Case Report

Peptic ulcer disease in pregnancy: A rare cause of rapidly progressing anemia in mid-trimester of pregnancy – A case report and literature review

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ABSTRACT

Peptic ulcer disease is a rare cause of rapidly progressing anaemia in pregnancy, especially during the second trimester of pregnancy. Making a definitive diagnosis in this group of patients is usually very tasking, especially when the common causes of anemia in pregnancy such as Malaria, Sickle Cell Disease, Upper respiratory tract infection, nutritional anemia, e.g., iron and folate deficiency anemia and ruptured ectopic gestation are excluded. We present a rare cause of rapidly progressing mid-trimester severe anemia in pregnancy secondary to peptic ulcer disease in pregnancy, along with the diagnostic challenges, multidisciplinary management, literature review, and the follow-up care.

Key words: Eosophagogastroduodenoscopy; Havana Specialist Hospital Limited; peptic ulcer disease in pregnancy; rapidly progressing severe anemia.

Introduction

Peptic ulcer disease is quite uncommon in pregnancy and a rare cause of rapidly progressing anemia in pregnancy. Making a definitive diagnosis and instituting timely and appropriate management may be quite challenging because of the remarkable anatomical and physiological changes that the gastrointestinal tract (GIT) has undergone during pregnancy and the safety of the investigating tools such as radiological investigations and restriction in drug use during pregnancy.

However, promptness in arriving at a definitive diagnosis and commencing appropriate management will prevent maternal and perinatal morbidities and mortalities in these patients.

This paper reports a rare cause of rapidly progressing severe anemia in mid-trimester of pregnancy, secondary to peptic ulcer disease in pregnancy, in a primigravid woman in a private tertiary health care facility in Lagos, Nigeria.

Case Report

An unbooked 29-year-old G_1Po^{+0} woman at 18 weeks gestation was admitted through our outpatient department with 2-day history of generalized body weakness, vomiting, and poor appetite. There was no history of bleeding per vaginam. She was not a known diabetic and there was no history of peptic ulcer disease. She was a known asthmatic and on inhaler use whenever she had an attack. Her last asthmatic attack was a year before presentation at our facility.

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There was no history of use of nonsteroidal anti-inflammatory drugs since her last menstrual period.

At admission, her vital signs were within normal limits. The provisional diagnosis was malaria in pregnancy. Her packed cell volume was 30%, hemoglobin concentration was 10.3 g/dl, complete blood count, total and differentials, and platelet counts were normal. Blood film showed two pluses of malaria parasites. The mid stream urine for microscopy, culture, and sensitivity showed slightly turbid urine and heavy growth of *Proteus* species sensitive to cefuroxime. Pelvic ultrasound scan showed viable intrauterine pregnancy at 18 weeks gestation with adequate liquor.

She was treated with arthemisinine–lumefantrine combination therapy, hematinics, intramuscular maxolone 10 mg thrice daily, Tab. Zinnat 500 mg 12 hourly, and intravenous infusion of 5% dextrose saline to alternate with 5% dextrose in water 500 ml 4 hourly.

After about 30 hours on admission, attention was drawn to the patient. She was restless, sweating, and had developed hematemesis. Her pulse rate was 122 beats per minute, blood pressure was 115/62 mmHg, random blood sugar was 105 mg/dl, packed cell volume was 15%. Hemoglobin concentration was 4.5 g/dl.

Abdominopelvic ultrasound scan was normal. A provisional assessment of severe anemia in the second trimester of pregnancy secondary to Gastrointestinal bleeding was made. We invited the Gastroenterologists and the Surgeons to review. An assessment of upper gastrointestinal bleeding in pregnancy possibly secondary to Mallory–Weiss syndrome was made. She was investigated and managed accordingly. Endoscopy of the upper gastrointestinal tract showed normal esophageal and cardial parts, along with several erosions and areas of severe inflammation in the fundus and the upper body of the stomach. The duodenal parts were normal. The repeated packed cell volume was 12% and hemoglobin concentration was 3.7 g/dl. She was given intravenous ranitidine 300 mg stat and 50 mg 6 hourly, intravenous omeprazole 80 mg 8 hourly, metochlopromide 10 mg 8 hourly and oxygen by face mask. She was transfused with 6 pints of blood within 48 hours of making the definitive diagnosis. When she was clinically stable, Cap Amoxycillin 1 g 12 hourly was added to the prescribed medications by the gastroenterologists. Her post-transfusion packed cell volume was 26% and hemoglobin concentration was 8.7 g/dl.

She was discharged home to see in the antenatal clinic and to continue with the routine antenatal hematinics and oral medications for peptic ulcer disease.

She complied with the therapy and had an uneventful antenatal clinic visits. Her packed cell volume ranged between 28 and 34%. She had an emergency cesarean section at 41 weeks for failed induction of labor for prolonged pregnancy. The puerperal period was uneventful. She was educated on the need to comply with peptic ulcer medications. She was discharged to the medical outpatient department for further management 6 weeks postpartum period.

Discussion

Peptic ulcer disease is quite uncommon in pregnancy and a rare cause of rapidly progressing anemia in pregnancy, especially in the first and second trimester. Many researchers reported a decrease in the incidence of peptic ulcer disease in pregnancy.[1-6] Various reasons or theories have been proposed for this reduction in the incidence, which includes the increase in the gestational hormones, especially progesterone, that cause an increase in gastric mucus synthesis, reduced gastrointestinal motility, and lower gastric acid production in pregnancy.

Similarly, during pregnancy there is an increase in plasma histamine, caused by placental histaminases synthesis, which increases the metabolism of maternal histamine, thereby reducing gastric acid secretion.[6,7] Amdeslasie reported an incidence of 1–6 in every 23,000 pregnancies.[7] Guberman *et al*. quoted an incidence of 1 in 4000 deliveries.[4] Other factors for reduced incidences in pregnancy include avoidance of alcohol, cigarette smoking, restriction in drug use during pregnancy, especially NSAIDS.[7,8] Interestingly, some authors has reported that the reduction is due to underevaluation of these patients because of the restriction in the use of investigation tools during pregnancy such as radiological investigations and endoscopy.[2,8,9,10,11] Frequent treatment of reflux esophagitis during pregnancy with antacids is also a major factor for underdiagnosis.[6,9,10,11] All these must have contributed to our inability to make a timely definitive diagnosis.

The similarities in the clinical presentations of patients with peptic ulcer disease in pregnancy and those of pregnancy symptoms or reflux esophagitis also pose a big challenge in making a definitive diagnosis.[7,10,11] However, nausea and vomiting are rarely seen after 20 weeks of pregnancy but peptic ulcer disease often becomes worse or more severe during the third trimester. Our patient presented with nausea and vomiting at 18th week of gestation and there was no previous history of peptic ulcer disease or the use of NSAIDs. The only pointer to peptic ulcer disease in this pregnancy which was hematemesis was seen about 30 hours on admission.[12,13]

Multidisciplinary approach to the management of these patients will improve outcome and reduce morbidities and mortalities. Thus, our patient was comanaged with a gastroenterologist and a surgeon who carried out the eesophagogastroduodenoscopy (EDG).

The goals of managing these patients are to relieve the symptoms, heal the ulcers, prevent complications, and recurrences and to minimize the occurrence of teratogenicity in the foetus. All these we did as a team to achieve optimal output.

In general, the first line of treatment involves lifestyle modifications such as avoidance of provoking foods, alcohol, smoking, drugs, for example, NSAIDS. Medical therapy includes sucralfate, an aluminum salt of sucrose, to provide a protective coating at the ulcer base, H_2-receptor antagonists such as cimetidine and ranitidine, proton-pump inhibitor such as omeprazole and antimicrobial therapy, when *Helicobacter pylori* infection is confirmed.[2,4,5,9,14] All these groups of drugs have been reported to be save during pregnancy.[15,16] However, the prostaglandin analogue such as Misoprostol should be avoided, because of the risk of uterine contraction and abortion. Similarly, tetracyclines are teratogenic and must be avoided in pregnancy.

Combinations of these drugs were used for our patient because of the severity of her condition. We however avoided the teratogenic ones.

Conclusion

Peptic ulcer disease, though said to be a rare occurrence during pregnancy, are seen more often than documented in obstetric practice because they are under-diagnosed. When it occurs in pregnancy, the challenges in making a definitive diagnosis may cause a delay in instituting a timely and appropriate treatment, resulting in an increase in maternal and perinatal morbidities and mortalities. Our case should actually be termed a "Near miss." Therefore, health care givers should always consider peptic ulcer disease as a differential diagnosis in pregnant women with gastrointestinal disorders.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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