

DOI: 10.21767/2471-9803.1000156

Spontaneous Sigmoid Colon Perforation Based on Endometriosis in Term Pregnancy – Case Report

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Received date: February 20, 2018; Accepted date: March 28, 2018; Published date: March 31, 2018

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Citation: Driak D, Bolehovska P, Spurkova Z (2018) Spontaneous Sigmoid Colon Perforation Based on Endometriosis in Term Pregnancy – Case Report. Crit Care Obst Gyne. Vol.4 No.1:3.

Abstract

The case report describes a woman with acute, spontaneous sigmoid colon perforation based on deep endometriosis in advanced stage of pregnancy. The 33-year-old white woman was admitted to our clinic at 37+6 weeks of pregnancy with symptoms of increasing abdominal pain successively changing the site of maximum. Due to suspicion of acute appendicitis, acute caesarean section, sigmoid resection and end-to-end anastomosis, appendectomy was performed. Spontaneous perforation associated with intestinal endometriosis in pregnancy is scarcely reported, some seven cases have been published in the English literature. To the best of our knowledge, this is the second case report of sigmoid colon perforation due to endometriosis during pregnancy.

Endometriosis affects approximately 10-15% of reproductive-aged women [5], however in subgroup of women suffering from sterility up to 50% and in subgroup of women suffering from pelvic pain up to 80%. Endometriosis does not develop before menarche and is very rare in women after menopause without hormonal (estrogene) replacement therapy [1,3,6]. Hormonal dependent lesions follow the regular cyclical fluctuating of 17beta-oestradiol and progesterone. Almost 1/3 of affected women had no symptoms, intensity and character of symptoms does not depend on the extent but localization, and substantial part of cases clears up spontaneously. About ½ half of patients cannot become pregnant, however, if pregnancy succeeds the state of patient may be improved. Despite activated macrophages in peritoneal fluid and altered endometrium, fertilization and nidation occur. During pregnancy due to physiological amenorrhea and hormonal changes, deposits of ectopic endometrium inactivate, regress, are resorbed and healed.

Key words:

Endometriosis; Sigmoid colon perforation; Term pregnancy

Introduction

Despite more than 100 years of research, endometriosis remains a rather strange and in some cases unpredictable disease. Its morphologic background is in endometrium-like tissue localized somewhere outside of endometrial cavity. With the exception of heart and spleen, endometriosis occurs at all organs and tissues of human body. Endometrioid lesions were histologically detected both at usual gynaecological localizations, e.g. ovaries, peritoneum, cervix, vagina, sacrouterine ligaments, scars after episiotomies and caesarean sections, and at extragenital sometimes curious sites, e.g. bladder, appendix, bowels, umbilicus, mamma, nasal mucosa, lungs, brain etc. Lots of theories have proved multifactorial etiology with genetic, anatomical, hormonal, immunological, angiogenetic and other, at the time unknown factors [1-4].

Most of lesions are typical, colored with hemosiderin and hemofuscin as degradation product of menstruating blood, however untypical lesions (stromal, whitish, yellowish, early reddish, subovarial adhesions, peritoneal defects – Allen-Masters syndrome) are not rare and make difficulties with visualization and detection. The therapy should be individualized and complex, i.e. surgical removal of lesions combined with long-term (6-48 months) hormonal blocking of menstruation. Due to atypical lesions and hormonal receptors variability, both surgical and hormonal therapy remains burdened with a high rate of recurrences [1,3,7-9]. Even though it is believed to be a progressive disease, progression was not seen in some women followed for more than 1 year without surgery [10].

Case Report

In February 2015, 8.15 a.m., a 33-year-old woman of caucasian race, I/0, was admitted in emergency duty to our clinic at 37+6 weeks of pregnancy with “contractions”. The woman did not suffer from any other complaints (diarrhoea, nausea, vomiting, fever, dysuria), tendency to constipation occurred over

a long period. The general physical examination was normal, cardio-pulmonally stabilized (blood pressure 144/71 torr, heart rate 94 bpm), mediosacral cervix 1.5 cm for one finger to the head, position longitudinal, membranes were not ruptured, no signs of bleeding. Group B Streptococci were negative. Ultrasound showed biometry of foetus matching to 38 weeks, normohydramnion, normal Doppler's flowmetry, placenta localized on the front wall without signs of abruption. Cardio-tocography was physiological.

The previous development of pregnancy was normal without hospitalization. The patient was on anti-anaemics (ferrous sulfate 1 tbl. p.o. a day), in outpatient's department of gynaecology examined for suspicion of endometriosis without any other dealing, in anamnesis no diseases, operation or other medications were reported. Actual laboratory tests with the exception of white blood cells $20.2 \times 10^9/L$, CRP 10.6 mg/L, AST 0.57 $\mu\text{kat/L}$, ALT 0.59 $\mu\text{kat/L}$, ALP 3.67 $\mu\text{kat/L}$, were in physiological range.

The patient expected the start of labour, however, the character of pains was not typical. The pain started early in the morning (5.00 a.m.) in left hypogastrium and during several hours spread to the whole abdomen, the patient localized the maximum of pains to uterus. There were no signs of peritoneal irritation. Despite of increasing intensity of pain, there were no development on cervix and finding remained almost the same. In the afternoon pain moved to the right mesogastrium, later both mesogastriums and uterus were painful, contractions could not be distinguished from signs of peritoneal irritation. Gynaecologist in duty recommended ultrasound on appendix and surgical consultation to exclude acute diverticulitis and appendicitis.

Radiologist's ultrasound indicated hydronephrosis grade III on the right side, some free fluid at bases of caecum, appendix not seen, colon descendent filled with content.

The same day together with surgeons with regard to increasing signs of peritoneal irritation and no development on cervix, we decided to perform acute caesarean section with abdominal revision focused on caecum and appendix under the antibiotics amoxicilinum/acidum clavulanicum 1.2 g and metronidazolium 0.5 g i.v.

The incision according to Pfannenstiel was performed, at the moment of peritoneal opening, the purulent secretion above and next to uterus was observed, the samples for aerobic and anaerobic microbial culture were taken. Baby-girl was measured: 3000 G/49 cm, Apgars's score 5-8-9, pH 7.284 and 7.307. After having sutured hysterotomy, together with surgeon peritoneal cavity and bowels were controlled. Lot of purulent secretion and

slightly irritated appendix in subcaecal position were observed, however, maximum of changes found at left meso-hypogastrium. There were detected fibrin coatings and signs of inflammation on the left side of uterus, localized peritonitis bordered by left edge of uterus and left adnexa, perforation on anti-mesenterial side of sigmoid intestine and surrounding wall of colon inflamed, infiltrated and fragile. In distance of 10 cm the sigmoid colon was covered with some not-inflamed diverticles. The diagnosis was established as the perforation of inflamed sigmoid diverticle.

About 20cm part of sigmoid colon was resected with end-to-end anastomosis. In addition, the appendectomy was performed as well. Peroperatively, antibiotics were switched to piperacilinum/tazobactam 4.5 g iv. After flushing of peritoneal cavity with iodine solution, stopping the diffusive bleeding from fragile, inflamed tissue, and drainage the abdominal wall was sutured lege artis, blood loss 500 mL, total time of operation included total anaesthesia 2 h 30 min.

After the operation, the patient was intensively observed at gynaecological emergency unit. Five days of parenteral nutrition (Oliclinomel 2000 mL i.v./24 hours and crystalloids), miniheparinization (enoxaparinum 0.4 mL sc./24 h), common analgetics, prokinetics (metoclopramidi hydrochloridum 10 mg iv. each 8 h), antiulcerosa (omeprazolium 40 mg iv. a 12 h) were administered, antibiotics were continued: 3 days of piperacilinum/tazobactam 4.5 g iv. each 8 hours, then switched to 7 days of ertapenemum 1 g iv. each 24 hours plus ofloxacinum 400 mg iv. each 12 hours. Since the 4th day after caesarean section the patient was treated with metoprolol tartrate 50-0-50 mg p.o. on for hypertension. Nasogastric sonde and rectal tube were on site up to 6th day after the operation when the peristaltics renewed completely.

In laboratory, blood count did not significantly changed - Hb 116.0 g/L, PLT $238.0 \times 10^9/L$, procalcitonin 0,42 $\mu\text{g/L}$, fibrinogen 7.71 g/L, liver enzymes were transiently elevated, WBC transiently increased to $26 \times 10^9/L$, CRP to 294.1 mg/L. The pathological level of Escherichia coli was detected in the microbial culture in purulent sample from abdominal cavity. The same pathogen was found in hemoculture and from the placenta's scratch.

Histopathological results confirmed perforation of sigma that was partially blocked with polypoid deposit of decidualized endometriosis extending into the lumen of intestine, other decidualized endometriosis was found in appendix (**Figure 1**). The patient healed per primam intentionem was discharged at good condition on the 15th day after surgery.

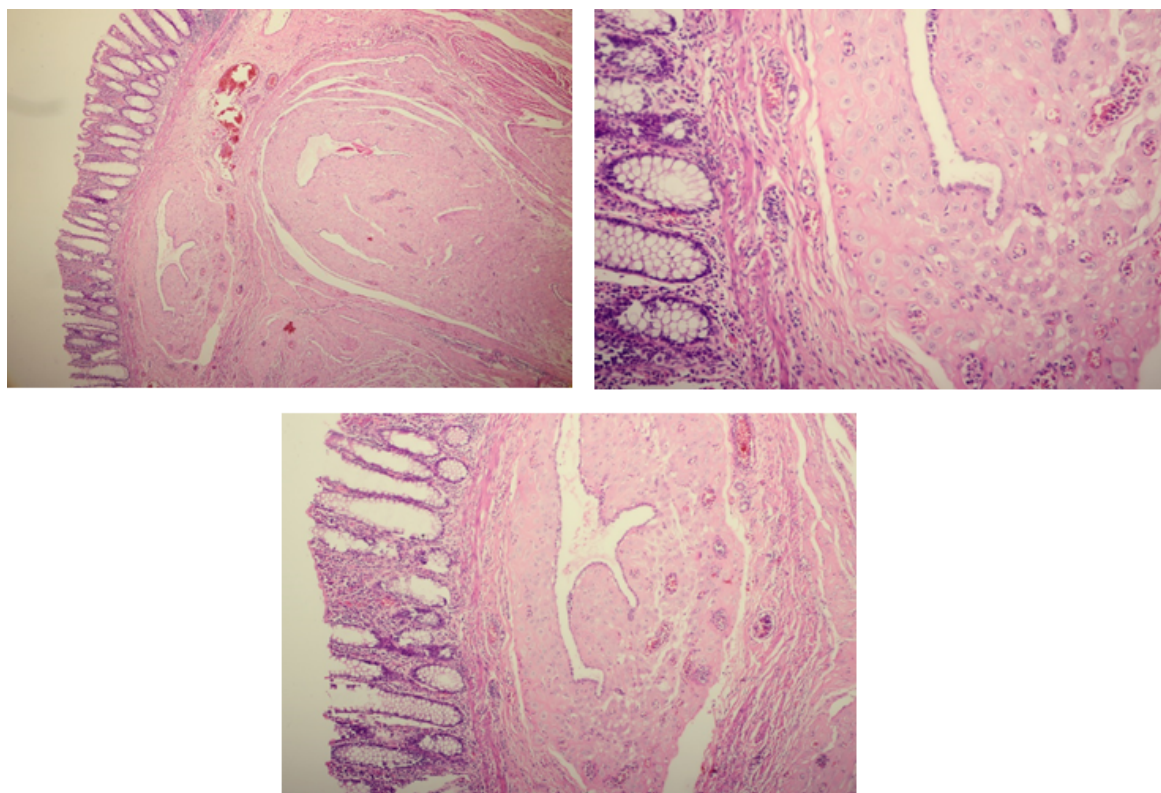


Figure 1 Histological results of sigma.

Discussion

As a rather enigmatic disease, pathophysiology, progression, and natural history of endometriosis remains poorly understood. Intestinal involvement is the most common of extragenital forms, and occurs in 3-7% [11], resp. 5-15% [3] of all patients with endometriosis. Distribution in bowels ranges (**Table 1**).

Table 1 Distribution of intestinal endometriosis [3].

Localization	Incidence [%]
rectum	72-85
sigmoideum	10-20
caecum and appendix	10
terminal ileum	5

Specific symptoms are absent in 30-95% of patients, intestinal endometriosis typically takes the form of asymptomatic serosal implants. The most common symptom, recurrent but acyclic abdominal pain reaches up to 70% of cases, the complete intestinal obstruction occurs in less than 1% [3,12]. Other symptoms like constipation, dyschesia, enterorrhage, hematochezia, painfull peristaltics, diarrhoea, painfull sitting, subileus, ascites, occur in 10-50% [3].

Spontaneous intestinal perforation due to endometriosis is a very rare complication, totally some twenty cases [13] and some seven cases in pregnancy have been reported in the

English literature [14]. Endometrioma surrounded with tubo-ovarian abscess secondarily eroding the sigmoid colonic wall in a 38-year-old, non-pregnant women was revealed by CT scanning, confirmed by surgery and published in 1988 [15].

In 1977, Clement as the first described perforation of the sigmoid colon in a 28-year-old woman during the 37th week of pregnancy [16]. Perforation of appendix in a 23-year-old Nigerian Igbo woman during the 35th week of pregnancy was reported by Gini et al. [17] and in a 25-year-old woman at the 26th week of gestation by Nakatani et al. [18]. In both cases, histological results showed appendiceal wall replaced extensively by decidualized endometriotic tissue.

In 2010, firstly Pisanu et al. published rectal perforation due to endometriosis in a 37-year-old woman hospitalized at the 30th week of gestation. The symptoms were suggestive of pyelonephritis, after 3 weeks the premature delivery through a caesarean section was performed. On the 2nd postoperative day abdominal CT showed pelvic abscess and free air in the peritoneal cavity, afterwards Hartmann operation was performed [13].

In 2013, firstly Nishikawa et al. published endometriosis-induced ileal perforation with massive hemorrhage that required caesarean section, right hemicolectomy and terminal ileum resection at the 33rd week of gestation [14].

The diagnosis of an intestinal perforation during pregnancy especially in the third trimester might be extraordinarily difficult. Symptoms are similar to appendicitis, diverticulitis, enterocolitis

(morbus Crohn), ischemic colitis, colon irritable syndrome, colorectal cancer, even though to pyelonephritis which can delay diagnosis and have fatal consequences. Hotly discussed may be our access to perform resection of sigmoid colon with end-to-end anastomosis. Lot of surgeons prefer temporary outlet (colonostomia) with returning after interval 4-6 months. Reasons for immediate anastomosis were minimal contamination of peritoneal cavity, peritonitis localized to relatively small region, anatomical condition, stable patient and total time of operation.

We suppose during pregnancy, the endometriosis became inactivated, chronic, and probably exacerbated in the final stage of pregnancy due to constipation and partial obstruction of large intestine. The endometriosis-induced intestinal perforation in pregnancy remains rare complication, however can cause life-threatening situation for both the mother and baby especially in case of delayed intervention and considerably preterm delivery. Above that, with regard to an increasing age of the first delivery, a growing number of women with severe endometriosis conceive through assisted reproductive technology. Multidisciplinary cooperation is necessary during patient care.

Conclusion

We reported the case of spontaneous sigmoid colon perforation based on endometriosis in in the 33-year-old woman at 38th week of pregnancy. According to PubMed, our work is the second case report of sigmoid colon perforation due to endometriosis during pregnancy.

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