CASE REPORT

Postpartum Respiratory Failure

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Abstract

This is a case report of a 29 years old primigravida who at 32 weeks of gestation presented with severe pain abdomen and backache. She had a massive concealed placental abruption that led intrauterine fetal death. Due to the progressively deteriorating maternal condition a cesarean section was performed under general anesthesia to deliver the dead fetus. After five days the patient developed acute respiratory failure and needed mechanical ventilation. The common causes of respiratory failure associated with pregnancy are discussed.

Keywords: Abruptio placentae, pre-eclampsia, respiratory failure, acute respiratory distress syndrome (ARDS).

INTRODUCTION

This is a case report of a patient who suffered a massive concealed placental abruption at 32 weeks of gestation that led to demise of her intrauterine fetus. A cesarean section was performed under general anesthesia to deliver the dead fetus. After a relatively uneventful postoperative period of five days the patient developed acute respiratory failure and needed mechanical ventilation.

The common causes of respiratory failure associated with pregnancy are acute respiratory distress syndrome (ARDS), pulmonary thromboembolism, amniotic fluid embolism, venous air embolism, sepsis, and pre-eclampsia/eclampsia related HELLP (**h**emolytic anemia, **e**levated liver function tests, **l**ow **p**latelets) syndrome. The purpose of this case report is to emphasize the fact that arriving at an exact etiologic diagnosis in a case of respiratory failure in the postpartum period is challenging and difficult. The cornerstone of successful management in such a case is quality supportive care.

CASE REPORT

A 29 years old primigravida at 32 weeks of gestation presented with severe pain abdomen and backache. She complained of nausea and vomiting. There was loss of fetal movements. Clinical examination revealed a 70 kg patient with pallor, a pulse of 98/minute, arterial pressure of 150/96 mm Hg, a uterus 32 to 34 weeks in size that was tense tender and rigid. It was not possible to palpate fetal parts. Fetal heart sounds (FHS) was absent. Vaginal examination revealed a cervix 1 to 2 cm dilated, 30 to 40% effaced and head at station-1. Ultrasonogarphy was inconclusive. Clinical diagnosis of abruptio placentae with intrauterine fetal death was made.

An attempt was made at vaginal delivery for six hours, but there was no progress and the patient became increasingly apprehensive and complained of severe abdominal pain. The abdomen was tender to palpate and she developed tachycardia and hypotension. Her rapidly deteriorating condition prompted an emergency cesarean delivery. Routine urgent investigations including coagulation profile were normal except for hemoglobin level of 9.2 gm%. Due to anticipated hemodynamic instability the patient was administered General Anesthesia. On opening up of abdomen couveliaire uterus was noted and there was blood in peritoneal cavity and peritoneal fluid was port wine colored. Placenta was totally separated with a large retro placental clot. There were multiple subserous and submucous fibroids. A still born female weighing 1500 grams was delivered. The patient had a massive atonic postpartum hemorrhage (PPH) which was managed by using prostaglandins, oxytocin and restoring intravascular volume with one and half liters of crystalloids and one liter colloids. Total blood loss was around two and half liters. She was given six units of fresh blood during postoperative period. Despite abruption her coagulation profile

was normal. She made satisfactory recovery by the fourth postoperative day and was afebrile throughout. However on the fifth postoperative day the patient developed progressively worsening breathlessness with falling oxygen saturation. The patient was extremely restless and hypoxic with central cyanosis with SpO_2 of 84%. She had an arterial pressure of 190/120 mm Hg and heart-rate of 170 per minute. X-ray chest showed diffuse patchy dense nonhomogenous opacities. Arterial blood gas analysis could not be performed due to unavailability in the hospital. The echocardiography was normal and therefore cardiogenic pulmonary edema was considered unlikely.

The patient was propped up in bed, administered oxygen by facemask, and given diuretics and morphine intravenously. There was no clinical improvement. She was put on conventional mechanical ventilation, initiated in the intermittent positive pressure ventilation (IPPV) mode and thereafter put on synchronous intermittent mandatory ventilation (SIMV). The tidal volume was set at 500 ml with a respiratory rate of 20 per minute, fraction of inspired oxygen of 50% and positive end expiratory pressure (PEEP) of 5 to 10 cm of water. Plateau pressures of less than 30 cm of water were targeted. After 48 hours she had gradually but progressively improved and was liberated from ventilatory support. Throughout this period her arterial pressure had remained high and was managed with infusion of antihypertensive drugs such as esmolol and thereafter with oral metoprolol. The blood pressure returned to normal four weeks postpartum and radiological clearing of lung fields took three weeks.

DISCUSSION

There are certain physiologic changes in pregnancy that make it more difficult for the pregnant woman to sustain any type of respiratory insult. Respiratory failure may reflect reduced pulmonary reserve to deal with the stresses of a conventional respiratory insult or the occurrence of pulmonary diseases specific to pregnancy.

The common causes of respiratory failure in pregnancy are acute respiratory distress syndrome, pulmonary thrombo-embolism, amniotic fluid embolism, venous air embolism, sepsis, and pre-eclampsia/eclampsia related HELLP (**h**emolytic anemia, **e**levated **l**iver function tests, **l**ow **p**latelets) syndrome. The various causes are listed in Table 1. Pulmonary edema usually represents a final common pathway of several of these above complications of pregnancy and the peripartum period.[1]

Table 1: Causes of respiratory failure in a pregnant patient

- Thromboembolism
- Amniotic fluid embolism
- Venous air embolism
- Aspiration of gastric contents
- Respiratory infections
- Asthma
- HELLP (**h**emolytic anemia, **e**levated **l**iver function tests, **l**ow **p**latelets) syndrome
- β^2-Adrenergic tocolytic therapy
- Pneumomediastinum and pneumothorax
- Acute respiratory distress syndrome
- Cardiomyopathy.

Our patient was a known case of pregnancy induced hypertension on regular follow-up. However, the arterial pressures measured were consistently on the higher side and may have contributed as a risk factor to abruptioplacentae.

Abruptioplacentae 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] The condition may be associated with disseminated intravascular coagulation (DIC). By far the most commonly associated condition is hypertensive disorder during pregnancy.[3] The classic signs and symptoms of abruptio placentae are vaginal bleeding, abdominal pain, painful uterine contractions, and uterine tenderness. Blood loss may be over 50% of maternal blood volume. Resuscitation with fluids, blood and correction of coagulopathy must be urgently done. Women with pre-eclampsia 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.

Abruptio and pre-eclampsia or eclampsia related respiratory failure is known to occur at a mean of 71 hours after delivery. Pulmonary edema in such cases is often associated with the dysfunction of other organ systems including disseminated intravascular coagulation (49 percent), acute renal failure (27 percent), hypertensive crisis (16 percent), cardiopulmonary arrest (13 percent), or cerebral edema (5 percent).[4]

Amniotic fluid embolism is another cause of respiratory failure during pregnancy.[5] This rare but catastrophic illness is associated with an extremely high mortality rate and occurs almost always occurs during labor and delivery (the time frame making it an unlikely possibility in our case). The pathogenesis of the disease likely involves the transfer of amniotic fluid-containing vasoactive substances into the maternal circulation. This results in the development of acute and rapid hypoxemia, acute pulmonary hypertension, and systemic hypotension. Aspiration of fetal squamous cells from a wedged right heart catheterization is no longer thought confirmatory.

Amongst the other causes, pneumonia is a relatively common cause of respiratory failure in pregnant patients. It usually is community acquired however may result from any infective insult. Transfusion associated acute lung injury (TRALI) should be considered whenever dyspnea, hypoxemia, and pulmonary infiltrates occur during or within six hours after transfusion of any blood product.[6] The last unit of blood was transfused three days prior to onset of respiratory failure in our patient making TRALI a remote possibility.

The diagnosis of pulmonary embolism and deep vein thrombosis could not be ruled out clinically or with laboratory assistance, as the possible cause and the patient was initiated on low molecular weight heparin. A normal echocardiography study performed during and after illness excluded cardiogenic pulmonary edema.

Treatment of respiratory failure in pregnancy is largely supportive, including mechanical ventilation, hemodynamic support, nutrition, and prophylaxis against thromboembolism. No specific therapy has as yet been proven effective for ARDS, other than treating the underlying cause.

REFERENCES

1. Sciscione AC, Ivester T, Largoza M, Manley J, Shlossman P, et al. Acute pulmonary edema in pregnancy. Obstet Gynecol 2003;101(3):511-15.
2. Sheirner E, Shoham-Vardi1, Hallak M, et al. Placental abruption in term pregnancy: Clinical significance and obstetric risk factors. J Matern Fetal Neonatal Med 2003;13:45-49.
3. Ananth CV, Smulian JC, Vintzileos AM. Incidence of placental abruption in relation to cigarette smoking and hypertensive disorder during pregnancy: A metaanalysis of observational studies. Obstet Gynecol 1999;93:622-28.
4. Ernst A. Critical illness during pregnancy and peripartum. In: Up to date, Rose BD (Ed), Waltham MA, 2007.
5. Gilmore DA, Wakim J, Secrest J, Rawson R. Anaphylactoid syndrome of pregnancy: A review of the literature with latest management and outcome data. AANA J 2003;71(2):120-26.
6. Kleinman S, Caulfield T, Chan P, Davenport R, McFarland J, et al. Toward an understanding of transfusion-related acute lung injury: Statement of a consensus panel. Transfusion 2004; 44(12):1774-89.