

Acute Pulmonary Edema Following Caesarean Delivery

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Abstract

The presentation of acute dyspnoea during pregnancy and immediate postpartum period is less common, but should bring a number of important conditions to the mind of the attending physician. Pulmonary embolism, amniotic fluid embolism, pulmonary aspiration, undiagnosed cardiac disease and peripartum cardiomyopathy are some of the potentially devastating causes that must be considered in differential diagnosis. Women of child bearing age may have asymptomatic mitral valve disease which becomes unmasked during the hemodynamic stress of pregnancy particularly in third trimester and labour. Irrespective of cause, initiation of appropriate resuscitation is the priority. After resuscitation physician should focus on diagnosis and specific treatment of clinical condition. Here we present a diagnostic dilemma in acute pulmonary edema following caesarean delivery managed successfully with drug therapy and noninvasive ventilation. Echocardiography proved to be of great value as diagnostic tool.

Keywords: Postpartum pulmonary edema; Varied etiology; Echocardiography

Introduction

The incidence of acute pulmonary edema during pregnancy is 0.08% [1]. Maternal heart disease complicates about 0.2-3% of pregnancies, majority of which have an underlying etiology of rheumatic origin [2]. Cardiac disease in pregnancy is associated with high maternal and fetal morbidity. Pulmonary edema can be of cardiogenic or noncardiogenic origin with acute or subacute onset accompanying respiratory insufficiency. Physiological cardiovascular changes during pregnancy in parturient impose diagnostic dilemma in establishing a diagnosis of heart disease on history taking and general examination particularly in under evaluated patient. Hence the differential diagnosis of pulmonary edema must include undiagnosed cardiac disease. Here we illustrate discussion on various possible etiologies of postpartum pulmonary edema, importance of echocardiography as a diagnostic tool and role of non-invasive ventilation in management in addition to drug therapy.

Case Report

A 23-year old female, unregistered, G₂P₂L₁, term gestation posted for emergency cesarean section in view of non-progress of labour. Her past medical, surgical and obstetric history unremarkable. Patient gave one day history of cough and cold without expectoration and fever. On day of surgery, patient was afebrile, heart rate 125/min, blood pressure 96/60 mm of Hg, respiratory rate 28/min. cardiorespiratory examination revealed tachypnea with clear air entry and tachycardia with regular rhythm, normal heart sounds without any murmur. Pulse oximetry reading on room air was 98%. Blood investigations were within normal limit except for thrombocytopenia (87000 mm³)

Following spinal anesthesia she had two episodes of vomiting consistent with hypotension, treated with injection ephedrine 18 mg intermittent intravenously and rapid fluid boluses of crystalloid and colloid. Total fluid infused was 1000 ml crystalloid and 500 ml colloid. Patient had persistent tachycardia and tachypnea throughout procedure. Baby cried immediately after birth and had Apgar score of 9/10. Injection oxytocin 20 U was given slow drip intravenously. On repositing uterus into the peritoneal cavity patient started complaining pain in abdomen and was given injection Pentazocine 18 mg intravenously. Patient was comfortable after this but developed cough with streaky mucoid expectoration towards the end of surgery. Total urine output was 250 ml, clear and blood loss was 800 ml.

After shifting to recovery room, patient became agitated and

experienced sudden onset dyspnoea and cough with mucoid expectoration. Cardiorespiratory examination revealed tachypnea (40/min) with use of accessory muscles, crepitation bilaterally at the base and apex with reduced breath sounds at bilateral base and tachycardia (140 bpm) with regular rhythm and no murmur. Saturation was 85% on oxygen mask (FiO₂-0.6) Peripheral edema was noted in both lower limbs. She was given injection furosemide 40 mg intravenously and transferred to intensive care unit.

Postoperative electrocardiogram showed sinus tachycardia and chest radiograph revealed bilateral pulmonary edema. Echocardiography showed mitral valve area of 1.6 cm² with mild dilatation of left atrium. No evidence of pulmonary hypertension, pericardial effusion, pulmonary embolism, clots, vegetation, regional wall motion abnormality. Arterial blood gas analysis revealed pH 7.42, pCO₂ 39 mm Hg, pO₂ 73 mm Hg and HCO₃ 25 mmol/L with nasal prongs (FiO₂-0.4). She was given noninvasive ventilation with BI-PAP (pressure support 16 and Positive end expiratory pressure 8) and started on diuretics, beta blocker and antibiotics. She showed marked symptomatic improvement and continued with noninvasive ventilation for 24 hours. Next day her pulmonary edema was largely resolved as assessed by repeat chest radiograph and complete improvement in cardio respiratory symptoms. Blood investigations included Hb-14.3 gm%, TLC-11.7 cmm, platelets-83 mm³, normal renal profile and electrolytes. Blood was negative for malaria parasite, dengue and typhoid antibodies. Coagulation profile was normal except raised fibrinogen and D-dimer which was correlated with postoperative inflammatory status. She was discharged after 7 days on continued treatment with metoprolol and furosemide.

Discussion

Pulmonary edema can be of cardiogenic origin due to underlying cardiac disease, myocardial ischemia, peripartum cardiomyopathy or

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noncardiogenic origin such as secondary to iatrogenic fluid overload, thyroid disease, tocolytic therapy, acute respiratory distress syndrome [1]. Preeclampsias, use of tocolytic agent, iatrogenic fluid overload, underlying cardiac disease are the important and common causes of pulmonary edema in parturient.

Preeclampsia patients are at increased risk for development of pulmonary edema. Decreased colloid osmotic pressure and endothelial damage results in leakage of fluid in pulmonary interstitial space which is accentuated by left ventricular dysfunction and increase in peripheral vascular resistance [3].

Iatrogenic fluid overload is commonest cause of pulmonary edema near term 4. Rapid transfusion of crystalloid and colloid in term patient with preexisting increased blood volume added with salt and water retention secondary to oxytocin administration must have made our patient vulnerable to volume overload [4]. This can be treated with judicious use of diuretics and fluid restriction. Injection oxytocin administration by slow intravenous drip and ergometrine by intramuscular injection helps in avoiding these adverse clinical effects [5].

Pulmonary edema secondary to undiagnosed cardiac disease is most probable cause of dyspnoea in our case as she was diagnosed to have mild mitral stenosis on echocardiography. Her previous delivery was uneventful probably because of progressive nature of disease which has evolved upto present size. Rapid transfusion of intravenous fluid to correct hypotension, auto transfusion due to uterine contraction, increase in cardiac output upto 31% and unmasking of venocaval compression must have resulted in marked increase in blood volume leading to pulmonary edema in setting of mitral stenosis [6,7]. In pregnancy with mitral stenosis, high cardiac output combined with a decrease filling time due to tachycardia results in increase left atrial pressure and hence pulmonary edema [2,7]. This article highlights the fact that, cardiac disease may only become evident in the late stages of pregnancy, after which the patient can easily decompensate with the additional hemodynamic stresses such as neuraxial anesthesia [2]. Useful diagnostic tool like echocardiography has proven to be of great help in pointing towards the certain diagnosis. It has helped in our patient in timely diagnosis and definitive management of pulmonary edema. many Management includes noninvasive ventilation as it provides high inspired oxygen concentration, displaces fluid from alveoli into pulmonary and subsequently systemic circulation, decrease work of breathing and decrease the need for tracheal intubation [8]. Thus it helps in improving physiological and clinical outcome. Drug therapy should include diuretics (furosemide bolus 20-40 mg over 2 min IV) to promote venodilation and diuresis, morphine (2-3 mg) to promote venodilation and anxiolysis [9] and Beta blocker to prevent maternal tachycardia thus reduces the incidence of pulmonary edema [7].

Other causes of pulmonary edema includes pulmonary embolism, as pregnancy is hypercoagulable disease. High incidence is found in postpartum period. Clinical features include tachycardia, tachypnea, chest pain and desaturation. Echocardiography excluded the diagnosis

of pulmonary embolism. Pneumonia can also result in acute onset dyspnoea and it is usually associated with elevated temperature, fatigue, rigors, and leukocytosis. Amniotic fluid embolism is diagnosis of exclusion which manifest as unexplained cardio respiratory collapse and usually confirmed at autopsy by the presence of fetal squamous cells in the maternal pulmonary circulation [10]. Peripartum cardiomyopathy is rare entity associated with left ventricular ejection fraction less than 45%. Echocardiography should be performed in all patients with suspected peripartum cardiomyopathy. Echocardiography findings will demonstrate left ventricular enlargement, mitral and tricuspid regurgitation and possibly a small pericardial effusion [1].

Conclusion

Postpartum dyspnoea has a wide variety of etiologies. Appropriate and timely resuscitation followed by careful, stepwise evaluation of symptoms and related findings to determine the underlying cause. Echocardiography plays important role in patients with diagnostic dilemma. Conservative management with non-invasive ventilation and medical line of treatment results in good recovery.

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