Spontaneous Rupture of the Liver in Hypertensive Disease of Pregnancy

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Summary

This is a case of spontaneous rupture of the liver in a 34-year old G^5P^{4+0} (3 alive) with hypertensive disease of pregnancy at gestational age of 34weeks. This case is reported because the condition is a recognized but rare complication of hypertensive disease of pregnancy. The typical features of this condition as described in the literature and as seen in this patient are emphasized as increased awareness can lead to early diagnosis and better prognosis.

Key Words: *Hypertensive disease of pregnancy, Abruptio placenta, Hemorrhagic shock, Hemoperitoneum, Spontaneous rupture of the liver in pregnancy.*

Introduction

Spontaneous hepatic rupture in pregnancy is rare and associated with significant maternal morbidity and mortality. It has been reported in association with severe pregnancy-induced hypertension and HELLP (hemolysis, elevated liver enzymes, and low platelets) syndrome[1]. Surgical intervention is necessary in capsular rupture with hemodynamic instability[1]. This disease should be considered when there occurs pain in the upper part of the abdomen and signs of hemorrhagic shock, even in the case of uncomplicated pregnancy[2]. Also high maternal and fetal mortality rate has been reported[3,4]. The treatment include treatment of hemorrhagic shock and clotting disorders, control of hepatic bleeding, and delivery[4,5]. A multidisciplinary approach of the critical care team often will effect a reduction in maternal morbidity and mortality[6]. This case serves to remind clinicians of the high maternal and perinatal mortality rate associated with complications of hypertension in pregnancy.

Case Report

Mrs. O. M was a 34-year old booked, G^5P^{4+0} (3 alive), trader was admitted via the antenatal clinic as a result of a blood pressure of 180/120mmHg at a gestational age of 34weeks. There was no history of headache, blurring of vision, epigastric pain/vomiting or abdominal pain. Patient was not a known

hypertensive or diabetic. She had spontaneous vaginal deliveries in 1995, 2000, and 2003 in hospital after uneventful pregnancies. She had a stillbirth in 1997 at 32weeks gestation but the cause was unknown to her.
She was not clinically anemic. Her pulse rate was 88 beats per minute, blood pressure was 170/120mmHg, and temperature was 36.2^{o}C. Her abdomen was soft with no definite area of tenderness, symphysio-fundal height was 35cm, singleton, longitudinal lie, cephalic presentation, and fetal heart rate was 146/min regular.
An impression of hypertensive disease in pregnancy was made. She had intravenous bolus of 10mg diazepam and intravenous hydrallazine 5mg slowly. The results of investigation done on admission were Na^{+} - 132mmol/l; K^{+} - 3.0mmol/l; HCO^{-}_{3} – 23mmol/l; Urea – 44mg/dl; Cl^{-} - 92mEq/l; Creatinine – 1.0mg/dl. Her packed cell volume (PCV) was 31%. Her hemoglobin was 10.5g/dl; total white cell count was 8.8 x 10^{9}/L; granulocyte was 79%; lymphocyte was 14%; monocyte was 7%; platelet count was 299 x 10^{9}/L. Total bilirubin 2.34mg/dl; direct bilirubin 1.53mg/dl; SGOT – 30U/L; SGPT – 22U/L; Alkaline phosphatase – 154U/L. There was no glycosuria or proteinuria on urinalysis and no pus or red blood cells on urine microscopy.
The blood pressure was stabilized in the range of 140/90 to 150/100mmHg on oral α-methyldopa and nifedipine. A daily fetal kicks chart was kept. An ultrasound scan (USS) done on admission was showed a singleton viable fetus in longitudinal lie, cephalic presentation. The measurements showed a biparietal diameter (BPD) of 8.4cm; head circumference of 31.2cm; abdominal circumference of 30cm and a femur length of 6.6cm. The USS gestational age was 34weeks ± 1week with the expected delivery date (EDD) of 26/6/06, and estimated fetal weight of 2.2kg. The amniotic fluid was of normal volume and the placenta was normal and posteriorly located. Blood coagulation profile was not done because of technical hitches in the laboratory then. However, the bed side clotting time was 8 minutes.
On the fourteenth day on admission, she suddenly complained of difficulty in breathing after an initial headache. She was found dyspneic, moderately pale, afebrile, but not jaundiced. Her chest was clinically clear, while the pulse was 110/min, regular and of good volume. The blood pressure was 70/50mmHg. The symphysio-fundal height was 38cm, longitudinal lie, and cephalic presentation with moderate tenderness of the uterus. The fetal heart rate ranged between 100 and 120/min. Vaginal examination revealed normal female external genitalia and no bleeding per vagina.

An assessment of a probably concealed Abruptio placenta with hemorrhagic shock was made. Double intravenous lines were set for her. She had an emergency lower segment caesarean section.
Surgery revealed haemoperitoneum of 1 litre with intact gravid uterus and adnexae. At this stage, general surgeons were invited. She was delivered of a live female baby in cephalic presentation with an Apgar score of 5 in the first minute and 7 in the fifth minute. Her birth weight was 2.43Kg and was handed over for the neonatologist's care. The placenta was posterior in the body of the uterus with retroplacental clots of about 250mls. The bladder was grossly normal. The hemoperitoneum was traced by the surgeons to the rupture of the right lobe of the liver. Two abdominal packs were applied to the hepatic bed after hemostatic sutures were administered to maintain hemostasis. She had four pints of blood transfused intraoperatively. Her estimated blood loss was 2 litres. The hepatic tamponade packs were removed 18hours postoperation after her clinical condition had been stabilized and with no bleeding from the drain sites.
She made satisfactory postoperation progress and was discharged home with her baby on the 16th day postoperation for follow-up in the postnatal and surgical outpatient clinics.
An abdominopelvic scan done at 6weeks postpartum was normal except for pockets of fluid collection seen at the right lower quadrant. The aspirated serous fluid that was sent for microscopy, culture and sensitivity and cytology reported no growth after 48hours of incubation. Cytopathology reported 2mls of greenish yellow fluid on macroscopy and smear consisted of few lymphocytes and cellular debris. No malignant cells were present. Diagnosis was negative for malignancy. She was finally discharged from the postnatal clinic 8weeks in satisfactory clinical condition.

Discussion

Hepatic rupture/hemorrhage is a rare but very serious complication of hypertensive disease of pregnancy. It is an important cause of maternal and fetal death[7]. The patient presented with hypertensive disease of pregnancy but developed spontaneous rupture of the liver. She was thought to have concealed type of abruptio placenta because of the clinical presentation of uterine tenderness, maternal shock, severe anaemia, and fetal distress. Placenta abruptio is a premature separation of a normally situated placenta before the delivery of the fetus. Bleeding resulting from this separation could be external or concealed[8]. The uterus is tetanic and painful with maternal hypotension and frequently, fetal death[8]. Placental abruption complicates approximately 1 to 2% of all pregnancies and remains a

significant cause of maternal and fetal morbidity[9]. Recent evidence support maternal hypertension disorders, maternal tobacco and cocaine use, age, multiple pregnancy, maternal thrombophilias and unexplained elevated maternal serum α-fetoprotein as risk for factors for abruptio[9]. The patient was resuscitated and planned for immediate delivery by emergency caesarean section to offer better chances of survival for the baby. Operation findings confirmed hepatic rupture. Specific symptoms of hepatic rupture could be established through identification of a triad: epigastric pain, hypotension without evident bleeding cause and clinical data of hypertensive disease of pregnancy[7,10]. The most frequent signs and symptoms of hepatic rupture were the sudden onset of abdominal pain, acute anemia and hypotension which were found in the patient presented[11]. Laboratory findings could include low platelet count and increased hepatic enzymes because most of these cases were associated with HELLP syndrome but this was not found in this patient[11]. Some authors advised that this disease should be considered when there occurs pain in the upper part of the abdomen and there are signs of hemorrhagic shock, even in the case of an uncomplicated pregnancy[2]. Ultrasonographic imaging is a valuable and readily available tool for the obstetricians in early diagnosis of this condition[10]. Prognosis becomes considerably positive if diagnosis is established early. A high index of suspicion and immediate recognition are keys to proper diagnosis and management of affected patients[12]. The multidisciplinary approach to the management of patients led to a remarkable decrease in the mortality rates[6,12]. Most patients died of hemorrhagic shock and organ failure[13]. The management should be aggressive by treating coagulopathy, hypovolemia or hemorrhagic shock, control of hepatic bleeding and delivery[4,5,14,15]. This patient had evacuation of the hematoma and hemoperitoneum; packing of the damaged liver after hemostatic sutures had been administered; draining of the operation site and delivery by caesarean section[15]. In every case fetal extraction by caesarean section constitutes the first therapeutic procedure[16]. Conservative management can be instituted in cases with hemodynamic stability[1,5]. Conservative management should consist of correction of hypovolemia and clotting disorders while surgical approach should be reserved for patients who cannot be stabilized hemodynamically[5]. More aggressive surgical techniques such as hepatic artery ligation or hepatic lobectomy, hepatic arterial embolization, total hepatectomy and liver transplant, or argon coagulation of hepatic artery should be reserved for refractory cases[13,15,17,18,19,20]. Since hematoma formation precedes hepatic rupture then, when diagnostic modalities such as sonography and computed

tomography identify patients with hematomas, these patients are at risk of rupture, and should be hospitalized until hematomas resolve[21].

Conclusion

In the reported case, spontaneous hepatic rupture resulted in a massive hemorrhage and aggressive therapy which involved the treatment of hemorrhagic shock, control of hepatic bleeding and delivery of the fetus was instituted.

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