CASE REPORT

Ogilvie's syndrome treated with an emergency laparotomy, right hemicolectomy and end ileostomy

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SUMMARY

Acute colonic pseudo-obstruction (ACPO), or Ogilvie's syndrome, is a rare clinical entity in which there is massive non-toxic colonic dilatation in the absence of a mechanically obstructing lesion. It is an important yet poorly recognised cause of surgical morbidity and mortality occurring typically in elderly patients with multiple comorbidities. ACPO can often be reversed conservatively with colonoscopic or nasogastric decompression. Surgical intervention is seldom necessary. We present a case of Ogilvie's syndrome in which a healthy 76-year-old man developed life-threatening pseudo-obstruction following rib polytrauma after a mechanical fall. Pneumatosis coli was evident radiologically, prompting emergency exploratory laparotomy. Operative findings of serosal tearing and ischaemic colitis necessitated treatment with right hemicolectomy and ileostomy formation. Microbiological and histopathological analyses proved negative for inflammatory, obstructive and infectious colitides. The case emphasises the importance of early recognition and timely intervention in the management of this rare yet potentially fatal cause of megacolon.

BACKGROUND

Acute colonic pseudo-obstruction (ACPO), also referred to as hypoganglionosis, chronic adynamic ileus, pseudo-Hirschsprung's disease, megacystis-microcolon-intestinal hypoperistalsis syndrome or Ogilvie's syndrome, is a rare clinical entity in which there is gross non-toxic distension of the colon in the absence of a clear mechanically obstructing lesion.¹ It is considered a disorder of intestinal motility, resulting in impaired propulsive function and eventual megacolon. Without timely and appropriate intervention, progression to the much feared complications of bowel ischaemia and visceral perforation with peritonitis may occur. Consequently, ACPO is associated with significant morbidity and is potentially fatal.²

As ACPO is a diagnosis of exclusion, sporadic in occurrence and under-reported, it is not surprising that certain epidemiological uncertainties exist, with true disease prevalence unknown. The stereotypical patient with ACPO possesses multiple chronic diseases, often neurological or with a contributory neuropathy, and tends to be a frail and/or elderly care home resident or hospital inpatient. Literature sources quote a mean age of onset of 64–74 years.³ The

exact mechanisms underlying the pathogenesis of ACPO remain poorly defined with onset associated with a diverse array of factors ranging from acute physiological insult (operative and non-operative) to chronic systemic disease, certain pharmacological agents and metabolic disturbances (table 1). In an analysis of a cohort of 1027 patients with ACPO, Wegener and Börsch found ACPO was most frequently linked to postoperative conditions; it is unusual to encounter Ogilvie's syndrome secondary to isolated events of non-surgical trauma in a well, non-comorbid patient (table 2).³

We outline a case of the sparsely encountered Ogilvie's syndrome, in which a patient presents with ACPO 2 weeks following a singular event of thoracic trauma with left-sided rib fractures after a simple mechanical fall. While the literature recognises non-surgical thoracic trauma as a predisposing factor for ACPO onset, this case retains unusual and noteworthy deviations from the classical ACPO presentation; the patient was non-comorbid, defying the typical demographics one associates with the syndrome. Additionally, the patient required emergency laparotomy, right hemicolectomy and fashioning of an end ileostomy. As previously stated, the pseudo-obstructed patient seldom requires surgical management to achieve cure. Finally, non-surgical thoracic trauma, a rare precipitant of ACPO, was the primary mechanism underlying the patient's presentation. The case report aims to raise awareness of Ogilvie's syndrome, highlights the ease at which the diagnosis may be overlooked and encourages the reading clinician to consider the syndrome as a potential differential even in the non-stereotypical patient presenting with obstructive symptoms and peritonitis.

CASE PRESENTATION

A 76-year-old Caucasian man with a virgin abdomen and no other medical comorbidities bar essential hypertension presented to local orthopaedic services with thoracic trauma from a simple mechanical fall. Six left-sided rib fractures were sustained and were managed conservatively. The patient was admitted under the orthopaedic team for 4 days in total during which time the abdomen was not routinely assessed. However, bowels were open prior to discharge, with no change in function noted. One week following discharge from orthopaedics, the

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Table 1 Summary of all known conditions associated with ACPO onset

Predisposing aetiological category for ACPO	ACPO inciting agents/events in aetiological category
Surgical	Orthopaedic surgery (hip/knee replacement, spinal surgery), postoperative immobility, organ transplantation
Cardiorespiratory	Cardiogenic shock, MI, congestive cardiac failure, severe COPD
Neurogenic	Stroke, spinal cord injury, Parkinson's disease and Parkinson-plus syndromes, vascular/Lewy body/Alzheimer's dementia, multiple sclerosis
Metabolic	Electrolyte imbalance (hypokalaemia, hyponatraemia, hypocalcaemia, hypomagnesaemia), diabetes with autonomic neuropathy, renal failure, hypothyroidism, hypoparathyroidism
Medications	Opiates, immunosuppression, chemotherapy (Vinca alkaloids), anti-Parkinson's
Oncological	Paraneoplastic disorder of small cell carcinoma of lung, paediatric haematological malignancy, allogenic stem cell transplantation
Obstetric	C-section
Infectious	Epstein-Barr virus, cytomegalovirus, varicella-zoster, herpes simplex viraemias
Autoimmune	Systemic lupus erythematosus, myasthenia gravis
Other	Major burns, trauma (non-surgical intra-abdominal, retroperitoneal, extra-abdominal), amyloidosis, mitochondrial disorders, sickle cell disease, idiopathic

ACPO, acute colonic pseudo-obstruction; COPD, chronic obstructive pulmonary disease; MI, myocardial infarction.

patient was readmitted under the general surgeons as an emergency complaining of generalised abdominal pain, absolute constipation and vomiting. Stool habits prior to presentation were unremarkable with no diarrhoea or progressive constipation. On examination, the patient was afebrile, haemodynamically stable and had marked distension of the abdomen. The patient was in sinus rhythm. Palpation of the abdomen revealed generalised tenderness with localised involuntary guarding and rebound tenderness in the right lower quadrant and no palpable masses or organomegaly. Tympanic resonance was noted on percussion and bowel sounds were auscultated. Hernial orifice and digital rectal examinations proved unremarkable.

History and clinical examination were in keeping with obstruction prompting immediate investigations of full blood count, C-reactive protein, urea and electrolytes, venous gases and abdominal X-ray (table 3) (figure 1). Microbiological analysis of the stool including *Clostridium difficile* screening was also conducted later after admission

Table 2 Predisposing aetiologies for ACPO ranked in terms of commonality. Note non-surgical trauma is the rarer aetiological category implied in ACPO onset. This was the underlying precipitant identified in our case

Aetiological category	Association incidence of aetiological category with ACPO (%)
Postoperative conditions	23
Cardiopulmonary disorders	17.5
Systemic disorders	15
Non-surgically induced trauma	11

ACPO, acute colonic pseudo-obstruction.

Table 3 Relevant biochemistry/haematology results

Parameter	Value (normal range)
CRP	79 (<4.0 mg/dL)
Lactate	1.2 (0–2 mmol/L)
White cell count	13.3 (4.0–11.0×10 ⁹ /L)

CRP, C-reactive protein.

as absolute constipation was present at initial assessment and samples could not be obtained. A nasogastric tube was passed and drip and suck regime commenced in an attempt to decompress the bowel conservatively and analgesia was administered.

Serum lactate was normal on venous gas analysis. Leucocytosis and elevated acute phase reactants were noted and the patient was started on intravenous co-amoxiclav and metronidazole to cover for potential intra-abdominal sepsis. Plain abdominal film showed prominent dilatation of the caecum in keeping with closed loop large bowel obstruction. Renal function and urea and electrolytes were normal thereby ruling out electrolyte imbalance and thus a chemical ileus as a potential cause for clinical and X-ray findings. Only a matter of hours after admission, conservative decompressive strategies failed. The patient clinically deteriorated, becoming hypotensive and tachycardic. This prompted aggressive fluid resuscitation and an emergency CT scan of the abdomen (figure 2). CT abdomen revealed gross caecal dilatation at 10.5 cm with concurrent small bowel distension. Of particular concern was the finding of linear pneumatosis coli within the caecum progressing through the ascending colon to the distal aspect of the hepatic flexure, indicative of visceral ischaemia and impending perforation. There was no evidence of acute mesenteric vessel thrombosis or an obstructing mechanical lesion within the colon to account for the clinical picture. The decision to abandon conservative and medical management and undertake emergency exploratory laparotomy was taken due to clinical peritonitis and radiological features of imminent visceral rupture. Laparotomy mirrored CT findings; massive caecal

Figure 1 Plain abdominal film of the patient showing prominent small bowel loops and colonic distension with marked caecal dilatation; features consistent with large bowel obstruction with a patent ileocaecal valve.

Figure 2 CT abdomen demonstrating right colonic and small bowel dilatation. No mucosal lesion or mass was present to account for the transition point at the splenic flexure. Pneumatosis coli is visualised in the caecum and ascending colon reflecting localised bowel ischaemia. No associated thrombus was present in the superior mesenteric artery.

distension and macroscopic ischaemic colitis extending to the hepatic flexure with superimposed serosal tearing were visualised. There was general distension of the small bowel,

transverse colon and sigmoid; however, these structures remained viable. There was a trace of free fluid within the pelvis. No luminal, mural or extramural obstructing colonic lesion was identified at surgery. A right hemicolectomy was performed, with the non-viable terminal ileum and proximal transverse colon resected and sent for histopathological analysis. An end ileostomy was fashioned and the patient was transferred to recovery.

Microbiology and histopathology data were available posthemicolectomy. Stool cultures including *C. difficile* screen proved negative for infective colitis as a potential precipitant of toxic megacolon. Additional virology screening for HIV, hepatitis B and hepatitis C was conducted with patient consent due to the unexplained aetiology of the obstruction. Viral serology was also negative. Similarly, histopathological analysis of the resected specimens was unable to identify a cause for obstruction. Macroscopic evaluation of the terminal ileum, caecum, ascending colon and appendix was normal. The resected terminal ileum and caecum were dilated to a maximum diameter of 10.5 cm and there were areas of focal haemorrhage within the mucosa. A 2.5 cm area of ulceration within the ileocaecal valve was identified and analysed further with microscopy. Microscopic evaluation of the ileocaecal ulceration and ascending colon confirmed acute ischaemic necrosis of the mucosa and submucosa and submucosal oedema and haemorrhage, respectively. The histopathological conclusion was that all resected specimens showed focal acute ischaemic necrosis

Figure 3 Management algorithm for acute colonic pseudo-obstruction (adapted from Amaro and Rogers¹⁰). NPO, nothing by mouth (nil per os); PEG, percutaneous endoscopic gastrostomy.

only—there was no evidence of mechanical, infective or inflammatory colitides to explain the patient's presentation. From amalgamation of radiological, microbiological and histopathological data, it was agreed that the patient had indeed presented with Ogilvie's syndrome, defying the usual demographic stereotype and atypically failing to respond to simple conservative management.

DIFFERENTIAL DIAGNOSIS

The differentials considered throughout this case included:

- ▶ dynamic (mechanical) bowel obstruction: mural (malignant/inflammatory stricture), extramural (congenital bands/adhesions, volvulus) or intraluminal (colorectal carcinoma) infective colitides and inflammatory bowel disease
- ▶ adynamic (functional) bowel obstruction: ileus, acute mesenteric arterial occlusion/mesenteric vein thrombosis or ACPO.

OUTCOME AND FOLLOW-UP

The patient was discharged 5 days after surgery having made a positive recovery. His ileostomy was pink and healthy. He is currently reviewed regularly in surgical outpatients and it is the intention that he will undergo further laparotomy for reversal of stoma with ileocolic anastomosis in due course.

DISCUSSION

The first description of ACPO was put forth in 1948 by Sir Heneage Ogilvie (1887–1971) in which he described acute bowel obstruction 'in the absence of any organic disease of the colon' in two patients with large oesophageal hiatus tumours. Ogilvie himself attributed ACPO to 'sympathetic deprivation'⁴; the interruption of sympathetic outflow a result of mass effect of the hiatal tumours upon the sympathetic chain.

However, in contrast to Ogilvie's postulations, modern consensus is that ACPO is secondary to dysregulation of both the sympathetic and parasympathetic supply to the enteric nervous system rather than sympathetic deprivation alone. Dysregulation of autonomic supply to the Auerbach and myenteric plexus precipitates failure of the physiological smooth muscle contractions of the small bowel and colon, thereby impairing normal gut peristalsis and predisposing the individual to ACPO.⁵ This proposed 'neurogenic' mechanism of pathogenesis helps to account for the frequent association of ACPO with primary neurological disease and comorbidities in which sensory, motor and autonomic neuropathy is a dominant component. However, it fails to satisfactorily explain the prevalence of ACPO in other chronic systemic inflammatory disorders, autoimmunity and paraneoplastic syndromes. This affiliation may be explained by the presence of high antineuronal and anti-calcium channel antibodies generated as a product of such disease processes.⁵ Furthermore, the production of anti-smooth muscle antibodies observed in autoimmune and paraneoplastic phenomena causes visceral autonomic myopathy of the small bowel and colonic smooth muscle, leading to dysregulated peristalsis and predisposition to ACPO.⁶ Although an unusual cause, there are cases in which ACPO has been reported as a direct result of non-surgical thoracic trauma alone. Not surprisingly, no pathogenic model has been yet elucidated in the case of this unusual but recognised precipitant.

Regardless of the underlying aetiology, appropriate management of patients with ACPO includes early

recognition and prompt intervention with either conservative, medical and/or surgical measures.⁷ In evaluation of the current evidence, Rex⁸ calculated the risk of visceral perforation in ACPO to be low at ~3% and thus need for surgical intervention is unusual. However, when perforation does occur, mortality rate is high at approximately 40%.⁹ This presents the surgeon with a difficult clinical decision; does one persevere with medical and conservative therapies or intervene invasively with emergency surgery? Recent guidelines from the American Society for Gastrointestinal Endoscopy¹⁰ outline management algorithms to aid the clinician in the management of suspected pseudo-obstruction scenarios (figure 3).

In the unusual clinical situation in which patients with ACPO demonstrate signs of peritonism or potential visceral perforation, as in our case, urgent surgical intervention is indicated. Surgical options include segmental or subtotal colonic resection with either exteriorisation or primary anastomosis. In practice, the majority of patients will respond to conservative colonoscopic/nasogastric decompression with concurrent intravenous fluid administration and discontinuation of medications that perpetuate colonic dysmotility. In those failing conservative interventions who remain stable with no clinical or radiological evidence of perforation, medical management may be trialled. The most convincing medical therapy for ACPO involves the administration of neostigmine, a reversible acetylcholinesterase inhibitor that enhances colonic propulsion and accelerates transit time through stimulation of the parasympathetic nervous system.⁷ In a randomised controlled trial, Amaro and Rogers¹⁰ reported a statistically significant benefit of neostigmine compared with placebo with the mechanism thought to be closely related to Ogilvie's original pathophysiology proposal. Colonic decompression may also be considered when neostigmine is ineffective and may be particularly useful in the comorbid patient unsuitable for open surgery.

Learning points

- ▶ Non-surgical thoracic trauma is a rarer, and thus easily overlooked, precipitant of Ogilvie's syndrome.
- ▶ The assessing clinicians should factor acute colonic pseudo-obstruction (ACPO) into their differential diagnoses when assessing the clinically obstructed elderly patient with multiple comorbidities.
- ▶ While pathogenesis is not fully understood, autonomic dysfunction is a significant risk factor for ACPO. The general surgeon should thus have a higher index of suspicion of Ogilvie's syndrome in patients with a background of neurological, chronic inflammatory and autoimmune disease.
- ▶ Ogilvie's syndrome often responds readily to conservative methods. Surgery with colonic resection and stoma formation or primary anastomosis is indicated in unusual incidences in which bowel ischaemia, impending visceral rupture or florid faecal peritonitis is evident.
- ▶ Early detection and prompt appropriate management are critical to minimising morbidity and mortality. Other specialties not accustomed to assessing the abdomen should seek early involvement of the general surgeon should suspicion of pseudo-obstruction arise. However, prompt referral is reliant on our colleagues' recognition of the syndrome.

Contributors AJR was responsible for literature review and compilation of the majority of the manuscript. J-PQ was responsible for conceiving the case study, obtaining approval for publication of the case from the patient, and contributed to editing of the manuscript. AB contributed to composing, reviewing and approving the final manuscript. MF reviewed and approved the final manuscript as the overseeing colorectal consultant surgeon responsible for the management of the patient's case.

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