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Case Report

Idiopathic spontaneous hemoperitoneum in pregnancy: a rare entity

Geeta Katheit Rai*, Niharika Aggarwal, Veena Vidyasagar, Sanjeevani Kaul

Department of Obstetrics and Gynecology, School of Medical Sciences and Research, Sharda University, Greater Noida, India

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***Correspondence:**
Dr. Geeta Katheit Rai,
E-mail: geetakatheit.06@gmail.com

ABSTRACT

Spontaneous hemoperitoneum in pregnancy (SHiP) is a very rare occurrence and is defined as sudden non-traumatic intraperitoneal bleeding in pregnancy and up to 42 days postpartum. The incidence is currently extremely difficult to estimate due to the lack of worldwide surveillance. We present a case of a 32-year-old G4P2L2A1 at 38 weeks of gestation. The patient was referred from a private hospital in a state of shock. The patient had a history of abdominal pain followed by fainting attack. The patient underwent laparotomy in view of suspected uterine rupture. Intraoperatively there was hemoperitoneum of unknown origin. The maternal and fetal outcome was poor in this case. In literature there are only very few reported cases of spontaneous hemoperitoneum in pregnancy.

Keywords: Idiopathic hemoperitoneum, Spontaneous hemoperitoneum in pregnancy, Intraperitoneal haemorrhage

INTRODUCTION

Spontaneous hemoperitoneum in pregnancy (SHiP) is defined as sudden non-traumatic intraperitoneal bleeding in pregnancy and up to 42 days postpartum. It is associated with high perinatal mortality and morbidity.[1] The incidence is currently extremely difficult to estimate due to the lack of worldwide surveillance. It is considered idiopathic when the source of bleeding is not identified during exploratory laparotomy. It is a very rare condition.[2] We report a rare case of idiopathic hemoperitoneum which presented with hypovolemic shock mimicking uterine rupture.

CASE REPORT

A 32-year-old lady referred from a 25 km away private hospital presented in the emergency department of our institute. She was G4P2L2A1 at 38 weeks of gestation with previous full term vaginal deliveries. The patient was referred in view of sudden 'Hyperglycaemia' detected at the referral hospital. History was elicited from the husband as the patient was unconscious at the time of admission. The patient was a booked antenatal case at a Government Hospital. At the day of presentation, she had pain abdomen in the morning and went to the private hospital for further management. About 5 hours after admission in that hospital, the patient felt dizzy, fell in the washroom and suddenly, the abdominal pain ceased. The blood sugar recorded at that time was 308 mg/dl. The patient was then referred to our hospital. Vitals recorded at the time of referral: Pulse rate-100/min, BP-90/50 mmHg, and SpO2-100% on room air. The patient had a spontaneous conception during the present pregnancy. She was a booked case, though had infrequent visits during the antenatal period. Most of the advised investigations were done. The antenatal period was uneventful. Obstetric score-G4P2L2A1-previous vaginal deliveries and spontaneous abortion at 2 months gestation.

At the time of presentation, the patient's general condition was poor. Her GCS score was 8/15, her radial pulse was not palpable and her BP not recordable. The patient's breathing was laboured with a respiratory rate of 32 cycles/min, SpO2was 60% on room air and 82% on 15l oxygen support. Her blood sugar was 150 mg/dl. On

examination, the patient had a pallor of 3+. On auscultation of the chest, there was tachycardia with a heart rate of 160 beats/min and her chest was clear. The patient's abdomen was distended with an abdominal girth of 110 cm and the abdomen was non-tender. On percussion of the abdomen, there was a dull note with no shifting dullness. Uterine contour could not be appreciated well and foetal parts were palpable. On auscultation, the foetal heart could not be localized. On vaginal examination, the cervix was 4-5 cm dilated and 2 cm in length. Membranes were present. The presenting part was high up. The provisional diagnosis of uterine rupture was made.

The patient was intubated, put on a ventilatory support and Inj Noradrenaline drip was started. SpO2 rose to 97%. Simultaneously, investigations were sent. The patient had 1 episode of generalized tonic-clonic seizure. On admission her ABG reports: pH - 6.3, pCO2 - 100 mmHg, pO2-22.4 mmHg, lactate-17mmol/l. Her haemoglobin was 5.1 gm/dl, platelet count 1.56 lakh/cumm.

Liver enzymes were raised with SGOT 744IU/l and SGPT 481 IU/. The patient's kidney functions were deranged. Her coagulation profile was normal. Her abdominal girth increased from 110 cm to 118 cm. Ultrasound of the abdomen was done which showed a single intrauterine fetus with no cardiac activity. There was fluid in peri hepatic and peri splenic areas which on tapping showed noncoagulable blood suggestive of hemoperitoneum. Explorative laparotomy was planned.

The patient's condition deteriorated from GCS 8/15 to 3/15. Her inotropic support was increased. She had an episode of cardiac arrest followed by 1 cycle of CPR and patient revived. The patient was taken up for exploratory laparotomy. There was hemoperitoneum of 1-1.2l with 800gm of clots in the peritoneal cavity. No rent was seen on the uterus. Incision over LUS was given. There was grade 2 MSL. A female baby with no sign of life was delivered weighing 2.8 kg.

The uterus was atonic, hence the decision for an obstetric hysterectomy was taken, and done. Part of the small intestine showed bluish discoloration. A call for a surgeon was sent. The spleen was grossly normal. There were cirrhotic changes in the liver with no evidence of hematoma or laceration. About 50-60 cm of jejunum had pre gangrenous changes. Small bowel, mesentery, and omentum showed no evidence of any bleeding. There was no evidence of any retroperitoneal hematoma. The large bowel was grossly normal. No source of hemoperitoneum was seen.

One unit of PRBC was transfused. As no source of hemoperitoneum was identified, patient's abdomen was closed. The patient had 2nd cardiac arrest during the immediate post operative period and was revived after good and effective CPR. After shifting the patient to ICU, she had a third cardiac arrest and could not be revived.

DISCUSSION

SHiP is a rare but life-threatening condition that is defined as blood within the peritoneal cavity of non-traumatic and non-iatrogenic aetiology.[3] The first case was described by Barber in a pregnant woman during labor in 1909.[4] The mortality rate is 8.6% in patients with arterial bleeding of defined origin, but when the bleeding site is not detected, the mortality rate can rise to 50%.[5] Various causes of hemoperitoneum in pregnancy are as follows.

Gynaecologic-obstetric causes

Uterine rupture, ectopic pregnancy, HELLP-syndrome with liver rupture, hematoma, placenta percreta, ectopic deciduosis, endometriosis, rupture of haemorrhagic ovarian cyst.

Rupture of a pelvic vessel

Uterine artery or vein, uterine varicose veins, utero-ovarian vessel, varix of broad ligament, idiopathic.

Non-obstetric-gynaecologic causes

Rupture of splenic artery aneurysm or vein, rupture of hepatic artery aneurysm or vein, rupture of a maternal umbilical vein. Coagulopathic haemorrhage, aortic aneurysm, visceral malignancy.

In a population-based study by Mazzocco et al for SHiP, there were a total 29 cases over the span of 3 years. This study showed higher chances of SHiP in women aged more than 35 years, women with previous surgery and women with ART. This study showed strong co relation of endometriosis with cases of SHiP6. Another Japanese retrospective study of 31 cases of SHiP also reported high diagnosis rate of endometriosis with SHiP1. In current case, there was no history of endometriosis as well as no evidence endometriosis during laparotomy.

Another gynaecologic cause of spontaneous intra-abdominal haemorrhage is ectopic deciduosis and rupture of pelvic vessels, probably due to a pathologic intrusion of the decidualized stroma into the vessel wall resulting in fragilization and eventual rupture of the vessel.[6] In the present case, no evidence of macroscopic lesions of deciduosis were seen.

In the study by Markou et al in 2015, two cases of idiopathic hemoperitoneum were reported in pregnancy and compared with other four cases which had been reported till then.[7] Usually, patient came to emergency with atypical complaints such as abdominal pain, nausea, vomiting and discomfort. Due to vague symptoms and normal examination findings, diagnosis of hemoperitoneum was delayed until patient developed hypovolemic shock. In all the cases, foetal outcome was good. However, in the present case there was fetal demise

which might have occurred due to hypovolemic shock leading to fetal hypoxia.

Management of idiopathic hemoperitoneum in pregnancy usually varies from case to case. In case of haemodynamic instability, exploratory laparotomy is required. Decision of caesarean section depends on the period of gestation, etiology of bleeding, and stability of both mother and fetus. In the study by Markou et al, caesarean section was not done, and bleeding was managed conservatively. Patients delivered later. In other cases, caesarean section was done along with laparotomy; likely indication being fetal distress due to fetal hypoxia caused by maternal hypovolemic shock. In cases, where the fetus is premature and there is no fetal distress, pregnancy can be continued with conservative management. In all the cases patients recovered after initial management whereas in the present case, unfortunately there was maternal mortality as well.

CONCLUSION

Idiopathic spontaneous haemorrhage during pregnancy is a very rare event but life-threatening event, for both the mother and the foetus. Given the potentially high perinatal mortality, emergency exploratory laparotomy should be performed by a multidisciplinary team. It generally presents with vague symptoms and is usually diagnosed after patient goes in hypovolemic shock. Prompt diagnosis and management can prevent maternal and fetal morbidity and mortality.

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