

Placental abruption in a setting of perforated acute appendicitis: a case study

INTRODUCTION

Placenta abruptio is defined as the premature separation of placenta from the placental bed. In the United States, the estimated incidence of placental abruption is from 0.6% to 1%, around 0.4 to 0.5% in Nordic countries and around 3.5 to 3.8% in Asian countries. In a systematic review conducted by Tikkanen M., placental abruption may cause fetal mortality as high as 60% and up to 12 % in developed countries¹. Risk factors include chronic hypertension, pre-eclampsia, thrombophilia disorders, premature rupture of membrane (PROM) and intrauterine infection. In a cohort study conducted by Abbasi et al, among 1203 pregnant patients who are complicated with acute appendicitis, there was a three-fold increase of preterm birth, four-fold increase of antepartum haemorrhage and two-fold increase of placenta abruption². Despite this risk, there are limited cases that reported on the association between placenta abruption and acute appendicitis^{3,4}. We reported a case of placenta abruption complicated by a case of perforated acute appendicitis.

CASE REPORT

A 27-year-old gravida 3 para 2 Malay lady presented to our hospital at 30 weeks 1 day of gestation with a complaint of contraction pain. Otherwise, fetal movement was good and patient denied any per vaginal bleeding, fever, prior trauma or massage. She visited the Emergency Department one day prior with complaint of epigastric discomfort but was subsequently discharged as gastritis. Her past medical history included history of caesarean section in 2016 for malpresentation which was uneventful, history of vacuum-assisted delivery in 2012, gestational diabetes mellitus and mild anaemia in this current pregnancy.

On examination, she was alert and not septic looking. Her temperature was 36.7, pulse rate was 123 beats per minute, blood pressure was 117/69 mmHg and oxygen saturation was 97% under room air. Cardiac and lungs assessment were unremarkable. Abdominal examination revealed tense, tender and woody hard abdomen. Uterus was palpable at 34 weeks and fetal parts were not felt. Haemoglobin level was 10.8 g/dL, total white count was 17.33 x 10³ m/uL. Renal profile was normal.

Transabdominal ultrasound showed fetal parameters ranging from 28 to 31 weeks with EFW of 1300g. Fetal heart was present. Retroplacental clot also was seen over the fundus around 5 x 4 cm. CTG was normal. An emergency caesarean section was subsequently performed for placental abruption and around 50 ml of retroplacental blood clot was found but no couvelaire uterus was seen. There was also multiple endometriotic spots found over postero-inferior part of the uterus and the pouch of Douglas was mildly obliterated.

Post-operatively, patient kept on complaining of minimal abdominal pain and distension. Abdominal girth measurement was noticeably increased from 93 cm to 103 cm. She was otherwise alert and not septic looking. She was persistently tachycardic, with heart rate ranging from 120 to 130 beats per minute. Other parameters were normal and she remained afebrile. ECG showed sinus tachycardia. CTPA was done to rule out pulmonary embolism and was negative. Abdominal X-Ray was also done and was normal. Patient was then referred to surgical team and a CECT of the abdomen was done. It showed small bowels dilatation with maximum diameter of 4.2 cm at the jejunum loops extending to the distal ileum. Terminal ileum, caecum and the rest of the bowel were collapsed. Free fluid was seen over subhepatic space and right paracolic gutter. An exploratory laparotomy was subsequently performed for intestinal obstruction secondary to adhesion.

Intraoperatively, a perforated appendicitis was found with partial transection over the body. There was also presence of faecolith seen with inflamed mesoappendix and interloop collection. Small bowel was healthy looking but dilated. Seropurulent collection at right side of the pelvis was also seen measuring around 600ml. Around 50ml of pus was drained and sent for culture and sensitivity.

DISCUSSION

Multiple studies have suggested the effect of inflammation on placental abruption. One of the hypotheses explored is the production of matrix metalloproteinases-9 (MMP-9), an enzyme involved in activating the proteolytic cascade in placental abruption by causing the degradation of extracellular matrix and basement membrane³. A proposed model by Ananth et al, suggested that inflammation causes recruitment of placental and decidual macrophages as well as pro-inflammatory cytokines such as tumor necrosis factor- α and interleukin- β at the fetal-maternal interface at the placenta¹. This causes further increase in the MMP, leading to necrosis and apoptosis which resulted in placental bed separation¹.

Lockwood et al in their study noted that there is an increased infiltration of neutrophils into the decidua in the group with placental abruption compared to controls proving that inflammation plays an important role in abruptio-related cases. Neutrophils are a source of elastase and MMP-9. The infiltration of neutrophils in placental abruption is enhanced by the increased expression of IL-8 in the decidua².

H. G Yilmaz et al, in their study conducted in a general hospital in Turkey reported a percentage of 8% of fetal mortality in a sample size of 52 pregnant women who developed acute appendicitis.

There is a significant difference in terms of pregnancy complication between perforated and non-perforated appendicitis (52% vs 17% respectively, with a p value of <0.013). In the study, it was concluded that late gestational age and delayed presentation have higher tendency for preterm labour and fetal mortality⁴.

Atypical physical presentation, and limited sensitivity and specificity of laboratory studies as well as ultrasound findings are some of the contributors to a delayed management from the onset of symptoms to the time of operation in this study population⁴. Physiological leucocytosis and presence of haematuria or leukocyturia in the pregnant population in this study also made the diagnosis of acute appendicitis difficult⁴. Atypical physical presentation of acute appendicitis among pregnant population might be likely due to upward displacement of the appendix as well as increased distance between the appendix and the abdominal wall during advanced pregnancy⁵.

CONCLUSION: Inflammation plays an important role in the pathophysiology of placental abruption and preterm labour. Diagnosis of acute appendicitis among pregnant population is often missed due to atypical presentation in this special population. There should be high suspicion index among clinician when examining pregnant women who presents with abdominal pain so that proper management could be done for the patient in the appropriate time window.

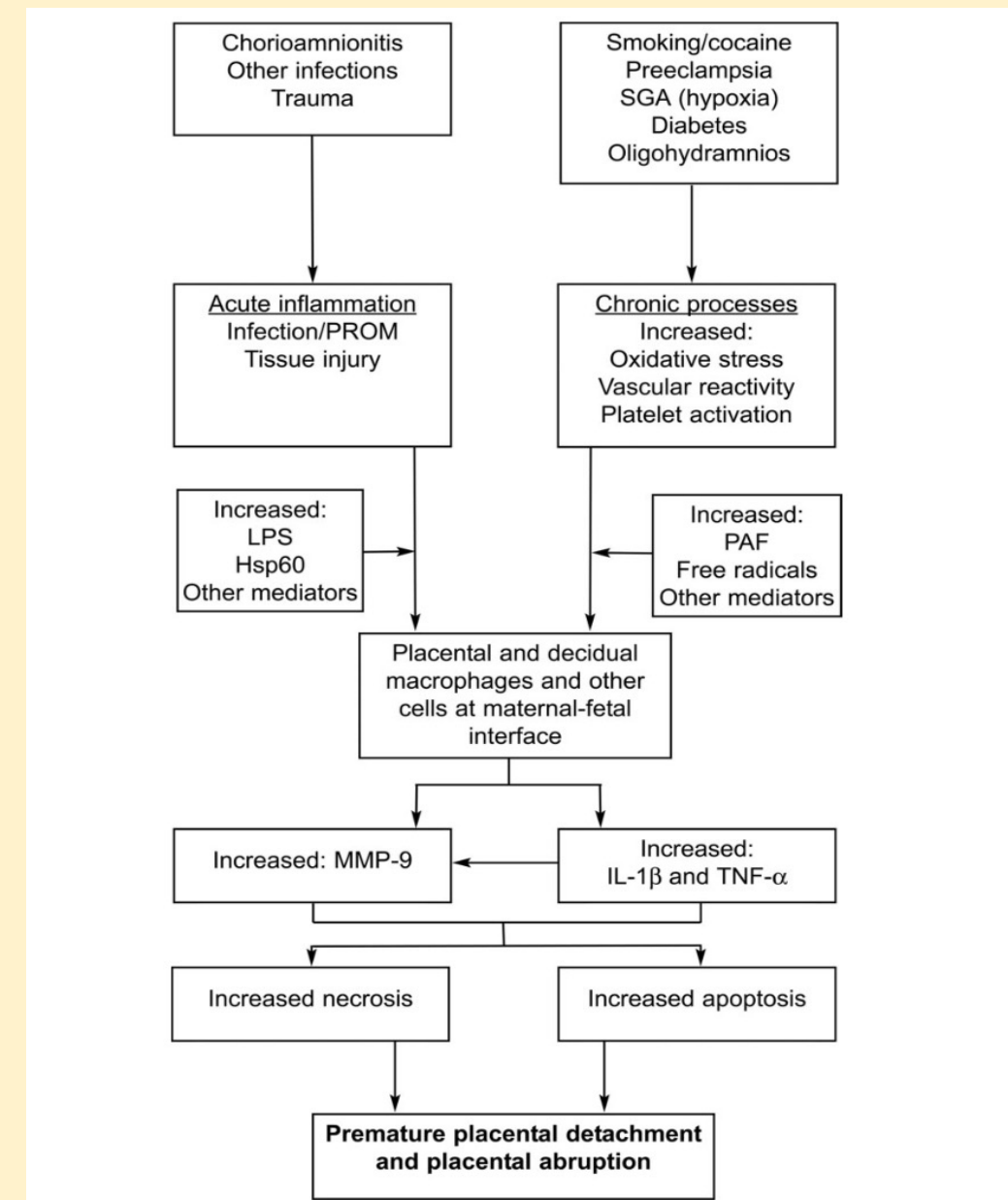


Figure 1. A proposed model by Ananth et al, showing the causal pathway between acute inflammation and placental abruption¹.

REFERENCE:

1. Ananth C, Getahun D, Peltier M, Smulian J. Placental Abruption in Term and Preterm Gestations. *Obstetrics & Gynecology*. 2006;107(4):785-792.
2. Lockwood C, Toti P, Arcuri F, Paidas M, Buchwalder L, Krikun G et al. Mechanisms of Abruption-Induced Premature Rupture of the Fetal Membranes. *The American Journal of Pathology*. 2005;167(5):1443-1449.
3. Xu P, Alfaidy N, Challis J. Expression of Matrix Metalloproteinase (MMP)-2 and MMP-9 in Human Placenta and Fetal Membranes in Relation to Preterm and Term Labor. *The Journal of Clinical Endocrinology & Metabolism*. 2002;87(3):1353-1361.
4. Yilmaz H, Akgun Y, Bac B, Celik Y. Acute appendicitis in pregnancy — risk factors associated with principal outcomes: A case control study. *International Journal of Surgery*. 2007;5(3):192-197.
5. Yale S, Tekiner H, Yale E. Physical Examination and Appendiceal Signs During Pregnancy. *Cureus*. 2022;.