Abruptio Placentae Leading to Fetal Death and Adult Respiratory Distress Syndrome

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Introduction

The term abruptio placentae denotes separation of normally implanted placenta after 20 weeks of gestation and before birth of fetus. The diagnosis is commonly made in the third trimester. Maternal/fetal death may occur due to massive hemorrhage or various associated pathologies including disseminated intravascular coagulopathy (DIC), adult respiratory distress syndrome (ARDS) and renal failure which may be interrelated. Abruptio accounts for about 10 % of all third trimester still births. Hypertensive disorders, both preexisting and pregnancy induced are linked with abruptio [1], although hypertension may develop de novo following an abruptio. We present a case of severe concealed abruptio placentae with intrauterine fetal death (IUFD) who developed ARDS after emergency cesarean delivery.

Case Report

A 29 year old primigravida, a booked antenatal case at 32 weeks of gestation reported for routine antenatal checkup on 27 Aug 2007. Her LMP was 14 Jan 07 and expected date of delivery (EDD) was 21 Oct 07. Her blood pressure (BP) was 150/90 mm Hg. All her routine antenatal investigations were normal including ultrasonography and triple screening done at 18 weeks of gestation. All her previous blood pressure recordings were normal. She was operated for chocolate cyst right ovary in 2005 and was put on danazol therapy for six months, followed by ovulation induction with clomiphene citrate. She had no significant past medical history. In view of her high BP recording she was advised admission for monitoring of her BP and follow up. However she refused admission due to domestic problems. On 28 Aug 07 at around 0200 hours she developed severe pain abdomen and backache associated with nausea and vomiting. This was followed by loss of fetal movements. She was brought to labor room around 0300 hours on the same day. Clinical examination revealed, pallor, pulse 98/minute, BP 150/96 mmHg. Uterus

was 32-34 weeks in size, tense, tender and rigid. It was not possible to palpate fetal parts. Foetal heart sounds were absent. Vaginal examination revealed, cervix 1-2 cm dilated, 30-40% effaced and station -1. Clinical diagnosis of abruptio placentae with IUFD was made. Routine urgent investigations including coagulation profile were normal except for hemoglobin of 9.2gm/dL. Artificial rupture of membranes was attempted was unsuccessful. Oxytocin drip was started to assist vaginal delivery with close monitoring. After six hours trial of vaginal delivery there was no satisfactory progress in labor. Pallor and tachycardia had increased although BP remained around 150/90mm Hg. She was taken up for emergency cesarean delivery. On opening up of abdomen Couvelaire uterus was noted. There was blood in peritoneal cavity and placenta was totally separated with a large retroplacental clot. There were multiple subserous and submucous fibroids. A still born female weighing 1500 grams was delivered (Fig. 1). Patient had massive atonic post partum hemorrhage (PPH) which was managed by using prostaglandins and oxytocin. Total blood loss was around four litres. She was given six units of fresh blood and the patient made satisfactory recovery till fourth post operative day, remaining afebrile throughout. On fifth post operative day patient developed sudden progressive breathlessness and fall in SpO₂. Chest radiograph showed diffuse patchy dense infiltrates (Fig. 2). She was put on mechanical ventilation with positive end expiratory pressure (PEEP). After 48 hours of ventilatory support she had gradual improvement. Her BP returned to normal after four weeks and radiological clearing of lung fields took three weeks post ARDS.

Discussion

An abruptio placenta is one of the important causes of ante partum hemorrhage. The incidence is up to 1.5% in pregnancies overall and 0.3% in pregnancies at term [2]. Abruptio severe enough to kill fetus is less commonly seen (1:420 deliveries) [3]. The abruptio may be revealed type with blood loss from the lower genital tract or concealed where bleeding is contained within

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Fig. 1 : Still born female

the uterus. The latter typically accounts for about one third of cases and may be associated with disseminated intravascular coagulation (DIC). A unifying etiological concept is lacking, but underlying disease of the decidua and uterine vessel seems to explain the diversity of associated factors. By far the most commonly associated condition is hypertensive disorder during pregnancy. Amongst the other factors that are associated with the development of abruption, cigarette smoking features high with an increase in risk of abruption up to 90% [4]. Other causes includes direct abdominal trauma, sudden uterine decompression, external cephalic version, acquired and inherited thrombophilias, high parity, myoma uterus and cocaine abuse [5,6]. The classic signs and symptoms of abruptio placentae are vaginal bleeding, abdominal pain, uterine contractions, and uterine tenderness. Ultrasonography is not sufficiently sensitive to reliably either diagnose or exclude abruptio placentae [7] so valuable time should not be lost performing a sonogram in presence of obvious fetal/maternal distress. Resuscitation with fluids, blood and correction of coagulopathy must be done urgently. Women with preeclampsia complicated by abruption need particular care in resuscitation as they tolerate hypovolemia poorly because of contracted intravascular volume. Delivery must be expedited, preferably vaginal, however if delivery does not occur within a reasonable time recourse to cesarean may be necessary. Postpartum hemorrhage must be anticipated following a severe placental abruption and prophylactic uterotonic drugs should be considered. Myometrial contractility is impaired by fibrin degradation products rather than by presence of extravasated blood [8]. Some potential precipitators of

Fig. 2: Chest radiograph (AP view) shows diffuse patchy infiltrates

ARDS associated with pregnancy includes infection, preeclampsia/eclampsia, hemorrhage and amniotic fluid embolism. Progression of ARDS is extremely rapid and patients usually develop respiratory failure within 48 hours of initial injury. However in our case patient developed ARDS on fifth postoperative day and there was no evidence of infection, so we assume this delayed ARDS could be due to preeclampsia, abruptio placentae, hemorrhage or related to blood transfusion.

Conflicts of Interest

None identified

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