

Postpartum caecal perforation due to endometriosis

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DECLARATIONS

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We present a rare case of postpartum caecal perforation secondary to endometriosis.

Introduction

The prevalence of endometriosis in women in the general population is in the region of 10% and it most commonly occurs in the pouch of Douglas, pelvic peritoneum and ovaries. However, extragenital sites such as the small and large intestine can be involved. Extra-uterine endometrial tissue behaves just as uterine endometrial tissue and, therefore, undergoes hormone-induced changes during the menstrual cycle including bleeding. The clinical consequence of this is severe pain, which is the commonest presentation of endometriosis.¹

During pregnancy the endometrial stroma undergoes decidualisation due to hormonal changes and the normally small cells of the endometrial stroma undergo enlargement. Ectopic endometrial tissue also undergoes decidualisation.^{2,3}

Case report

We present the case of a 33-year-old woman who, at 37 weeks' gestation, had an elective lower segment Caesarean section (LSCS) for placenta praevia. Immediately postoperatively the patient and baby were well, however, three days post Caesarean our patient became unwell with abdominal pain, distension, tachycardia and signs of peritonitis.

She had a past medical history of terminal ileal and caecal Crohn's disease diagnosed nine years previously that was well-controlled and for which she had been on 100 mg azathioprine daily before her pregnancy. This was discontinued during pregnancy and she remained asymptomatic from her Crohn's. She had previously had an appendicectomy but there was no history of endometriosis.

Given the patient's past medical history, intra-abdominal sepsis secondary to Crohn's disease or a collection post LSCS was suspected and an urgent computerized tomography (CT) scan of her abdomen and pelvis was organized. The CT scan showed free intraperitoneal air, free fluid/blood and areas of hyperdense material in the Pouch of Douglas with gas bubbles suspicious for infection (Figure 1).

The findings at emergency laparotomy were as follows:

- (1) 1 cm perforation of the caecum with free intestinal content within the peritoneal cavity;
- (2) thickened caecal pole with no appendix;
- (3) remainder of colon and small bowel normal;
- (4) Caesarean suture line intact.

The bowel was decompressed. The caecal perforation was excised and sent for histology and the perforation was over-sewn with an omental patch. Postoperative recovery was uneventful.

Histology from the excised caecum showed decidualised endometriosis, which infiltrated bowel wall with endometrial foci reaching all the way through to the mucosa with evidence of ulceration. The histological conclusion was that the caecal perforation was due to decidualised endometriosis. There was no evidence of Crohn's disease.

Discussion

Bowel perforation secondary to endometriosis is very rare. During a literature search seven



Table 1

Summary of cases of bowel perforation secondary to endometriosis related to pregnancy

Author	Year	Pregnant	Site of perforation	Other significant notes
Haufler ⁴	1931	Yes	Jejunal	6 months pregnant
Gini <i>et al.</i> ⁵	1981	Yes	Appendix	35 weeks pregnant
Nakatani <i>et al.</i> ⁶	1987	Yes	Appendix	26 weeks pregnant
Clement ⁷	1977	Yes	Sigmoid	37 weeks pregnant
Rud ⁸	1979	Yes	Sigmoid	
Flobert <i>et al.</i> 9	1984	Yes	Sigmoid	Postpartum (41 weeks)
Schweitzer et al. ¹⁰	2006	Yes	Sigmoid	Term pregnancy

reported cases of bowel perforation secondary to endometriosis were found involving pregnant or postpartum women (Table 1). Our case is the first reported case, to our knowledge, of caecal perforation in a postpartum woman and only the second case of postpartum perforation.

The histology of our case showed that like normal endometrial tissue in pregnancy the ectopic endometrial tissue in the caecum also underwent decidualisation in response to hormonal changes during pregnancy. Endometrial stromal cells can thus invade through bowel wall causing weakness of this area.

In the third trimester and postpartum, endometrial lesions tend to contract as observed by McArthur and Ulfelder,² the mechanism of which remains obscure. The timings of the bowel perforations in previous cases secondary to endometriosis in pregnant women in the literature – during the sixth month, during the 26th, 35th and 37th week, at term and postpartum support their observation. This contraction in an area already weakened by endometrial stromal infiltration can cause perforation as suggested by both McArthur in 1965² and Garg *et al.* in 2009.³ We suggest that this is what caused the bowel perforation in our patient.

We hope that this case will serve as another cause to be considered in the differential diagnosis of an acute abdomen in a postpartum woman.

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