Appendicitis in pregnancy mimicking haemolysis, elevated liver enzymes and low platelets syndrome

Pooja Sikka MD DNB, Neelam Aggarwal MD, Seema Chopra MD and Vanita Suri MD

Department of Obstetrics and Gynaecology, Postgraduate Institute of Medical Education and Research, Chandigarh-160012, India

Summary: We report a patient with pregnancy at term in whom appendicitis mimicked haemolysis, elevated liver enzymes and low platelets (HELLP) syndrome. A high index of suspicion of appendicitis should be kept in patients with abdominal pain and biochemical evidence of HELLP.

Keywords: appendicitis, pregnancy, HELLP

INTRODUCTION

Pregnancy can obscure the accurate diagnosis of acute appendicitis and its complications due to various gestational, anatomical and physiological changes. The incidence of complications increases with increasing gestational age and includes risk of abortions, preterm labour, fetal and maternal mortality. Early diagnosis is therefore critical but can be missed due to atypical presentations. We present a patient in whom appendicitis mimicked haemolysis, elevated liver enzymes and low platelets (HELLP) syndrome.

CASE REPORT

A 30-year-old G₂P₁₀₀₁ at 39 weeks gestation presented with jaundice and persistent right hypochondrial pain. Her antenatal course had been uncomplicated except for two blood pressure (BP) measurements of 130/90 mmHg at her local hospital. She reported nausea, yellowish discolouration of eyes and urine for the past one week. There was no history of vomiting, headache, visual disturbances, vaginal discharge or bleeding, fever or constipation. Her previous medical history was unremarkable except for a lower segment caesarean section (LSCS) for non-progress of labour four years back.

On examination she was obese, had icterus, her pulse rate was 90/minute, BP 130/90 mmHg and temperature was 37.2°C. Abdominal examination revealed term-sized uterus with single living fetus and Pfannensteil scar of previous LSCS. There was right hypochondrial tenderness on deep palpation with no guarding, rigidity or rebound tenderness. There were no signs of imminent eclampsia.

Her laboratory results were haemoglobin – 10.2 g%; total leukocyte count – 10,800/cubic mm; differential leukocyte count showed 80% neutrophils, platelet count – 80,000/cubic mm; serum electrolytes were normal; blood urea – 32 mg/dL; serum creatinine – 1.2 mg/dL; serum bilirubin – 4.2 mg/dL

with conjugated bilirubin being 2 mg/dL; serum aspartate transaminase – 88 units/L; serum alanine transaminase – 106 units/L; and serum alkaline phosphatase – 322 units/L. Prothrombin time index was 100%. Urine routine and microscopic examination was normal except for albumin of 1+. Urine culture was sterile. Viral markers for hepatitis A, hepatitis B, hepatitis C and hepatitis E were negative. Ultrasound showed normal liver, spleen, kidneys and a single live intra-uterine fetus corresponding to 37 weeks gestation with a normal biophysical profile of 10/10.

Because of high BP records, proteinuria, right hypochondrial pain, thrombocytopenia and hyperbilirubinaemia with deranged liver enzymes, a diagnosis of preeclampsia with HELLP syndrome was considered and decision for termination of pregnancy was made. On her per vaginal examination cervix was uneffaced, firm and os was closed. The presenting part was high up.

A few hours later she went into spontaneous labour and she had fetal bradycardia for which she was taken up for emergency LSCS under general anaesthesia. The abdomen was opened by Pfannensteil incision. On entering the peritoneal cavity flakes of thick pus were seen. The uterus was opened in the lower segment, baby delivered and the uterus was closed in two layers. The baby was healthy with APGAR score of 8,9. Both ovaries were normal. Surgical consultation was sought intraoperatively. The abdomen was explored by a vertical midline incision. On exploration an abscess was found in the right upper quadrant just below the diaphragm. The abscess cavity was opened and a ruptured appendix was seen. Appendectomy and peritoneal lavage was done. A drain was placed in the right upper quadrant and the abdomen closed. She was administered broad spectrum antibiotics intravenously and made an unremarkable recovery. Histopathological examination confirmed acute inflammation in the appendix.

DISCUSSION

Although rare, appendicitis is one of the most common causes of acute abdomen in pregnancy. The incidence of appendicitis

in pregnancy is 0.05–0.07%.¹ This incidence is similar to that in the general population but pregnant patients are more prone to complications, especially perforations (43–55%).^{2–4} The risk of perforation increases with gestational age and perforation in the third trimester often leads to preterm labour, intra-uterine fetal death and peritonitis.² The overall fetal mortality is 2–8% but increases to 35% with perforation and peritonitis.^{5,6} It is thus critical to diagnose appendicitis and its complications early to improve maternal and fetal outcomes. Due to atypical presentations, diagnosing acute appendicitis poses significant challenges to the treating doctor.

Pregnancy is associated with certain changes that can delay the diagnosis of appendicitis especially the cephalad displacement and lateral rotation of caecum and appendix. That is why during the third trimester, the patient complains of pain higher and more laterally as was described by our patient. Guarding and rigidity are difficult to elicit due to stretched abdominal muscles. According to a review of 22 patients, the typical presentation (painful abdomen, nausea, vomiting, fever, leukocytosis) of appendicitis is not reliable.² The clinical and biochemical profile of our patient was suggestive of pre-eclampsia with HELLP syndrome. We were lucky that the patient went into spontaneous labour and had fetal bradycardia for which LSCS was done. Had she delivered vaginally, the diagnosis of appendicitis would have been delayed and she could have developed diffuse peritonitis leading to grave consequences. Retrospectively her jaundice, raised liver enzymes and thrombocytopenia could all be due to sepsis. The patient could have been having coexisting gestational hypertension.

Acute fatty liver of pregnancy can also present with similar features. However this rare differential was unlikely due to mildly raised liver enzymes and a normal prothrombin time index.

To conclude, this patient probably had sepsis due to the ruptured appendix, causing haemolysis and biochemical abnormalities leading to a presumptive diagnosis of HELLP syndrome. A detailed history, high levels of suspicion and clinical skills continue to be the best way to diagnose appendicitis in pregnancy. Acute appendicitis can mimic HELLP syndrome and should form an important differential diagnosis in patients with HELLP syndrome with abdominal pain.

Conflicts of interest: None

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