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Systematic review: acute colonic pseudo-obstruction

M. D. SAUNDERS & M. B. KIMMEY
Division of Gastroenterology, University of Washington, Seattle, WA, USA

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SUMMARY

Acute colonic pseudo-obstruction is the clinical syndrome of acute large bowel dilatation without mechanical obstruction that is an important cause of morbidity and mortality. Acute colonic pseudo-obstruction occurs in hospitalized or institutionalized patients with serious underlying medical and surgical conditions.

The pathogenesis of acute colonic pseudo-obstruction is not completely understood but likely results from an imbalance in the autonomic regulation of colonic motor function. Metabolic or pharmacological factors, as well as spinal or retroperitoneal trauma, may alter the autonomic regulation of colonic function, leading to excessive parasympathetic suppression or sympathetic stimulation. This imbalance results in colonic atony and dilatation.

Early recognition and appropriate management are critical to minimizing morbidity and mortality. The mortality rate is estimated at 40% when ischaemia or perforation occurs. The best-studied treatment of acute colonic pseudo-obstruction is intravenous neostigmine, which leads to prompt colon decompression in the majority of patients after a single infusion. In patients failing or having contraindications to neostigmine, colonoscopic decompression is the active intervention of choice. Surgery is reserved for those with peritonitis or perforation.

INTRODUCTION

Colonic pseudo-obstruction is a term used to characterize a clinical syndrome with symptoms, signs, and radiographic appearance of large bowel obstruction without a mechanical cause.[1] According to presentation, pseudo-obstruction syndromes can be subdivided into acute and chronic forms. Acute colonic pseudo-obstruction (ACPO) is characterized by massive colonic dilatation in the absence of mechanical obstruction.[2] ACPO is also referred to as acute colonic ileus or Ogilvie's syndrome, the latter because of the original report by Sir William Ogilvie of two cases of colonic ileus due to retroperitoneal malignancy with invasion of the celiac plexus.[3] ACPO is an important cause of morbidity and mortality. Ischaemia and perforation are the feared complications of ACPO. Spontaneous perforation has been reported in 3–15% of cases with a mortality rate estimated at 50% or higher when this occurs.[4]

The clinical presentation of ACPO has been extensively documented in the literature. However, despite the increasing awareness of this condition, its diagnosis remains difficult and is often delayed. Early detection and prompt appropriate management are critical to minimizing morbidity and mortality. The aim of this article is to review current evidence pertaining to the diagnosis, pathogenesis, evaluation and management of patients with ACPO.

METHODS

A MEDLINE literature search of the English language literature from 1999 to 2005 was performed using the following keywords and MESH terms: acute colonic pseudo-obstruction, intestinal pseudo-obstruction, colonic ileus, Ogilvie's, adynamic ileus, neostigmine and

Correspondence to: Dr M. B. Kimmey, University of Washington, 1959 NE Pacific St (AA103K), Box 356424, Seattle, WA 98195, USA.
E-mail: kimmey@u.washington.edu

colonoscopic decompression. Additional references were obtained from the bibliographies of the identified articles.

PATHOPHYSIOLOGY

The pathogenesis of ACPO is not completely understood although it likely results from an alteration in the autonomic regulation of colonic motor function.[5] The vast majority of patients (>95%) with ACPO have the syndrome in association with one or more multiple predisposing factors or clinical conditions (Table 1). In a large retrospective series of 400 patients, the most common predisposing conditions were non-operative trauma (11%), infections (10%) and cardiac disease (10%).[6] Caesarean section and hip surgery were the most common surgical procedures. In a further retrospective analysis of 48 patients, the spine or retroperitoneum had been traumatized or manipulated in 52%.[7] Over half the patients were receiving narcotics, and electrolyte abnormalities were present in approximately two-thirds. Thus, multiple metabolic, pharmacological, or traumatic factors appear to alter the autonomic regulation of colonic function resulting in pseudo-obstruction. The mechanisms through which these different conditions temporarily suppress colonic motility and induce dilatation are unknown.

The parasympathetic nervous system increases contractility, whereas the sympathetic nerves decrease motility in the colon.[5] An imbalance in autonomic innervation, produced by a variety of factors, leads to excessive parasympathetic suppression or sympathetic stimulation. Because the vagal supply to the large bowel terminates at the splenic flexure and the parasympathetic innervation of the left colon originates from the sacral plexus, it has been proposed that transient parasympathetic impairment at the sacral plexus may cause atony of the distal large bowel and result in functional obstruction.[6] Alternatively, hyperactivity of inhibitory neurones to the large bowel, as a result of increased sympathetic drive, may play an important role in the pathophysiology of ACPO.[5] Mechanoreceptors located within the wall of the large intestine, when stimulated by distention, activate a reflex pathway whose final effect, via efferent sympathetic nerves targeting the myenteric plexus or colonic smooth muscle, is the inhibition of colonic motility (colo-colonic reflex).[5] However, despite improved knowledge of the pathophysiology of colonic motility, the precise mechanisms underlying ACPO remain poorly understood.

Table 1. Predisposing conditions associated with acute colonic pseudo-obstruction (ACPO) – an analysis of 400 cases*

Condition	Number	Percentage
Trauma (non-operative)	45	11.3
Infection (pneumonia, sepsis most common)	40	10.0
Cardiac (myocardial infarction, heart failure)	40	10.0
Obstetrics/gynaecology	39	9.8
Abdominal/pelvic surgery	37	9.3
Neurological (Parkinson's disease, spinal cord injury, multiple sclerosis, Alzheimer's disease)	37	9.3
Orthopaedic surgery	29	7.3
Miscellaneous medical conditions (metabolic, cancer, respiratory failure, renal failure)	128	32
Miscellaneous surgical conditions (urologic, thoracic, neurosurgery)	47	11.8

* Associated conditions in 400 patients, reported by Vanek and Al-Salti[6] Some patients had more than one associated condition.

CLINICAL PRESENTATION

The exact incidence of ACPO is unknown. It most often affects those in late middle age (mean age of 60 years), with a slight male predominance (60%).[6] ACPO occurs most often in hospitalized or institutionalized patients with serious underlying medical and surgical conditions. Abdominal distention usually develops over 3–7 days but can occur as rapidly as 24 h.[6] In surgical patients, symptoms and signs develop at a mean of 5 days post-operatively.

The clinical features of ACPO include abdominal distention, abdominal pain (80%) and nausea and/or vomiting (60%).[6] Passage of flatus or stool is reported in up to 40% of patients. Patients with ischaemic or perforated bowel have similar symptoms but are more likely to be febrile.[6] On examination, the abdomen is tympanitic and bowel sounds are typically present. Fever, marked abdominal tenderness and leucocytosis are more common in patients with ischaemia or perforation but also occur in those who have not developed these complications.[6]

DIAGNOSIS

The diagnosis of ACPO is suggested by the clinical presentation and confirmed by plain abdominal

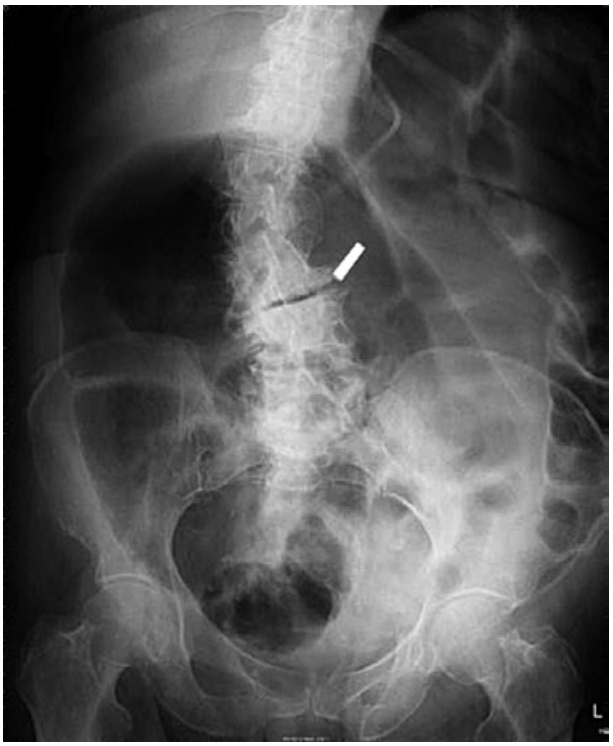

Figure 1. The diagnosis of acute colonic pseudo-obstruction (ACPO) is confirmed by plain abdominal radiographs, which show varying degrees of colonic dilatation.

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 megacolon caused by severe *Clostridium difficile* infection,[8] and ACPO. A water-soluble contrast enema or computerized tomography scan should be obtained to exclude mechanical obstruction if gas and distention is not present throughout all colonic segments including the rectum and sigmoid colon.

MANAGEMENT

The appropriate evaluation of a patient with suspected ACPO requires early recognition and diagnosis, exclusion of mechanical obstruction or other causes of pseudo-obstruction, assessment for signs of peritonitis or perforation which would warrant urgent surgical intervention, and initiation of appropriate treatment measures. The degree and duration of colonic distention often determines the pace and sequence of management options.

The clinical dilemma facing the clinician caring for a patient with ACPO is whether to treat the patient with conservative measures and close observation vs. proceeding with medical or endoscopic decompression of the dilated colon. The outcome of patients with ACPO is determined by multiple factors. The severity of the underlying illness has the greatest influence on patient outcome. ACPO often afflicts debilitated patients, which explains the significant morbidity and mortality even with successful treatment of the colonic dilatation. Other factors that influence outcome are increasing age, maximal caecal diameter, delay in decompression and status of the bowel.[6]

The risk of spontaneous colon perforation in ACPO is low but clearly exists. Rex summed all available reports in the literature and determined the risk of spontaneous perforation to be approximately 3%.[9] The mortality rate in ACPO is approximately 40% when ischaemia or perforation are present compared to 15% in patients with viable bowel.[4]

Retrospective analyses of patients with ACPO[6, 10] have attempted to identify clinical factors that predict which patients are more likely to have complications such as ischaemia or perforation. Based on LaPlace's law, increasing diameters accelerate the rise in tension experienced by the colon wall. The risk of colonic perforation has been reported to increase with caecal diameter >12 cm[6] and when distention has been present for more than 6 days.[10] In the large retrospective series, no cases of perforation were seen with a caecal diameter <12 cm.[6] However, at diameters >12 cm, there is no clear relationship between risk of ischaemia or perforation and the size of the caecum. The duration and progression of colonic distention may be more important. Johnson and Rice reported a mean duration of distention in patients who perforated of 6 days compared with 2 days in those who did not.[10] A twofold increase in mortality occurs when caecal diameter is >14 cm and a fivefold increase when delay in decompression is >7 days.[6] Thus, the decision to intervene with medical therapy, colonoscopy or surgery is dictated by the patient's clinical status.

Treatment options for ACPO include appropriate supportive measures, pharmacological therapy, colonoscopic decompression and surgery. Despite extensive literature documenting the clinical features of ACPO, there are few controlled clinical trials on the treatment of this condition, and most evidence for efficacy of treatments comes from anecdotal reports, retrospective reviews, or uncontrolled studies.

Supportive therapy

Supportive therapy (Table 2) is the preferred initial management of ACPO and should be instituted in all patients.[11] Patients are given nothing by mouth. Intravenous fluids are administered and electrolyte imbalances are corrected. Nasogastric suction is provided to limit swallowed air from contributing further to colonic distention. Laxatives are avoided, particularly lactulose, which provides substrate for colonic bacterial fermentation, resulting in further gas production. A rectal tube should be inserted and attached to gravity drainage. Medications that can adversely affect colonic motility, such as opiates, anticholinergics and calcium-channel antagonists are discontinued if possible. Ambulation and mobilization of patients are encouraged. The knee–chest position with hips held high has been advocated as aiding in evacuation of colonic gas.[11] The benefits of any particular component of these supportive measures are unknown as these measures have not been studied individually.

Conservative management is successful as the primary treatment in the majority of patients.[11–17] Sloyer *et al.* reported the outcome of 25 cancer patients with ACPO (mostly non-gastrointestinal malignancies).[12] The mean caecal diameter was 11.7 cm (range: 9–18). Of the 24 patients treated conservatively, 23 (96%) improved by clinical and radiological criteria with the median time to improvement of 1.6 days (mean: 3 days). There were no perforations or ACPO-related deaths. In another retrospective series of 151 patients, 117 (77%) had spontaneous resolution of ACPO with conservative treatment.[17] These studies demonstrate that the initial management of ACPO should be directed towards eliminating or reducing factors known to contribute to the problem.

Table 2. Supportive therapy for acute colonic pseudo-obstruction

Nothing by mouth (NPO)
Correct fluid and electrolyte imbalances
Nasogastric tube suction
Rectal tube to gravity drainage
Limit offending medications
Frequent position changes, ambulate if possible

Patients with marked caecal distention (>10 cm) of significant duration (>3–4 days) and those not improving after 24–48 h of supportive therapy are candidates for further intervention. In the absence of signs of peritonitis or perforation, medical therapy with neostigmine should be considered the initial therapy of choice.

Medical therapy

Neostigmine. The only randomized-controlled therapeutic trial for ACPO studied the use of intravenous neostigmine.[18] Neostigmine, a reversible acetylcholinesterase inhibitor, indirectly stimulates muscarinic parasympathetic receptors, thereby enhancing colonic motor activity, inducing colonic propulsion and accelerated transit.[19] The rationale for using neostigmine stems from the imbalance in autonomic regulation of colonic function that is proposed to occur in ACPO. Neostigmine was first used for manipulation of the autonomic innervation to the gastrointestinal tract by Neely and Catchpole over 30 years ago in studies on small bowel paralytic ileus.[20] Neostigmine, administered intravenously, has a rapid onset of action (1–20 min) and short duration (1–2 h).[21] The elimination half-life averages 80 min, which is prolonged in patients with renal insufficiency.[22]

A randomized, double-blind, placebo-controlled trial evaluated neostigmine in patients with ACPO with a caecal diameter of >10 cm and no response to 24 h of conservative therapy.[18] Exclusion criteria were suspected ischaemia or perforation, pregnancy, severe active bronchospasm, cardiac arrhythmias and renal failure. Patients were randomized to receive neostigmine, 2 mg, or saline by intravenous infusion over 3–5 min. The primary end point was the clinical response to infusion, defined as a reduction in abdominal distention by physical examination. Secondary end points included the change in measurements of colonic diameter on radiographs and abdominal girth. Patients not responding within 3 h to initial infusion were eligible for open-label neostigmine. A clinical response was observed in 10 of 11 patients (91%) randomized to receive neostigmine compared with 0 of 10 receiving placebo (Figure 2). The median time to response was 4 min.

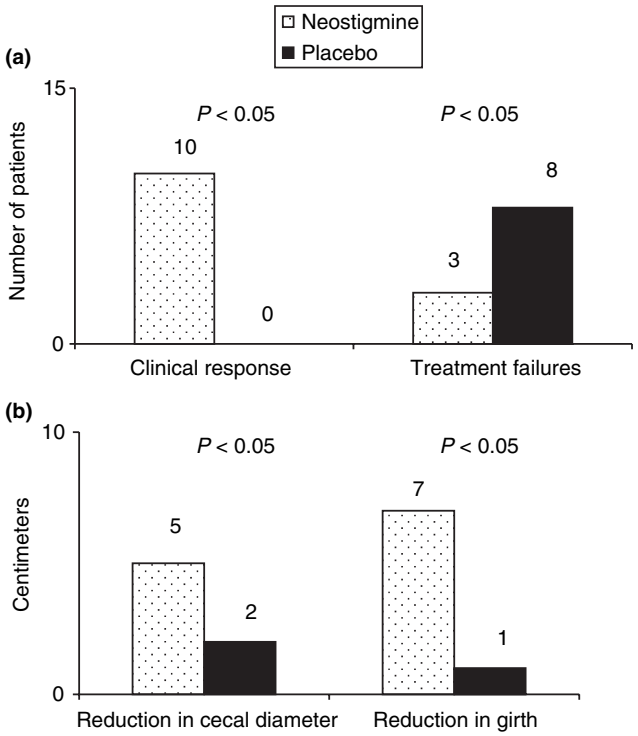

Figure 2. Results of a randomized-controlled trial of neostigmine treatment for acute colonic pseudo-obstruction (ACPO). (a) Illustrates clinical response and treatment failures and (b) reductions in caecal diameter and abdominal girth.

Median reduction in caecal diameter (5 vs. 2 cm) and abdominal girth (7 vs. 1 cm) were significantly different in favour of neostigmine over placebo (Figure 2). Eight patients not responding to initial infusion (seven placebo, one neostigmine) were administered open-label neostigmine, and all had prompt decompression. Of the 18 patients who received neostigmine, either initially or during open-label treatment, 17 (94%) had a clinical response. The recurrence of colonic distention after neostigmine decompression was low (11%). The most common side-effects observed with neostigmine were mild abdominal cramping and excessive salivation. Symptomatic bradycardia requiring atropine occurred in two of 19 patients.

There are also several uncontrolled, open label and retrospective series supporting the use of neostigmine in this condition.[17, 23–28] Collectively, rapid decompression of colonic distention was observed in 88% of patients with a recurrence rate of 7% (Table 3). The cost of neostigmine is minimal, with a 2 mg ampule for parenteral use costing approximately $3 US.[18] The cost to the patient after storage and handling fees are included is approximately $15 US.

Although neostigmine was associated with a favourable safety profile in the reported clinical trials, caution should be used when administering the medication. Neostigmine should be given with the patient kept supine in bed with continuous electrocardiographic and vital sign monitoring, as well as clinical assessment for 15–30 min following administration (Table 4). Contraindications to its use include bowel obstruction, ischaemia or perforation, pregnancy, uncontrolled cardiac arrhythmias, severe active bronchospasm and renal insufficiency (serum creatinine >3 mg/dL).

Thus, neostigmine appears to be an effective, safe and inexpensive method of colonic decompression in ACPO. The published data supports its use as the initial therapy

Table 3. Neostigmine for colonic decompression in patients with ACPO

Study	Number	Design	Intravenous dose (mg)	Decompression	Recurrence
Ponec *et al.*[18]	21 (neostigmine 11, placebo 10)	RCT (OL in non-responders)	2.0 over 3–5 min	10/11 in RCT 17/18 total	2
Hutchinson and Griffiths[23]	11	OL	2.5 in 1 min	8/11	0
Stephenson *et al.*[24]	12	OL	2.5 over 1–3 min	12/12 (2 patients required 2 doses)	1
Turegano-Fuentes *et al.*[25]	16	OL	2.5 over 60 min	12/16	0
Trevisani *et al.*[26]	28	OL	2.5 over 3 min	26/28	0
Paran *et al.*[27]	11	OL	2.5 over 60 min	10/11 (2 patients required 2 doses)	0
Abeyta *et al.*[28]	8	Retrospective	2.0	6/8 (2 patients required 2 doses)	0
Loftus *et al.*[17]	18	Retrospective	2.0	16/18	5
Total	122			107 (88%)	8 (7%)

OL, open label; RCT, randomized-controlled trial; ACPO, acute colonic pseudo-obstruction.

Table 4. Administration of neostigmine

Neostigmine, 2 mg, intravenous infusion over 3–5 min
Atropine available at bedside
Patient kept supine, on bedpan
Continuous electrocardiographic monitoring with vital signs for 30 min
Continuous clinical assessment for 15–30 min

of choice for patients not responding to conservative therapy if there are no contraindications to its use. In patients with only a partial response or recurrence after an initial infusion, a repeated dose is reasonable and often successful. If the patient fails to respond after two doses, proceeding with colonoscopic decompression is advised.

Other pharmacological therapy

There are only anecdotal reports using other prokinetic agents in ACPO. Erythromycin, a motilin receptor agonist, has been reported to be successful in treating patients in a few case reports.[29, 30] Armstrong *et al.* reported decompression in two patients with ACPO with oral erythromycin (500 mg four times daily) for 10 days.[29] In another report, a patient with ACPO had resolution after 3 days of intravenous erythromycin therapy.[30] The potential of newly developed motilin receptor agonists to stimulate colonic motility remains to be determined.

Cisapride, a partial 5-HT_4 receptor agonist, has been employed with some success in patients with ACPO.[31] However, this agent is no longer available for use in the United States and Canada because of class III antiarrhythmic properties. Second generation 5-HT4 partial receptor agonists, such as tegaserod, may be more active at the colonic level than cisapride.[32] Although theoretically useful, no formal assessment of tegaserod in controlled trials in ACPO is available.

Non-surgical decompression

Non-surgical approaches to mechanical decompression have included radiological placement of decompression tubes, colonoscopy with or without placement of a decompression tube and percutaneous cecostomy performed through a combined endoscopic-radiological approach. Colonoscopic decompression is preferred among these invasive, non-surgical options given the reported experience in the literature, recently totalling many hundreds of patients.[11]

Colonic decompression is the initial invasive procedure of choice for patients with marked caecal distention (>10 cm) of significant duration (>3–4 days), not improving after 24–48 h of supportive therapy, and who have contraindications to or fail pharmacological therapy (neostigmine). Colonoscopy is performed to prevent bowel ischaemia and perforation. It should not be performed if overt peritonitis or perforation is present. It is unclear whether mucosal ischaemia identified at endoscopy is a contraindication to proceeding with decompression. Traditional wisdom has been to withdraw the endoscope and proceed with surgery. However, there are case reports of patients with ischaemia complicating ACPO being successfully managed with colonoscopic decompression.[33] Patients with mucosal ischaemia identified during colonoscopy may be considered for non-operative management if they lack peritoneal findings and colonoscopic decompression is successful.

Colonoscopy in ACPO is a technically difficult procedure and should be performed by experts. Oral laxatives and bowel preparations should not be administered prior to colonoscopy. Colonoscopes with large diameter accessory channels (3.8 mm) or a dual channel colonoscope, are preferable to allow suctioning of stool and gas. Air insufflation should be minimized and the entire colon need not be examined. The colonoscope should be advanced as far as possible. Prolonged attempts at caecal intubation are not necessary because reaching the hepatic flexure usually suffices. Gas should be aspirated and the viability of the mucosa assessed during slow withdrawal of the endoscope. A tube for decompression should be placed in the right colon with the aid of a guidewire and fluoroscopic guidance. Commercially available, disposable, over-the-wire colon decompression tubes are available. If available, fluoroscopy should be used to keep guidewires straight and to minimize loop formation during tube advancement into the right colon.

The efficacy of colonoscopic decompression has not been established in randomized-clinical trials. However, successful colonoscopic decompression has been reported in many retrospective series (Table 5), now totalling many hundreds of patients.[7, 34–37] Rex reviewed the available literature of patients with ACPO treated with colonoscopy.[38] Among 292 reported patients, 69% were estimated to have a successful initial decompression,

Table 5. Colonoscopic decompression in acute colonic pseudo-obstruction

Study	Number	Successful initial decompression (%)	Overall colonoscopic success (%)	Complications (%)
Nivatvongs *et al.*[34]	22	68	73	<1 (no perforations)
Strodel *et al.*[35]	44	61	73	2 (1 perforation)
Bode *et al.*[36]	22	68	77	4.5 (1 perforation)
Jetmore *et al.*[7]	45	84	36	<1 (no perforations)
Geller *et al.*[37]	41	95	88	2 (2 perforations)

determined by a reduction in radiographically measured caecal diameter. About 40% of patients treated without decompression tube placement had at least one recurrence, requiring an additional colonoscopy. Thus, an initial decompression colonoscopy without tube placement can be considered to be definitive therapy in <50% of patients.[38] To improve the therapeutic benefit, decompression tube placement at the time of colonoscopy is strongly recommended. The value of decompression tubes has not been evaluated in controlled trials, but anecdotal evidence suggests that it may lower the recurrence rate. In the series reported by Geller *et al.*, the overall clinical success of colonoscopic decompression was 88%. However, in procedures where a decompression tube was not placed the clinical success was poor (25%).[37] Tube placement is not, however, completely effective in preventing recurrences. Decompression colonoscopy has a reported perforation rate of approximately 3%,[37] a figure that is much higher than in patients without ACPO.

Percutaneous cecostomy, performed through a combined endoscopic-radiological approach, can be considered in high surgical risk patients.[39–41] A recently published small case series described five patients (two patients with ACPO) in which percutaneous endoscopic cecostomy was the definitive treatment.[41] In one ACPO patient, the cecostomy tube was removed without incident after 10 weeks. In the other patient, the tube cecostomy remained in place. There were no complications reported with tube placement in this small series. It is uncertain when percutaneous endoscopic cecostomy should be considered over other forms of mechanical decompression, such as colonoscopy with decompression tube placement or surgical cecostomy. A controlled trial comparing percutaneous endoscopic cecostomy to colonoscopy with decompression tube placement would be welcome. At the present time, percutaneous endoscopic cecostomy should be reserved for patients failing neostigmine and colonoscopic decompression who have no evidence of ischaemia or perforation and who are felt to be at high risk for surgery.

Surgical therapy

Surgical management is reserved for patients with signs of colonic ischaemia or perforation or who fail endoscopic and pharmacological efforts at decompression. Surgical intervention is associated with significant morbidity and mortality, likely related to the severity of the patients' underlying medical condition. In a large, retrospective series, 179 patients underwent surgery for ACPO with resulting morbidity and mortality rates of 30% and 6%, respectively.[6] The type of surgery performed depends on the status of the bowel. Without perforated or ischaemic bowel, cecostomy is the procedure of choice because the success rate is high, morbidity is relatively low, and the procedure can be performed under local anaesthesia.[6] In cases of ischaemic or perforated bowel, segmental or subtotal colonic resection is indicated, with either exteriorization or primary anastomosis.

CLINICAL GUIDELINES

An evidenced-based guideline for the treatment of ACPO was recently published by the American Society for Gastrointestinal Endoscopy.[11] The guideline recommends conservative therapy as the initial preferred management. Potentially contributory metabolic, infectious and pharmacological factors should be identified and corrected. Active intervention is indicated for patients at risk of perforation and/or failing conservative therapy. Neostigmine is effective in the majority of patients (randomized-controlled trial). Colonic decompression is the initial invasive procedure of choice

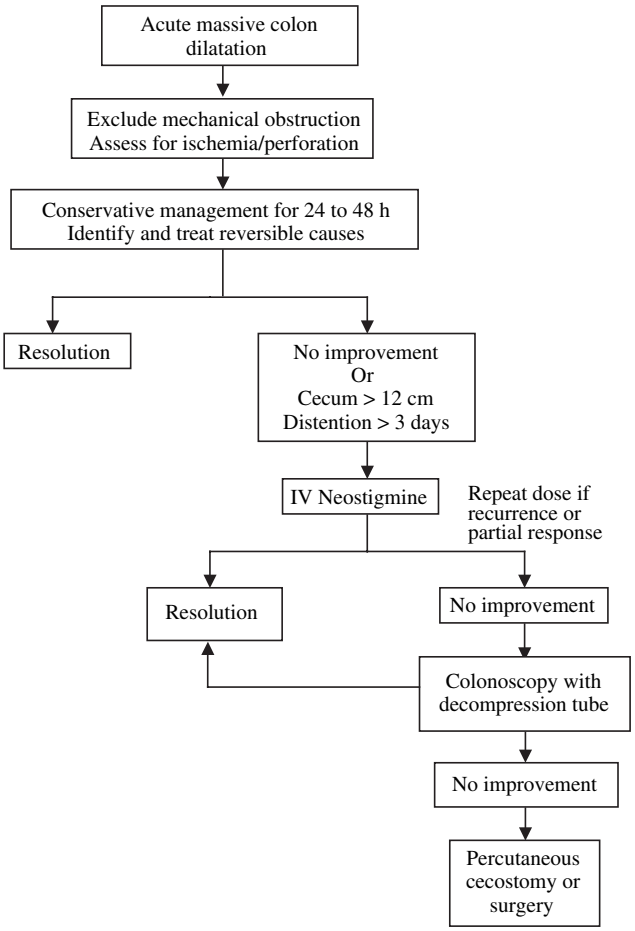

Figure 3. Suggested management algorithm for acute colonic pseudo-obstruction.

for patients failing or who have contraindications to neostigmine (observational studies only). Surgical decompression should be reserved for patients with peritonitis or perforation and for those failing endoscopic and medical therapy. A proposed algorithm for the management of ACPO is detailed in Figure 3.

CONCLUSION

ACPO is a syndrome of acute dilatation of the colon without mechanical obstruction that results from an imbalance in the autonomic control of the colon. Evaluation involves exclusion of mechanical obstruction and assessing for signs of ischaemia or perforation. Appropriate management includes supportive measures and selective use of neostigmine and colonoscopic decompression. Neostigmine is the only therapy for ACPO proven to be efficacious in a controlled clinical trial. Patient outcome is determined by the severity of the predisposing illness, patient age, maximal caecal diameter, duration of colonic distention and viability of the bowel. Of these factors affecting outcome, the latter three are amenable to intervention. Thus, early recognition and management are critical to minimizing morbidity and mortality.

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