



Ogilvie's syndrome after an emergency caesarean section: A case report

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ABSTRACT

Ogilvie's syndrome, or acute colonic pseudo-obstruction, is a rare presentation occasionally observed post-partum, particularly following caesarean sections. Challenges in diagnosis often lead to delays in initiation of treatment, which significantly increases complications, including caecal ischaemia, perforation, sepsis and death. This case report describes the development of Ogilvie's syndrome within 24 h of an emergency caesarean section which was promptly recognised and confirmed by computed tomography, which demonstrated caecal dilatation of 9.7 cm without evidence of mechanical obstruction. An elevated level of C-reactive protein of 320 mg/L raised early clinical suspicion of caecal ischaemia, and this was managed endoscopically. Fortunately, the patient did not have any significant complications despite the high morbidity and mortality rate associated with Ogilvie's syndrome, highlighting the importance of clinician awareness and early initiation of management.

1. Introduction

Ogilvie's syndrome is a rare complication which can have significant complications, and has been observed to develop following caesarean sections without specific identifiable risk factors [1]. The overall prevalence is estimated to be 1 in 1000 admissions annually [2]. It can be challenging to identify and distinguish from other causes of post-operative abdominal pain [3], with resulting delays to diagnosis and treatment therefore increasing morbidity and mortality. There is a low level of general clinician awareness about Ogilvie's syndrome, which can prolong the time from patient development of symptoms to senior clinician review and involvement of a multidisciplinary team [4]. This is particularly concerning as the mortality rate has been observed to be as high as 45% [5] and is strongly associated with increased time to diagnosis [6]. The rates of Ogilvie's syndrome are likely to increase with the increasing rates of caesarean sections globally [7], with 21% of all births currently via caesarean section [7].

We present a case of Ogilvie's syndrome developing after an emergency caesarean section with early recognition enabling escalation of care to senior clinicians and multidisciplinary team involvement. Despite initial response to conservative management, elevated inflammatory markers raised concern for caecal ischaemia. An emergency endoscopic decompression was required, following which the patient made a full recovery.

2. Case Presentation

This case study describes the clinical course of a 45-year-old woman in her fifth pregnancy receiving antenatal care in a hospital doctor's clinic. She had had four previous caesarean sections and was planned for an elective caesarean section at 38 weeks of gestation secondary to suspicions of placenta accreta on magnetic resonance imaging (MRI). Her current pregnancy was complicated by insulin-dependent gestational diabetes, an elevated body mass index (BMI) of 41.3 kg/m² and recurrent presentations with small-volume antepartum haemorrhage. The patient's latest obstetric ultrasound, at 34 weeks of gestation, demonstrated a foetus with an estimated weight on the 66th percentile and normal biophysical profile.

The patient presented to the birthing unit at 36 + 5 weeks of gestation with contractions and 150 mL antepartum haemorrhage necessitating an emergency caesarean section under general anaesthetic. She had a midline laparotomy and fundal uterine entry, with no intraoperative findings of placenta accreta. She proceeded to an uncomplicated bilateral salpingectomy and supraumbilical hernia repair with a total estimated blood loss of 400 mL. Postoperatively she received fentanyl via patient-controlled analgesia and venous thromboembolism prophylaxis with enoxaparin and calf compressors.

Day 1 postoperatively, the patient tolerated a normal diet and oral hydration; however, she had not passed flatus or opened her bowels and was unable to mobilise secondary to pain. She experienced generalised lower abdominal pain and examination revealed a soft distended

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abdomen, audible bowel sounds and no clinical signs of peritonism. Her patient-controlled analgesia was changed to oral opioid analgesia on day 2, and 12 h following this she experienced severe worsening of her abdominal pain. Clinical review raised concerns for bowel obstruction, with the patient not having passed flatus postoperatively and worsening abdominal distension with new palpation tenderness.

A computed tomography (CT) scan of the abdomen and pelvis demonstrated severe adynamic ileus with dilatation of the small bowel (3.4 cm), caecum (9.7 cm), ascending colon (7.5 cm) and transverse colon (7.1 cm). No clear transition point was identified; however, there was gradual tapering from the splenic flexure distally. There were no signs of pneumoperitoneum and no other intraabdominal abnormalities. The clinical and radiographic findings were consistent with Ogilvie's syndrome.

The general surgery team were consulted, and the patient underwent conservative management with a nasogastric tube insertion, fluid balance monitoring, and administration of prokinetics and fleet enemas. She responded well, with an improvement in her abdominal pain and decreasing distension.

There was consideration of medical management with neostigmine; however, biochemistry demonstrated a C-reactive protein (CRP) of 320 mg/L, raising concerns for ischaemic necrosis of the caecum and increased risk of perforation. This prompted an urgent endoscopic decompression and placement of a rectal tube. The procedure revealed no evidence of mucosal ischaemia. The patient's abdominal pain and distension improved significantly, after which she was able to tolerate oral intake and pass flatus following removal of the rectal tube. Her CRP decreased from 320 to 161 mg/L on day 1 after the bowel decompression. The patient continued to recover well and was discharged four days later.

3. Discussion

Ogilvie's syndrome is a rare postoperative complication characterised by caecal and colon dilatation without mechanical or anatomical obstruction [8]. Its aetiology is not well understood and is hypothesised to be secondary to parasympathetic dysfunction [6] resulting in erratic peristalsis and progressive dilatation [9]. This is potentially aggravated by the gravid uterus, which may compress the sacral parasympathetic plexus supplying the colon [5,10]. Delay to diagnosis and treatment increases risks of complications, including bowel ischaemia and perforation, which is associated with a significant increase in morbidity and mortality [1].

Whilst gynaecological surgery and caesarean sections account for an estimated 10% of cases [10] of Ogilvie's syndrome, it is also associated with major orthopaedic surgery as well as hospitalised geriatric patients with poor functional status and significant comorbidities [8]. Given the rising caesarean section rates observed globally and within Australia [7,11], it is likely that Ogilvie's syndrome will become increasingly prevalent. This makes it especially important for healthcare workers to be aware of the clinical presentation, to be able to distinguish Ogilvie's syndrome from other causes of post-caesarean abdominal pain as well as enabling early diagnosis and treatment [4].

A challenge faced in the diagnosis of Ogilvie's syndrome postpartum is the lack of identifiable risk factors. Small-scale observational data reveal that it is more common after caesarean section than after vaginal delivery, and following emergency caesarean compared with elective caesarean, and the indications for caesarean section for patients who develop Ogilvie's syndrome most commonly include pre-eclampsia, HELLP syndrome, multiple pregnancy and antepartum haemorrhage [1]. Currently there is insufficient evidence indicating strong risk factors, and thus there is a need for high awareness of the possibility of Ogilvie's syndrome in all postpartum patients.

Additionally, the initial patient symptom is usually that of abdominal pain and distension [1], which can be mistaken for more common conditions, including postoperative ileus and constipation [9].

Subsequently there are inevitable delays to reaching the correct diagnosis and initiating appropriate treatment, which increases the risk of complications, including colon necrosis and perforation [6,9]. Given the broad differentials for abdominal pain and distension after caesarean section, timely pathology and imaging are required. Typically, a CT scan of the abdomen and pelvis is the most easily accessible and high-yield imaging for determining the aetiology of the symptom. In the case of Ogilvie's syndrome, CT imaging will also enable accurate measurement of the degree of caecal and colonic dilatation to guide management [1,10].

Of note, a 2019 case study demonstrated that even in the presence of CT imaging revealing grossly dilated loops of bowel without evidence of mechanical obstruction, the diagnosis of Ogilvie's syndrome can be missed [3]. In that case study there was concurrent CT evidence of appendiceal thickening and fat stranding leading to the presumed diagnosis and conservative management of acute appendicitis. The patient's case was complicated by caecal necrosis requiring a right hemicolectomy [3]. This further highlights the need for increased awareness [4] of Ogilvie's syndrome and the importance of early diagnosis and management in decreasing morbidity.

Once the diagnosis of Ogilvie's syndrome has been made, the consensus is that management can be conservative, medical, endoscopic or surgical. The decision has to be guided by the patient's clinical presentation, the presence of electrolyte abnormalities and the caecal diameter on CT imaging [12]. In an uncomplicated presentation without evidence of caecal ischaemia or necrosis and a caecal diameter less than 9-10 cm, it is suitable to trial conservative management for up to 48 h [5,10,12]. Medical management with intravenous neostigmine has been shown to be highly effective in managing Ogilvie's syndrome and can be considered when there is failure of conservative therapy or as an adjunct to endoscopic therapy [12,13]. In the case study the patient had a borderline caecal diameter of 9.7 cm and an elevated CRP of 320 mg/L, indicating an increased risk of ischaemic necrosis approaching caecal perforation. Therefore, immediate endoscopic decompression and assessment were judged beneficial, with the endoscopy revealing no evidence of mucosal ischemia. If these management steps are not successful, or there is evidence of ischemia and perforation, in its guidelines the American Society for Gastrointestinal Endoscopy recommends referral for surgical management [12]. Whilst a laparoscopic approach may be beneficial as an initial diagnostic tool, in the case of ischemic necrosis or perforation an exploratory laparotomy is required to assess the extent of non-viable bowel and enable resection [10].

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Patient consent

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