Review Acute colonic pseudo-obstruction after caesarean section

Authors Aparna Kakarla / Helena Posnett / Asha Jain / Mark George / Alok Ash

Key content:
• In acute colonic pseudo-obstruction (Ogilvie’s syndrome) there is an adynamic ileus without mechanical obstruction, characterised by dilatation of the caecum and right colon.
• In obstetrics it is usually associated with caesarean section.
• Early diagnosis and appropriate treatment are imperative to avoid caecal rupture, faecal peritonitis and the associated high maternal mortality.
• Conservative and pharmacological therapies are effective in many women, but surgical intervention may be required.

Learning objectives:
• To learn to recognise the condition.
• To understand the different management strategies.
• To manage the condition most appropriately for each individual case.

Keywords acute colonic pseudo-obstruction / caesarean section / colonic decompression / maternal mortality / Ogilvie’s syndrome
Introduction

Acute colonic pseudo- obstruction (ACPO), or Ogilvie’s syndrome, was first described by Sir Heneage Ogilvie in 1948. It is an acute surgical condition characterised by massive dilatation of the colon in the absence of mechanical obstruction. It usually occurs in high-risk and seriously ill or postoperative patients. If untreated, massive distension can cause rupture or perforation of the caecum and faecal peritonitis, which are associated with a high mortality.

Incidence and clinical significance

The true incidence of ACPO is unknown as many mild cases resolve spontaneously and no reliable national or international data exist on its frequency. Only the severe form of adynamic ileus presents as ACPO in hospitalised patients and it is associated with a wide variety of medical and surgical conditions (Box 1). There are no data to suggest that the incidence is related to race. ACPO is generally a disease of elderly patients, although it can occur at any age. It has a male: female ratio of 1.5:1.

In young, fit and healthy women of child bearing age, caesarean section seems to be the most common operative procedure associated with this syndrome. It has also been described after vaginal delivery, forceps delivery, caesarean hysterectomy, and during pregnancy with preterm labour, pre-eclampsia and multiple pregnancies.

Overall, however, this condition is relatively rare despite the rising caesarean section rate. It is not known which group of women is predisposed to developing this complication. No parity is immune and there is no correlation between ACPO and indication for caesarean section. It is estimated that approximately 20 patients in the UK die per annum as a result of ACPO. In the last Confidential Enquiry into Maternal and Child Health (CEMACH) report there were four deaths from ACPO, all occurring after caesarean section. In two of the cases there was perforation of the caecum and in one case perforation of the sigmoid colon. In the fourth case there was marked pseudo-obstruction but no perforation; death was attributed to cardiac arrhythmia secondary to associated electrolyte disturbance.

Pathophysiology

The exact pathophysiology of the condition is not clearly understood but most likely involves an imbalance between the sympathetic and parasympathetic colonic innervation. Normally the parasympathetic nervous system increases the contractility of the colon, whereas the sympathetic nerves decrease the motility. The two systems act on the smooth muscles of the bowel, maintaining a balance which promotes normal bowel function.

Ogilvie’ originally attributed the syndrome to sympathetic deprivation. Bachulis and Smith, in a review of 35 patients, suggested a possible role of prostaglandin abnormality. A later analysis of the largest series on the condition suggested a temporary neuropraxia of the sacral parasympathetic nerves S2–4 as the underlying pathology. These nerves pass through the inferior hypogastric plexus in close proximity to the cervix, vagina and broad ligaments, before terminating to supply the left colon, and could be injured during surgery or trauma in this area. This may help explain the occurrence of ACPO in the obstetric/gynaecological setting, but it is likely that the pathogenesis is multifactorial (Box 1). These factors can alter the autonomic regulation of the colon, either by excessive parasympathetic suppression or sympathetic stimulation, or both. This leads to an adynamic distal colon that is similar to Hirschsprung’s disease, except with normal ganglion cells observable on autopsy.

Hence, the obstruction is functional or ‘pseuod’. Recent data indicate that the volatile short-lived gas, nitric oxide (NO), one of the major inhibitory neurotransmitters released by enteric neurones, can be overproduced in some pathological conditions, causing gut dysmotility and dilatation. Volume depletion occurs as bowel contents stagnate in the dilated loops of the pseudo-obstructed bowel, are lost through vomiting, or are sequestered in the bowel wall or transudated into the peritoneal cavity. Water loss is accompanied by electrolyte loss. If the ileocecal valve remains competent, preventing reflux into the ileum, a closed-loop obstruction develops.
The caecum is the part of the colon with the largest diameter. Therefore, a small amount of pressure leads to an enormous increase in its size and wall tension, and hence it dilates more rapidly than the remainder of the colon. If left untreated, it is prone to rupture through the serosa between the taeniae due to direct mechanical stretching. Animal studies and retrospective data suggest a critical threshold diameter of 12 cm for the caecum for rupture, when the intracaecal pressure rises to more than 26 cm H₂O. At this pressure, blood flow in the wall of the bowel is considerably diminished by the prolonged compression of the vessels, which is associated with a risk of ischaemic necrosis and perforation.

The seriousness of this condition is reflected in an overall mortality of approximately 15% with early intervention, which rises to 36–50% when the bowel is perforated or ischaemic. Other factors that significantly influence the mortality rate include: acuteness of onset of the condition; baseline state and associated co-morbidities of the patient; mode of treatment; delay in decompression; complications of surgery; and arrhythmia secondary to electrolyte imbalance. These prognostic factors should be incorporated into decision-making regarding intervention for ACPO.

Clinical features after caesarean section

The woman is usually 2–12 days post caesarean section. Signs and symptoms mimic those of mechanical large bowel obstruction. Most women (80%) have abdominal pain. Initially this manifests as dull cramps with no specific localisation, typical of hollow viscus distension. Bowel movements cease or only a small amount of faecal fluid or flatus is passed. Nausea can be present but vomiting is usually a late symptom. The early features are tachycardia and a raised white cell count in the absence of any sepsis or signs of peritonism. The temperature is usually normal (pyrexia indicates sepsis, bowel necrosis and/or perforation) and progressive abdominal distension is the most typical finding (90–100%). Bowel sounds may vary – they can be hyperactive or normal (40%), high-pitched, tinkling or absent (60%) – depending on the duration. The woman becomes acutely ill with dehydration, oliguria and features of electrolyte imbalance; some women have renal and liver function abnormalities. Localised tenderness in the right iliac fossa over the caecum indicates impending rupture. Laboratory findings are not diagnostic.

Differential diagnosis includes mechanical bowel obstruction, volvulus, bowel perforation, peritonitis and intra- or retroperitoneal bleeding. A plain abdominal x-ray should be taken as this is the most useful diagnostic test to show a typical picture of large bowel dilatation, especially the caecum (Figure 1), which tails off at the splenic flexure or rectosigmoid. Mechanical obstruction can be ruled out by using a water-soluble radiographic contrast enema. In ACPO there will be free flow of the contrast medium to the caecum. Pneumoperitoneum confirms bowel perforation.

Management

Management of the condition should include general measures, drug treatment, endoscopic decompression and surgery. It should involve a multidisciplinary team of an obstetrician/gynaecologist, anaesthetist, gastroenterologist, surgeon, radiologist, midwife/nurse and theatre personnel.

Conservative therapy

In general, initial treatment of caecal dilatation less than 10 cm in diameter consists of a conservative approach, with the woman kept nil by mouth, nasogastric decompression and correction of fluid and electrolyte imbalance. Optimal body positioning and mobilisation out of bed are often advised. However, most women with ACPO typically are not ambulatory. In some women a central venous line may be warranted. In addition to administration of intravenous fluid, this facilitates monitoring of the woman’s haemodynamic status. Fluid replacement is usually in the form of normal saline or Hartmann’s solution, with colloids reserved for those women in severe hypovolaemic shock. Drug therapy should be reviewed: any medication that can adversely affect colonic motility — for example, narcotic analgesics and anti-cholinergic drugs — should be stopped, changed or avoided.
An indwelling urinary catheter helps assess hourly urine output and fluid balance. Blood culture is indicated if sepsis is suspected. Due to depleted blood volume in the first week after a caesarean section these women are at an increased risk of venous thromboembolism (VTE); they should, therefore, be commenced on VTE prophylaxis.

If the condition occurs during pregnancy, the fetal condition should be monitored carefully and antenatal corticosteroids administered if the gestation is less than 34 weeks. ACPO by itself is not an indication for delivery by caesarean section. Rieger et al.26 and Imai et al.27 describe two cases during pregnancy that were successfully managed by conservative therapy. Both women underwent caesarean sections later for obstetric indications but without any recurrence of the condition after the operation.

Conservative therapy can be tried for 24–48 hours before pharmacological or endoscopic intervention. The reported success of conservative management alone is variable, with rates from 20–92%.3 The direct benefits of any individual component of conservative care are, however, unknown because these measures have not been studied as single interventions.

**Pharmacological therapy**

A variety of pharmacological agents, for example, erythromycin, metoclopramide and cisapride (which has now been withdrawn), have been tried for active reversal of ACPO with inconsistent results. The only consistently positive results have been with neostigmine (Table 1).26–30 In particular, a recent prospective, double-blind, placebo-controlled trial27 has shown promising results: 50% of patients had an immediate clinical response within a median time of four minutes (range 3–30 minutes) in the treatment group compared with no response in the placebo group ($P < 0.001$); and 73% had a sustained response three hours later in the treatment group compared with 20% in the placebo group ($P = 0.04$). However, this study is too small to be significant for the risk of colonic perforation and mortality.

**Neostigmine**

Treatment consists of an intravenous injection of 2.5 mg neostigmine administered over a period of 3–5 minutes. The response is noted after 30 minutes and again after three hours. An immediate clinical response is defined as the passage of flatus with a reduction of abdominal distension on physical examination within 30 minutes. A sustained response is measured by a reduction in abdominal girth and colonic diameter on abdominal x-rays three hours after the injection. The rationale for using neostigmine is to restore the ‘normal’ autonomic balance by inhibiting acetylcholinesterase activity to facilitate impulse transmission across myoneural junctions in the bowel wall. Side effects include excessive salivation, vomiting, bronchoconstriction, symptomatic bradycardia, hypotension and syncope.26,28 Hence, such treatment must be accompanied by close monitoring of cardiorespiratory status and atropine must be available as an antidote for these muscarinic adverse effects.

Prior to neostigmine therapy, a mechanical bowel obstruction must be ruled out. In addition, women with the following are not considered suitable for medical treatment:37

- baseline pulse rate of less than 60 beats per minute
- systolic blood pressure of <90 mm Hg
- clinical and radiological signs of bowel perforation
- active bronchospasm requiring treatment
- prior treatment with prokinetic drugs, for example metoclopramide, less than 24 hours before evacuation
- active gastrointestinal bleeding
- serum creatinine level of >260 μmol/l
- documented hypersensitivity to neostigmine.

Neostigmine has a short half-life. Therefore, it is essential to continue with the general conservative treatment in order to maintain a sustained improvement after the immediate response. The treatment is considered to have failed if there is no response after three hours and decompression should be undertaken promptly in these women.

Other acetylcholinesterase inhibitors (for example, pyridostigmine and physostigmine) are not widely used: intravenous formulations of pyridostigmine are not always available and physostigmine easily crosses the blood–brain barrier.38 A selective peripheral opioid receptor antagonist (ADL 8–2698) has been shown to achieve more rapid return of flatus and bowel movement and

<table>
<thead>
<tr>
<th>Study</th>
<th>Neostigmine dosage and administration (IV = intravenous)</th>
<th>Number of patients</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hutchison and Griffiths27</td>
<td>2.5 mg IV over 1 minute</td>
<td>11</td>
<td>Decompression in eight patients</td>
</tr>
<tr>
<td>Stephanieen et al.29</td>
<td>2.5 mg IV over 1–3 minutes</td>
<td>12</td>
<td>Decompression in 12 patients, two patients required two doses</td>
</tr>
<tr>
<td>Turegano-Fuentels et al30</td>
<td>2.5 mg IV over 15 minutes</td>
<td>16</td>
<td>Decompression in 12 patients</td>
</tr>
<tr>
<td>Perez et al.31</td>
<td>2.5 mg IV over 40 minutes</td>
<td>11</td>
<td>Decompression in eight patients after first dose; in two patients after second dose. No response in one patient</td>
</tr>
<tr>
<td>Trevisani et al.32</td>
<td>2.5 mg IV over 60 minutes</td>
<td>28</td>
<td>Decompression in 28 patients</td>
</tr>
<tr>
<td>Abeyta et al.33</td>
<td>2.0 mg IV (bolus)</td>
<td>8</td>
<td>Decompression in six patients after first dose; in two patients after second dose.</td>
</tr>
</tbody>
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earlier hospital discharge with no increased pain. However, this drug is not widely available. New colokinetic agents, including 5-HT(4) receptor agonists and motilides (motilin receptor agonists), may represent other useful medical therapeutic options to treat ACPO.

**Endoscopic therapy**

Colonoscopic decompression was first used to treat this condition in 1977. Its use has been reported in several series, totalling many hundreds of patients who do not respond to, or who relapse after neostigmine, although its efficacy has not been established in randomised clinical trials. Among the series with more than 20 cases, the reported success rate varied from 61–78%. However, because this procedure is usually carried out as an emergency on an unprepared bowel, it can be technically difficult to ensure patency of the suction channel of the scope. It can fail because of refractory caecal dilatation and can precipitate bowel perforation (in up to 3% of patients). The
condition may recur in up to 40% of patients after initial decompression. Other treatments, for example caecostomy, have been used to provide colonic venting. However, the optimal caecostomy technique has not been established and it has its own complications, for example: pressure necrosis from the external bumper of the tube, development of profuse granulation tissue, cellulitis of the abdominal wall and sepsis.

Open surgery

Open surgery is mandatory when perforation or ischaemia are suspected. It usually involves bowel resection with temporary diversion, requiring a second stage closure. Stoma care forms an important part of care after resection surgery. Surgical management carries greater morbidity. Potential complications include abscess formation, ileus and bleeding.

Aftercare

Although ACPO is rare in obstetric practice it has serious consequences in terms of maternal morbidity and mortality. Any woman with abdominal distension in the early puerperium, particularly after a caesarean section or vaginal delivery, should always be carefully evaluated to rule out this condition. These young and apparently healthy mothers are psychologically traumatised by this unexpected complication after childbirth. They require adequate explanation of the condition, on repeated occasions if required, by a senior obstetrician and surgeon. Emotional support is vital, as is proper training for stoma care if a laparotomy with a surgical resection is required. The woman and her family should be allowed enough time to settle down and reflect, and a trained stress counsellor may be helpful. Appropriate follow-up should be arranged with the consultant obstetrician and the surgeon. At present there are insufficient data to comment on or predict any recurrence of the condition in future pregnancies and childbirth.

Summary

ACPO is a well-documented condition, which presents with features of large bowel obstruction without a mechanical cause. Obstetricians should be aware that this rare complication usually occurs after caesarean section and can lead to serious maternal morbidity and mortality, mainly due to caecal perforation and/or ischaemia. Careful assessment of the gastrointestinal state of a woman who has had a caesarean delivery is essential (Figure 2). Plain abdominal x-ray is the most useful diagnostic test. ACPO should initially be treated conservatively with correction of metabolic, electrolyte and fluid imbalance. Most women respond to pharmacological therapy with neostigmine. When neostigmine is unsuccessful or contraindicated, a decompressive procedure, typically by colonoscopy, should be employed. Open surgery is mandatory if caecal rupture or necrosis is suspected. An early diagnosis and timely intervention is essential to avoid caecal rupture and its associated high maternal mortality rate. Proper explanation, adequate emotional support and appropriate follow-up by a senior obstetrician are vital in the management of the condition.

References

3 Carpentari S, Holstrom B. Ogilvie Syndrome. [www.emedicine.com/med/topic2691.htm].


