Ogilvie Syndrome Complicated By Caecal Perforation In A Post-Caesarean Section Patient: A Case Report

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Citation

Abstract
Ogilvie syndrome or acute colonic pseudo-obstruction is a condition characterised by progressive dilatation of the proximal colon with no evidence of mechanical obstruction. It is associated with various medical and surgical conditions. In 50-60% of cases the preceding cause is trauma or surgical procedure, most commonly caesarean section. However there is little known about it in obstetric practice and it presents a diagnostic and therapeutic challenge. We report a case of Ogilvie syndrome following an emergency caesarean section, which resulted in caecal perforation on the fourth post-operative day.

INTRODUCTION
Ogilvie syndrome (Acute colonic pseudo obstruction) is a clinical syndrome characterised by acute massive dilatation of the colon with no organic obstruction of the distal colon. Sir William H Ogilvie first reported the syndrome in 1948, in two patients with metastatic cancer (1). Since then, despite accurate characterisation of the syndrome, Ogilvie’s syndrome (OS) remains difficult to diagnose and is still associated with significant morbidity and mortality.

CASE REPORT
A 30-year-old second gravid had an uneventful pregnancy. She was admitted to delivery suite at term with spontaneous rupture of membranes and in early labour. On vaginal examination the cervix was 3-4 cm dilated and liquor was stained with meconium. The admission cardiotocogram showed prolonged late decelerations in view of which fetal blood sampling was attempted but was unsuccessful. Consequently an emergency lower segment caesarean section (CS) was performed under a spinal anaesthetic. The CS was uncomplicated and straightforward.

The patient was initially well post operatively and was allowed to eat and drink 6 hours following the CS. However 72 hours after the caesarean section, she developed abdominal pain, distension, nausea and was unable to pass flatus. Her temperature was mildly elevated at 38°c and abdominal examination revealed a distended abdomen with some lower abdominal tenderness. The bowel sounds were absent on auscultation. An initial diagnosis of adynamic ileus was made and she was kept nil by mouth. She was also started on intravenous antibiotics in view of pyrexia and mild leucocytosis. The electrolyte levels were reported as normal. Her symptoms of pain and pyrexia improved after 12 hours, bowel sounds were noted to be normal and she was passing flatus. The abdominal distension however persisted. In view of improvement in clinical symptoms, she was allowed to eat and drink. Over the next 24 hours, the abdominal distension increased and she started to complain of increasing abdominal pain. On examination she was tachycardic with a markedly distended and tender abdomen. A supine abdominal x ray revealed some extra luminal gas. A CT scan was then carried out to confirm the diagnosis and it showed large amount of free air in the abdomen and the pelvis suggestive of viscus perforation.

The patient therefore underwent an emergency laparotomy. At laparotomy, diffuse faecal peritonitis was noticed due to a posterior caecal perforation. Abdominal exploration and palpation of the remainder of colon did not reveal any mechanical obstruction and perforation was thought to be due to pseudo obstruction. A limited right hemicolectomy with loop ileostomy was performed. Her post-operative recovery was complicated by subphrenic and pelvic collections, which were later, drained under an anaesthetic. She also developed mild pleural effusion, which resolved spontaneously. She was finally discharged home 35 days after her initial admission to the hospital. A gastrografin enema was done just prior to her discharge and it revealed no abnormalities in the colon. The histology of the resected...
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The colon showed necrosis around the edges of perforation, thereby confirming the diagnosis of colonic pseudo-obstruction.

DISCUSSION

Acute pseudo obstruction is characterized by progressive colonic dilatation in the absence of any mechanical obstruction. It is associated with various medical conditions such as metabolic derangements, stroke, myocardial infarction, acute respiratory failure and sepsis. In 50-60% of cases it develops after surgical procedures especially caesarean sections, orthopaedic and thoracic procedures. If untreated, it may lead to progressive dilatation resulting in necrosis and perforation typically of the caecum with a high mortality ranging from 35% to 72% (1).

The exact aetiology of OS is unknown and appears to be multifactorial. Currently the most commonly accepted theory is dysfunction of autonomic nervous system (2,3,4). It has been postulated that the normal parasympathetic outflow from sacral segments 2, 3 and 4 are disrupted, causing a functional obstruction where the proximal and distal nerve supplies to the colon overlap.

The initial management is generally conservative, with nasogastric suction, correction of fluid electrolyte imbalance, placement of rectal tube, gentle enemas and if possible decompression of the colon with a flatus tube (5,6). Frequent evaluation of colonic dilatation should be done with serial abdominal X-rays. Pharmacologic agents that increase GI motility like neostigmine and cisapride have been used with varying degrees of success. Endoscopic decompression of the colon may be used if conservative treatment fails, or if caecal diameter reaches 12 cm (6,7). Recurrence of symptoms (40%) and failed procedure occur in 15-20% following colonoscopy and occasionally serial decompression may be required (8). Caecostomy is another option for patients not responding to conservative management or recurring after colonoscopy. Surgery is indicated when colonic decompression has failed or there is evidence of ischemia or perforation. However, surgery is associated with a high mortality.

OS is an established post-operative complication, but since it occurs rarely it may be overlooked or treated as an adynamic ileus because of the similarity in symptoms. The diagnosis is particularly difficult in puerperium since mild pyrexia, leucocytosis and some lower abdominal tenderness is generally present following CS. The hallmark symptom is development of marked abdominal distension over a short period of time and therefore a diagnostic abdominal X-ray should not be delayed even if the bowel sounds are normal.

This case highlights the need for a greater awareness, amongst obstetric practitioners, of this potentially lethal syndrome, since it may be successfully treated if recognized early.

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References

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