Review

Acute colonic pseudo-obstruction

R. De Giorgio[1] and C. H. Knowles[2]

[1]Department of Clinical Medicine and Centro Unificato di Ricerca BioMedica Applicata, University of Bologna, Bologna, Italy, and [2]Centre for Academic Surgery, Royal London Hospital, London, UK

Correspondence to: Dr R. De Giorgio, Department of Clinical Medicine, Building 5 – 'Nuove Patologie', St Orsola-Malpighi Hospital, Via Massarenti 9, 40138 – Bologna, Italy (e-mail: deg@aosp.bo.it)

Background: **Acute colonic pseudo-obstruction is characterized by clinical and radiological evidence of acute large bowel obstruction in the absence of a mechanical cause. The condition usually affects elderly people with underlying co-morbidities, and early recognition and appropriate management are essential to reduce the occurrence of life-threatening complications.**

Methods: **A part-systematic review was conducted. This was based on key publications focusing on advances in management.**

Results and conclusions: **Although acute colonic dilatation has been suggested to result from a functional imbalance in autonomic nerve supply, there is little direct evidence for this. Other aetiologies derived from the evolving field of neurogastroenterology remain underexplored. The rationale of treatment is to achieve prompt and effective colonic decompression. Initial management includes supportive interventions that may be followed by pharmacological therapy. Controlled clinical trials have shown that the acetylcholinesterase inhibitor neostigmine is an effective treatment with initial response rates of 60–90 per cent; other drugs for use in this area are in evolution. Colonoscopic decompression is successful in approximately 80 per cent of patients, with other minimally invasive strategies continuing to be developed. Surgery has thus become largely limited to those in whom complications occur. A contemporary management algorithm is provided on this basis.**

Paper accepted 23 January 2009

Published online in Wiley InterScience (www.bjs.co.uk). **DOI:** 10.1002/bjs.6480

Definitions

Motility disorders of the gastrointestinal tract encompass a wide spectrum, ranging from mild functional disturbances (for example dyspepsia) to severe conditions characterized by significantly disturbed transit, with or without visceral dilatation. One example of the latter is 'acute colonic pseudo-obstruction', which is defined as a massive dilatation of the colon with obstructive symptoms but in the absence of mechanical obstruction. Its first description is attributed to Sir William Heneage Ogilvie, who in 1948 recognized this syndrome in two patients with sudden onset of abdominal pain, constipation and large bowel dilatation, hence the eponym Ogilvie's syndrome[1]. In the original report, the clinical picture was associated with a retroperitoneal neoplasm infiltrating and destroying prevertebral ganglia, with the hypothesis that altered ('deprivation of') colonic sympathetic input played a pathogenic role[1]. Notably, similar patients had been described by Zimmerman in 1930[2].

Although subsequent descriptions of 'false colonic obstruction' followed[3], the term pseudo-obstruction was not adopted until 1958, when a case series from the Department of Clinical Surgery in Edinburgh built on earlier (since the 1930s) descriptions of 'spastic ileus' that were often associated with neurological injury[4]. Thirteen heterogeneous case reports were included, which in current taxonomy would represent a mix of chronic intestinal pseudo-obstruction, postoperative ileus and acute colonic pseudo-obstruction[5]. Although differentiating primary from secondary, and intestinal from colonic, this paper and others specifically describing 'pseudo-obstruction of the large bowel'[6,7] or pseudo-obstruction of the colon[8] never actually alluded to the term 'acute colonic pseudo-obstruction'. This term was not adopted in the published literature until the early 1980s[9]. Whether the eponym should be applied to all cases or just those

with retroperitoneal neoplasia remains a debated issue. Nonetheless, it should be noted that the avoidance of the general use of eponyms has been encouraged by several educational organizations[10].

Another important distinction of the definition is that there *must* be clinical features of large bowel obstruction. There is thus a boundary between this condition and the much more common observation in some patients of similar radiological colonic dilatation and abdominal distension in the absence of obstruction. Such patients, who represent an almost weekly referral from medical colleagues in any large acute general hospital setting, have a form of megacolon[11] that, despite commonly overlapping aetiological factors (such as senility, neurological disease and drugs affecting gut motility), is characterized by severe constipation rather than frank obstruction. The diagnosis of acute colonic pseudo-obstruction typically results from resolution of a differential diagnosis that includes mechanical obstruction of the large bowel (for example malignant and benign strictures and volvulus). However, the condition may occasionally also accompany more proximal or panenteric chronic intestinal pseudo-obstruction syndromes[1,12].

Methods

Despite the current trend towards systematic data synthesis in secondary research, it was deemed that lack of high-quality studies other than three randomized controlled trials using neostigmine would limit the use of such formal methodology. Nevertheless, a literature search was performed using Medline and Premedline from 1950 to October 2008. MeSH as well as free-text terms were chosen to negate problems of current and historical syntax, with eight iterations of colonic pseudo-obstruction and Ogilvie's syndrome thus incorporated. MeSH terms were 'colonic pseudo-obstruction/diagnosis'[Mesh] OR 'colonic pseudo-obstruction/drug therapy'[Mesh] OR 'colonic pseudo-obstruction/etiology'[Mesh] OR 'colonic pseudo-obstruction/pathology'[Mesh] OR 'colonic pseudo-obstruction/physiopathology'[Mesh] OR 'colonic pseudo-obstruction/radiography' [Mesh] OR 'colonic pseudo-obstruction/surgery'[Mesh] OR 'colonic pseudo-obstruction/therapy'[Mesh]. The search was further limited by restrictions to humans, English language and studies with abstracts (thereby excluding numerous case reports). The bibliographies of relevant papers and two textbooks were hand searched and cross-referenced, including for earlier papers. No formal quality assessment or selection criteria were employed except for therapeutic trials where a cut-off of ten participants was necessary for inclusion.

Epidemiology

The exact prevalence of acute colonic pseudo-obstruction is unknown, but may be inferred from the incidence of large bowel obstruction where it is responsible for at least 20 per cent of cases[13,14], or from its frequency of complicating certain operations. It occurs in about 1 per cent of hospitalized patients undergoing orthopaedic procedures, including lower limb joint replacement and spinal operations[15]. It affects 0·3 per cent of patients with severe burns[16]. The highest prevalence is observed in late middle age (around 60 years) and it is slightly more common (60 per cent) in men[17]. Acute colonic pseudo-obstruction is a serious condition with considerable clinical and social impact. Published data and reviews[18] clearly show that, because of multiple co-morbidities, delayed diagnosis and inappropriate treatment, it is responsible for considerable morbidity, with a mortality rate of 25–31 per cent overall and 40–50 per cent with ischaemia or perforation[10,17–20].

Aetiology

Predisposing factors

Although a few patients without any obvious underlying disease are affected, most develop acute colonic pseudo-obstruction in association with a wide spectrum of illnesses (*Table 1*). These include myocardial infarction, neurological diseases (such as Parkinson's and Alzheimer's), severe infections (particularly those induced by Gram-negative bacteria), electrolyte imbalance/metabolic alterations (for example hypokalaemia), surgery or trauma. Other situations may arise from, or be compounded by, drugs (such as antidepressants, phenothiazines, antiparkinsonian agents and opiates/narcotics) or be related to advanced age[17,19–21]. Large retrospective evaluations of 400[18] and 1027[20] patients have demonstrated that operative (23 per cent) and non-operative (11 per cent) trauma, infections (10 per cent) and cardiac disease (10–18 per cent) are the most common predisposing conditions[17,20].

Pathophysiology

The pathophysiology of acute colonic pseudo-obstruction is obscure. A variety of hypotheses relating to 'imbalanced' extrinsic autonomic innervation prevails in the review literature[11,18,21–23]. Although these are credible on the basis of association with prevertebral and retroperitoneal trauma or disease[1,7,21,24], and responses to pharmacological therapy, there is no direct evidence in their support.

Table 1 Commonly associated factors in the development of acute colonic pseudo-obstruction

Cardiovascular
Stroke
Myocardial infarction
Congestive heart failure
Metabolic
Electrolyte imbalance
Liver or renal failure
Alcohol abuse
Drug induced
Antidepressants
Opiates
Phenothiazines
Antiparkinsonian agents
Infective/inflammatory
Systemic sepsis
Herpes zoster infection
Pneumonia
Acute cholecystitis
Acute pancreatitis
Pelvic abscess
Neurological
Parkinson's disease
Alzheimer's disease
Multiple sclerosis
Low spinal cord disease
Neoplasia
Leukaemia
Retroperitoneal tumour
Radiotherapy or disseminated pelvic neoplasia
Postsurgical
Renal transplantation
Caesarean section
Gynaecological or pelvic surgery
Hip surgery
Post-traumatic
Mechanical ventilation
Spinal cord trauma
Pelvic trauma
Femoral fracture

It should also be noted that favourable responses to parasympathetic stimulation using neostigmine are not evidence that decreased parasympathetic activity is *de facto* the cause of pseudo-obstruction. Once dilated, there is some experimental evidence that stretch-sensitive mechanoceptors, located in the gut wall, contribute to self-maintenance of colonic contractile inhibition[25]. Why visceral dilatation should follow such impairment of motility is unknown.

It is well recognized that chronic visceral dilatation of unknown origin, such as chronic idiopathic intestinal pseudo-obstruction, may be the result of intrinsic morphofunctional changes of the enteric nervous system, smooth muscle or interstitial cells of Cajal[26,27]. Such changes have rarely been sought in colonic tissue of patients undergoing surgery for acute colonic pseudo-obstruction. In any event they would be difficult to interpret, given the likely presence of complications such as ischaemia in operated patients. Nevertheless, a recent study has demonstrated that four of six patients with non-inflammatory acute colonic pseudo-obstruction had a reduced number of ganglion cells[28]. There are no animal models of acute colonic pseudo-obstruction; those of postoperative ileus and toxic megacolon provide the best contemporary speculative mechanisms. Numerous rodent models of postoperative ileus demonstrate the role of reflex autonomic dysfunction in the acute dysmotility phase (broadly sympathetic and neuroendocrine overactivity)[29], and of migration and activation of leucocytes (macrophages and polymorphonuclear cells) into the muscularis propria in the later phase[30]. The latter effect is promoted by mast cell-dependent permeability changes[31,32] and attenuated by cholinergic activity[33] or loss of intrinsic nitric oxide activity[34]. Nitric oxide, an inhibitory neurotransmitter, has also been shown to be overproduced in experimental models of[35], and patients with[36], toxic megacolon.

Clinical presentation and investigation

Acute colonic pseudo-obstruction is characterized by abdominal distension, pain, nausea and/or vomiting, with a failure to pass flatus and stools documented in up to 60 per cent of patients[11,12,17,20,23]. Massive colonic dilatation may cause ischaemia and perforation, with the subsequent clinical finding of peritonism. Such complications affect 3–15 per cent of patients[17,19,20]; advanced age, large caecal diameter and delay in decompression increase the risk. Despite best care, the mortality associated with these complications is about 50 per cent. Physical examination in the uncomplicated situation typically reveals a tympanic, non-tender abdomen, with high-pitched 'tinkling', reduced or absent bowel sounds. Patients with complications present with marked abdominal tenderness and systemic features such as fever and tachycardia. The differential diagnosis in hospitalized or institutionalized patients includes mechanical obstruction and, increasingly, toxic megacolon due to *Clostridium difficile* infection, which should always be excluded by appropriate stool testing[37].

Diagnosis relies on accurate clinical observation and plain abdominal radiography showing degrees of colonic dilatation, mainly involving the proximal colon[38–40]. Plain abdominal and chest radiographs can also give some indication of colonic diameter as well as detecting the presence of

free air, suggesting perforation. However, in all situations of large bowel obstruction, no matter how clear the diagnosis appears on plain radiology, a water-soluble contrast enema[13,14] or computed tomography (CT)[41] 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 (as confirmed ultimately by surgery). Contrast enema has a sensitivity of 96 per cent and specificity of 98 per cent[40]; CT with intravenous contrast has a sensitivity and specificity both of 91 per cent[42]. CT has the additional advantages of allowing more accurate measurement of bowel diameter[28] 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, submucosal oedema and, with advancing necrosis, intramural gas. Adjacent fat stranding is much less pronounced with ischaemia than with inflammation and may help differentiate between the two. Colonoscopy has been described as a diagnostic test in acute colonic pseudo-obstruction[43]; it has the advantage of being potentially therapeutic (see below).

Management

Treatment of acute colonic pseudo-obstruction depends on the severity of the clinical picture and the perceived risk of imminent ischaemia and perforation. An algorithm illustrating therapeutic options currently available is shown in *Fig. 1*; others have been published previously[19,23,44].

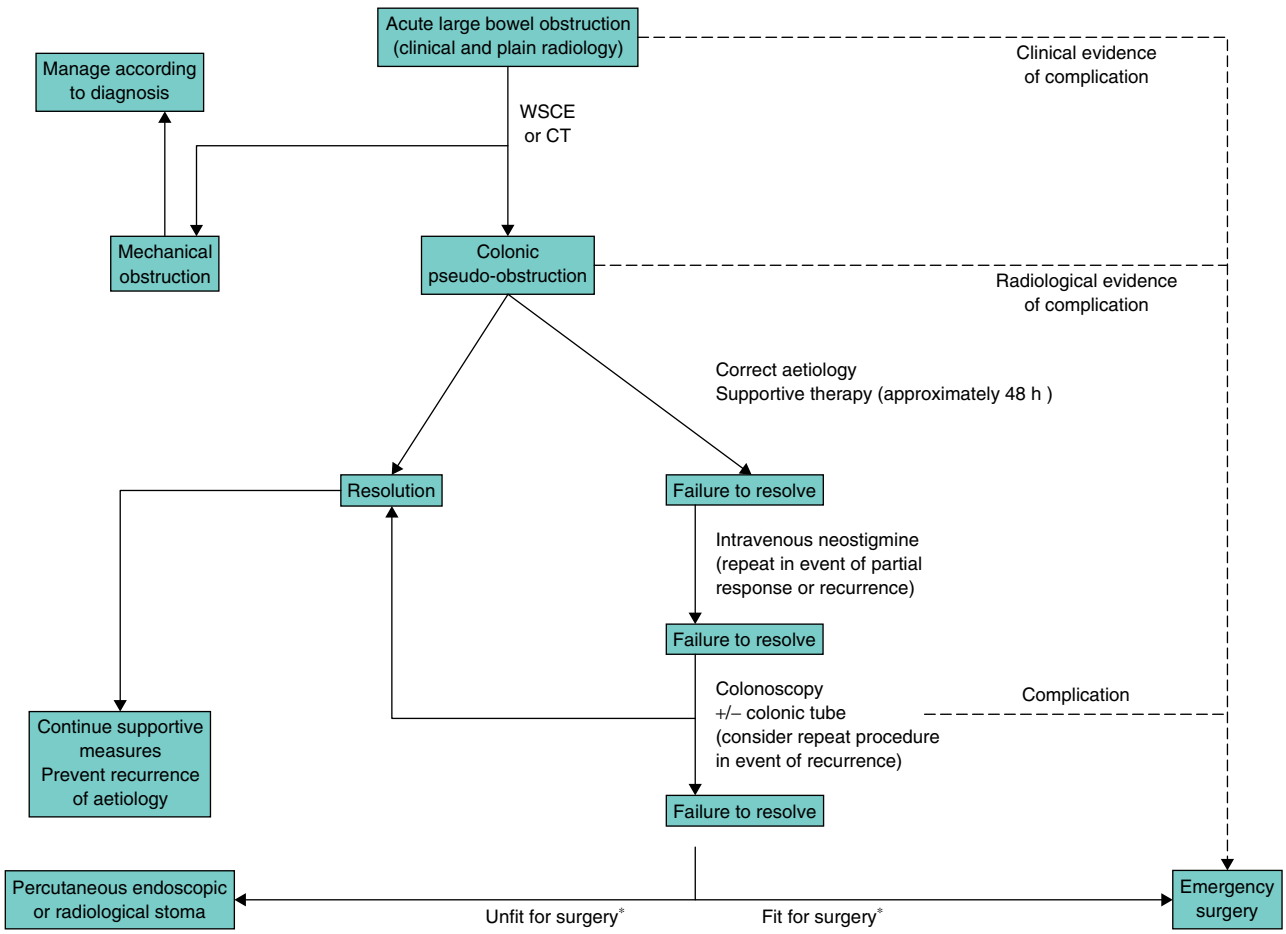

Fig. 1 Algorithm illustrating sequential diagnostic and treatment options for patients with acute colonic pseudo-obstruction. WSCE, water-soluble contrast enema; CT, computed tomography. *No firm evidence can be given for decision making between surgery and percutaneous stoma interventions

Supportive measures

Supportive therapy must always be provided, even in those in whom an invasive approach is immediately necessary. Such measures may range from the basic, such as fasting, intravenous fluid replacement and correction of electrolyte imbalances, especially hypokalaemia and hypomagnesaemia[45–48], to those particular to intensive care. A rectal tube connected to gravity drainage may occasionally be effective as a supportive measure if intermittent diarrhoea is problematic in a bed-bound patient. All drugs delaying gut motility (such as opiates, anticholinergics and calcium-channel blockers) should also be avoided. Laxatives, particularly osmotic compounds such as lactulose, are contraindicated as they may promote colonic bacterial fermentation, thereby increasing gas production.

The limit of purely supportive measures cannot be stated exactly but in most situations should not exceed 48–72 h[19,22,23]; a duration of 6 days has been shown to lead to greater risk of complication[38]. If during this time overt tenderness in the right iliac fossa develops, radiological signs of gross caecal distension, and clinical and biochemical features of sepsis, should be sought urgently. The exact 'at risk' caecal diameter is commonly cited to be 12 cm. However, some with diameters smaller than 10 cm can perforate and those larger than 16 cm may recover spontaneously[39]. In the largest, albeit retrospective, analysis of 400 patients, a twofold increase in mortality was observed when the caecal diameter was at least 14 cm[17]. For this reason, serial radiographs and blood tests should be performed every 12–24 h[49]. Success rates as high as 96 per cent have been achieved with conservative measures[49], but others report poorer results[50]. If conservative therapy is not clearly failing and there are no complications, the next step is pharmacological or colonoscopic decompression.

Pharmacological therapy

Based on the concept of parasympathetic dysfunction, intravenous neostigmine has been tested in controlled trials[51] and remains the mainstay of treatment. No other agent has been tested in a controlled fashion. Neostigmine is a reversible acetylcholinesterase inhibitor that increases the activation of muscarinic receptors by preventing the breakdown of acetylcholine, thus promoting colonic motor activity and intestinal transit[21,52,53]. Oral administration of neostigmine is not recommended in acute colonic pseudo-obstruction because of its erratic absorption in the gastrointestinal tract[21].

Three placebo-controlled double-bind randomized trials have documented the effectiveness of neostigmine[51,54,55] (*Table 2*). Ponec and colleagues[51] recorded that ten of 11 patients receiving the drug intravenously showed marked clinical and radiological improvement, compared with ten patients in the placebo group who had no response. Open-label neostigmine administration to placebo-treated patients resulted in effective decompression in seven. Similar results were obtained in a further randomized controlled trial of 20 patients[54]. Van der Spoel and co-workers[55] performed a trial in patients in intensive care. Thirteen of 24 patients received neostigmine (0·4–0·8 mg/h continuous intravenous administration over 24 h); 11 of the 13 passed stools compared with none of 11 in the placebo-treated group. After 24 h, the non-responders were given either neostigmine or placebo in a cross-over fashion. Eight of 11 patients passed stools *versus* none in the placebo group. Overall, 19 of 24 neostigmine-treated patients passed stools. No major side-effects were recorded during this study[55].

Most non-randomized studies show similar success rates of around 80 per cent (*Table 2*)[41,50,56–62]. Two studies have investigated possible predictive factors of response to neostigmine. In one of these, Loftus and colleagues[50] enrolled a total of 151 patients and found that predictors of sustained response included female sex, advanced age, absence of postoperative status and minimal opioid use. In contrast, a smaller study of 27 patients suggested that neostigmine responders were more likely to be postoperative patients (11 of 15), without electrolyte imbalance and not taking antimotility drugs[41].

Although neostigmine can be regarded as an effective and inexpensive tool with which to induce colonic decompression in acute colonic pseudo-obstruction, its use is not devoid of untoward effects (*Table 3*). Serious side-effects include bronchospasm, bradycardia and hypotension, potentially leading to syncope. During infusion, therefore, the vital signs and electrocardiogram should be monitored, with medical support immediately on hand. If bradycardia is severe, atropine should be administered promptly. Risk can be reduced by using intravenous infusion rather than bolus administration, or starting with a dose of 1 mg instead of 2 mg[22]. Overall, the benefits derived from one or two doses of neostigmine in patients with acute colonic pseudo-obstruction largely outweigh the risks related to administration. Nevertheless, caution with neostigmine is needed in patients with a history of myocardial infarction, active bronchospasm, renal failure (serum creatinine above 3 mg/dl) or who are receiving beta-blockers (*Table 4*)[22,23,25].

Table 2 Studies using neostigmine in the treatment of acute colonic pseudo-obstruction

Reference	Year	Study design	No. of patients	Dose and duration of infusion	Success on first dose (%)	Recurrence rate (%)	Success on second dose (%)	Overall long-term response rate (%)
Hutchinson and Griffiths[56]	1992	Prospective	11	2 mg 1 min*	73	—	—	—
Stephenson *et al.*[57]	1995	Prospective	12	2·5 mg 1–3 min	93	17	100	100
Turégano-Fuentes *et al.*[58]	1997	Prospective	16	2·5 mg 60 min	75	—	—	81
Ponec *et al.*[51]	1999	Prospective RCT	11	2 mg 3–5 min	91	27	—	64
Amaro and Rogers[54]	2000	Prospective RCT	20	2 mg 3–5 min	94 (17 of 18)	27	—	89 (16 of 18)
Paran *et al.*[59]	2000	Prospective	11	2·5 mg 1 h	64	22	100	82
Trevisani *et al.*[60]	2000	Retrospective	28	2·5 mg 3 min	93	8	100	93
Van der Spoel *et al.*[55]	2001	Prospective RCT	13	0·4–0·8 mg/h over 24 h	85	0	—	85
Abeyta *et al.*[61]	2001	Retrospective	10	2-mg bolus	60	0	75	90
Loftus *et al.*[50]	2002	Retrospective	18	2 mg 3–5 min	89	31	—	31
Mehta *et al.*[41]	2006	Prospective	19	2 mg 15 min	84	38	83	79
Sgouros *et al.*[62]	2006	Prospective	25	2 mg 3–5 min	88	23	40	88

Adapted from reference 20. *After guanethidine (20 mg in 100 ml saline solution) had been infused intravenously over 40 min. RCT, placebo-controlled randomized double-blind trial.

Table 3 Side-effects of acetylcholinesterase inhibitors

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

Table 4 Relative contraindications to use 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 > 3 mg/dl

Other potential therapies for acute colonic pseudo-obstruction have not met with as much success. 5-Hydroxytryptamine type 4 (5-HT_4) receptor agonists induce prokinetic effects through acetylcholine and tachykinin release from excitatory myenteric motor neurones in human stomach and small bowel[63]. Cisapride had been used successfully in some patients[64,65] but has now been withdrawn from the market because of the rare occurrence of severe cardiac adverse reactions[66]. Newer 5-HT_4 receptor agonists (for example tegaserod, renzapride and prucalopride) have yet to be tested in patients with acute colonic pseudo-obstruction. Macrolide antibiotics such as erythromycin stimulate gut motility, with proven use in the treatment of diabetic gastroparesis[67]. Studies in experimental models have demonstrated that erythromycin mimics the excitatory actions of motilin through its ability to activate motilin receptors. Limitations

include its short half-life, rapid onset of tachyphylaxis, and relative paucity of colonic motilin receptors in humans compared with experimental animals, such as rabbits, in which erythromycin acts as a colonic prokinetic[68]. These features may account for the apparent lack of effect of erythromycin on human colonic motor activity[69] and explain why successful outcomes with erythromycin treatments are described only in case reports[70,71]. Alvimopan, a novel peripherally acting μ-opioid receptor antagonist with modest beneficial effects in patients with postoperative ileus[72], has not been tested for acute colonic pseudo-obstruction.

The administration of polyethylene glycol has been evaluated after initial resolution of colonic dilatation using neostigmine or endoscopic decompression in a randomized controlled trial of 30 patients[62]. Patients were randomized to receive 29·5 g glycol daily or placebo. Glycol therapy resulted in a significant reduction in recurrent caecal dilatation (33 per cent in the placebo group *versus* none in the treatment group), an increase in stool and flatus evacuations, a decrease in caecal and colonic diameter, and reduction in abdominal circumference[62].

Endoscopic decompression

Although the efficacy of colonoscopy as a decompressive measure has not been assessed in randomized trials[54], colonoscopic decompression has been reported to be successful in approximately 80 per cent of patients with acute colonic pseudo-obstruction[73–79]; up to 20 per cent of them may require further colonoscopy owing to recurrence[76–78]. It should be noted that this procedure is both laborious and potentially hazardous, as it is carried out on unprepared bowel. It should only be performed by experienced endoscopists with adequate equipment, such as colonoscopes with large-diameter accessory channels for optimal suctioning and potential guidewire insertion[23]. As little air insufflation as possible must be used to avoid further dilatation, and copious washout and suction are usually required. Instrument blockages and soiling of the endoscopist are common. Considering the risk of perforation of about 2 per cent[18,54,78], it is important to evaluate both the patient's general condition and the degree of colonic dilatation before starting. One clear benefit of colonoscopy is the ability to inspect the condition of the mucosa.

When ischaemia is evident, surgery will probably be required and decompression should be discontinued. For this reason, it is appropriate for colonoscopy to be carried out on the operating table when clinical features suggest a high likelihood that surgery will be required[39]. Whether suspected ischaemia is an absolute contraindication to proceeding with decompression remains a matter of debate according to an international working group[44]. Although described[23,78–81], the need to place a colonic tube at endoscopy is debatable given the high success rates of colonoscopy alone. Nevertheless, tube placement, usually in the right colon using a guidewire and fluoroscopy, is popular in the USA[23,80] and is supported by some evidence. Two non-randomized studies have shown significantly reduced rates of recurrence compared with endoscopy alone. There were none of 11 *versus* four of 11 recurrences in one series[82] and one of 15 *versus* six of 14 in another[83]. Other studies, however, have found no difference in recurrence with or without tube placement[84].

Percutaneous endoscopic colostomy is a minimally invasive procedure that comprises the endoscopically guided insertion of a plastic tube into the caecum or left colon, allowing decompression and irrigation. Only three studies have used this technique in pseudo-obstruction[85–87], with two employing caecostomy[85,86] and one a left-sided colostomy[87]. The larger of the caecostomy series contained six patients, of whom one had a significant complication (peritonitis) while the rest had a successful outcome, with three tubes remaining in place at the time of reporting[86]. The left-sided colostomy series contained five patients with colonic pseudo-obstruction of whom one died from peritonitis; three had a successful outcome with one tube remaining *in situ*. This study contained a total of 31 patients with mixed indications, and overall success rates in the whole series were tempered by a high frequency of complications[87].

Radiological treatment

CT-guided transperitoneal percutaneous caecostomy has been reported using methods similar to those employed for other indications such as faecal incontinence. The procedure has most commonly been used for patients unresponsive to maximal pharmacological and endoscopic therapy, and also considered unfit for surgery. Of four available studies, two are case reports[88,89]; the two series contain only two[90] and five[91] patients. The latter reported few complications and achieved full resolution of colonic dilatation in all patients[91]. Larger studies are needed to assess the efficacy and safety of this procedure, and to compare it with other interventions, including surgical decompression.

Surgery

The surgeon should be cautious about intervening in acute colonic pseudo-obstruction[39]. Operation is indicated only

for realized or imminent perforation, or in patients who have not responded to maximum non-surgical measures. Surgery is associated with high morbidity and mortality rates. This is hardly surprising in such complicated circumstances, generally including pronounced medical co-morbidities. Mortality rates vary between 30 and 60 per cent[17,19,20,92,93], and are much higher than those for patients who avoid surgical intervention[17].

If the colon is viable and without perforation, the favoured surgical option is some form of venting stoma. A caecostomy or an appropriate colostomy may be chosen, although on-table colonic tube placement has also been described[19]. Tube placement has been performed laparoscopically in one case report[94]. Stomas have a relatively low immediate morbidity[39] but they may be associated, despite claims to the contrary[19], with both recurrence of pseudo-obstruction[39] and a longer-term morbidity stemming from a flush proximal stoma that may be impossible to reverse. No comparative data exist to allow a firm recommendation of one type of stoma over another, or of a stoma rather than resection. In the presence of complications, segmental or subtotal colonic resection is indicated, with exteriorization[19] or ileorectal anastomosis. The matter of whether to proceed with surgery in preference to less invasive endoscopic or radiological interventions cannot be answered from the data currently available.

Overview

Acute colonic pseudo-obstruction is a life-threatening condition for which prompt diagnosis and management can limit the occurrence of complications (for example ischaemia or perforation) and reduce morbidity and mortality. A greater understanding of its pathophysiology, with further developments in pharmacological therapy and advances in minimally invasive endoscopic, radiological and surgical technology, will no doubt improve future management.

Acknowledgements

This work was supported partly by grants from the Italian Ministry of University and Research (COFIN Project no. 2007EN8F7T_005) and Ricerca Fondamentale Orientata funds from the University of Bologna to R.De G. R.De G. is a recipient of grants from Fondazione Del Monte di Bologna e Ravenna and Fondazione Cassa di Risparmio di Bologna, Bologna, Italy. C.H.K. is supported by the Higher Education Funding Council for England. The authors declare no conflict of interest.

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