecostomy requires vigorous postoperative tube care to ensure adequate function and minimal morbidity. Abdominal wall cellulitis and sepsis have been reported as complications.

2. Patients who have a perforated bowel, need a *total colectomy, ileostomy or a hartmann procedure.* Decompressive transverse colostomies and diverting ileostomies produce a mortality rate up to 40% in absence of colonic necrosis, rising to 50% in presence of colonic necrosis and peritonitis. Exploratory laparotomy is indicated in patients with massive catcall distention presenting with pneumoperitoneum development of peritonitis, respiratory failure and diagnostic uncertainty (33,37). Reported overall mortality rate of surgical procedures in patients with acute colonic pseudo-obstruction is 10-15%.

Prognosis

In spite of optimal management, mortality rates of 3 to 50% have been reported. Risk factors are old age, underlying diseases, caecal size and delay of adequate therapy. Early diagnosis and clinical awareness in case of painless abdominal distention in a patient with a predisposing condition are important in avoiding significant morbidity and mortality.

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