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*Case Report*

# Acute Postpartum Pulmonary Edema in a 32-Year-Old Woman Five Days after Cesarean Delivery

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***Abstract***

*Acute dyspnea after pregnancy is a rare presentation, and a number of important conditions may accompany it. Pulmonary embolism, amniotic fluid embolism, pneumonia, aspiration and pulmonary edema are some of the potential causes that must be considered. The percentage of pregnancies that are complicated by acute pulmonary edema has been estimated 0.08%. The most common contributing factors include the administration of tocolytic agents, underlying cardiac disease, iatrogenic fluid overload and preeclampsia. Here we report a case of 32-year-old woman of 5th postpartum day following lower uterine cesarean section with acute dyspnea from her first pregnancy who was admitted in coronary care unit with history of one episode of raised blood pressure 160/90 mm Hg and cough on 1st postoperative day. Clinical examination and relevant investigations explored that it was a case of bilateral pulmonary edema. Patient was kept in ventilator and was treated with nitroglycerine (GTN), frusemide and ACE inhibitor. After diuresis, considerable improvement was observed in her respiratory status. From the 4th day, the patient became hemodynamically stable and was weaned off the ventilator. After five days, all the biochemical parameters became normal and she had no dyspnea.*

***Key words:*** *Acute postpartum pulmonary edema, Cesarean delivery*

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## Introduction

Pulmonary edema of cardiac origin is a common medical condition. It can vary in severity and may be chronic, subclinical or acute with associated severe respiratory compromise. Resuscitation is the foremost priority, followed by formulation of a differential diagnosis to address the underlying condition. In a young previously healthy postpartum patient, the differential diagnoses must be expanded to include some less prevalent causes, such as peripartum cardiomyopathy and cardiac failure secondary to tocolytics. A clinician needs to be aware of the pertinent physiologic differences that accompany pregnancy, such as increased cardiac output, expansion of blood volume, physiologic anemia and decreased systemic vascular resistance. Here we report a postpartum patient who presented with acute pulmonary edema with severe respiratory compromise.

## Case Report

A 32-year-old woman of fifth postpartum day following LUCS with acute dyspnea from her first pregnancy arrived at a private clinic. Her only previous respiratory complaint was mild cough. She had one episode of rising BP 160/90 mm Hg on 1st postoperative day. The patient reported a 1-day history of mild cough that progressed to severe shortness of breath and chest heaviness while she

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was lying in bed. She looked pale, unwell and was only able to speak 1-2 words. Her temperature was normal, oxygen saturation was 75% on room air, pulse rate was 140/min, blood pressure was 160/100 mm Hg and respiratory rate was 30/min. On auscultation, breath sounds were diminished and basal crepitations were present at the lung bases. Peripheral edema was noted. Peripheral $O_2$ saturation was 85% on $O_2$ inhalation with face mask. Her pulse rate was 130/min, ECG showed sinus tachycardia and chest radiography revealed bilateral pulmonary edema. Peripheral cyanosis was increasing and intravenous diuretics were given. Clinical condition of the patient was not improved. Then she was shifted to CCU in another private hospital for cardio-respiratory support. In CCU, blood gas analysis was done and based on its reports the patient was kept in ventilator with $FIO_2$ 80%. Nitroglycerine (GTN) was started at an infusion dose rate of 5 to 10 µg/min and the dose rate was increased by 5 µg/min every 5 minutes till desired effect, frusemide was started 100 mg IV bolus followed by continuous infusion at 40 mg/hour and ACE inhibitor was given sublingually. After diuresis, considerable improvement was observed in her respiratory status. $FIO_2$ reduced to 70%. Her investigation reports are mentioned below. WBC 18,000/cu mm, ESR 100 mm in 1$^{st}$ hour, SGPT 38 U/L, serum albumin 3.8 gm/dL, serum creatinine, urea, calcium values were normal, CK-MB was 51 U/L rising to 79 U/L, serum potassium 3 mmol/L, ECG showed sinus tachycardia with anteroseptal MI, urinary albumin (+), sugar (–) on the 1$^{st}$ day. After 72 hours, ECG showed normal sinus rhythm with LVH, echocardiography was normal, WBC 13000/cu mm, ESR 60 mm in 1$^{st}$ hour, blood pressure 140/90 mm Hg, chest radiography normal, Hb 9.6 gm/dL. After 4 days, her pulmonary edema was largely resolved, $O_2$ saturation 99%, ECG was within normal limit. She was hemodynamically stable and was weaned off the ventilator. After 5 days, her $O_2$ saturation was 98% on room air, pulse rate 90/min, blood pressure 140/80 mm Hg and she had no dyspnea. Then she was discharged with medications which included antibiotics, frusemide, ACE inhibitor, steroid inhaler and bromazepam.

## Discussion

The differential diagnoses for dyspnea without pulmonary edema include pulmonary embolism, amniotic fluid embolism, pneumonia, foreign body aspiration, psychogenic dyspnea. Dyspnea with pulmonary edema includes cardiogenic and noncardiogenic causes. Cardiogenic causes are peripartum cardiomyopathy, preeclampsia related with heart failure, underlying cardiac disease (e.g. valvulopathy), myocardial ischemia and sepsis with poor cardiac output. Noncardiogenic causes are iatrogenic fluid overload, thyroid disease, tocolytic therapy or medication-related sepsis and acute respiratory distress syndrome. In this case, the results of subsequent investigations appeared to rule out pulmonary embolism, amniotic fluid embolism, pneumonia and sepsis. Ischemic changes in ECG and elevated level of cardiac markers were found, but echocardiogram was normal. Careful review of the patient's charts revealed no tocolytic therapy. Postpartum cardiomyopathy (PPCM) is a rare entity, but a mortality rate of 50% has been suggested.[1] The cause of this form of cardiomyopathy is unknown. It is defined by four criteria.

1. Absence of an identifiable cause of pulmonary edema.
2. Development of cardiac failure within the last month of pregnancy or within 5 days of delivery.
3. Absence of recognizable cardiac disease before the last month of pregnancy.
4. Left ventricular systolic dysfunction demonstrated on echocardiography.

The actual incidence of the illness varies largely by geographic region, with an incidence of 1 in 15,000 people in the United States and 1 in 100 people in Zaire, Nigeria.[2,3] The causes of PPCM remain unknown, but a number of risk factors have been proposed.[4]

a) Age greater than 30 years
b) Multiparity
c) African descent
d) Pregnancy with multiple fetuses
e) A history of preeclampsia, eclampsia or postpartum hypertension
f) A history of maternal cocaine use

Postpartum cardiomyopathy most commonly presents with dyspnea, but presenting complaints may include cough, orthopnea, paroxysmal nocturnal dyspnea and chest pain.[1] Echocardiography should be performed in all patients with suspected PPCM

Echocardiography findings will demonstrate left ventricular enlargement, mitral and tricuspid regurgitation and possibly a small pericardial effusion.[5] Women with documented PPCM should be warned that there is a high risk of cardiac deterioration and death with any subsequent pregnancy.[6]

In our case, electrocardiogram demonstrated sinus tachycardia but echocardiography was normal. This along with other diagnostic criteria, made PPCM highly unlikely in our patient. Preeclampsia is an illness that is characterized by proteinuria and hypertension occurring after 20$^{th}$ week of gestation. Edema may also be present, although it is not essential for diagnosis. Patient with preeclampsia or eclampsia may develop refractory hypertension, neurologic dysfunction, renal failure, liver rupture or failure, hemolytic anemia, elevated liver enzymes and low platelet count syndrome, pulmonary edema, and/or disseminated intravascular coagulation which is usually associated with concomitant placental abruption. The pathogenesis of edema associated with preeclampsia and eclampsia is multifactorial. The incidence of pulmonary edema may be as high as 3% with this illness.[7,8]

Excessive elevations in pulmonary vascular hydrostatic pressure compared with plasma oncotic pressure may produce pulmonary edema in some women, particularly during the postpartum period. However, not all preeclamptic patients with pulmonary edema demonstrate this phenomenon.[8] The treatment of preeclampsia and eclampsia-related pulmonary edema is largely supportive and should include supplemental oxygen and fluid restriction. Definitive treatment is delivery of the fetus when the mother is stabilized, which usually leads to resolution of the symptoms within 48 hours. Diuretics should be administered only in the presence of clinically significant fluid overload. Consideration of actual circulatory volume and the possibility of reducing placental perfusion should be kept in mind. Our patient did not meet the case definition of preeclampsia. She did not have proteinuria (24-hour urine sample was negative) and had no pre- or postnatal record of proteinuria.

In one case series, 26 of the 37 patients developed pulmonary edema postpartum (at a mean of 71 hours after delivery), and 4 patients died.[9] Older and multigravida patients appeared to be at increased risk of pulmonary edema in this setting. Pulmonary edema was often associated with the dysfunction of other systems. Of the 37 patients, 18 had disseminated intravascular coagulation, 10 had acute renal failure, 2 had a hypertension crisis, 5 had a cardiopulmonary arrest and 2 had cerebral edema.[9]

Amniotic fluid embolism is a rare but potentially fatal outcome of pregnancy. The classic presentation involves the acute onset of severe dyspnea, hypoxemia and hypotension, followed within minutes by cardiac arrest. It usually occurs during labor and delivery.[10] Although it has been reported following uterine trauma and abortion, respiratory failure and disseminated intravascular coagulation frequently develop if patients survive. The patient typically has a sudden deterioration in her condition and may have cardiac arrest. One study on 46 patients found a dismal maternal mortality rate of 61%, with neurologically intact survival seen in 15% of women.[10] In the same study, the authors found that cases of amniotic fluid embolism had similar characteristics of an anaphylactic reaction, suggesting that actual pathology may be a severe allergic reaction. True confirmation of the diagnosis can be complicated and controversial. Using a Swan-Ganz catheter to obtain fetal squamous cell from the pulmonary artery was formerly the gold standard.[11,12] However, the specificity of this finding is unclear, as squamous cells can be found in the maternal pulmonary circulation in the absence of clinical findings of amniotic fluid embolism.[12] A study by Vanmaele and colleagues[13] emphasizes the role of pulmonary artery catheterization to diagnose and monitor obstetric patient with respiratory compromise and left ventricular dysfunction. The diagnosis is usually made on the basis of clinical signs and symptoms. In our patient case, a Swan-Ganz catheter was not used. However, the onset of symptoms 5 days after delivery, absence of hypotension, rapid response to vasodilator therapy and the lack of left ventricular dysfunction on her echocardiogram made the diagnosis of amniotic fluid embolism extremely unlikely.

Iatrogenic fluid overload is another possible cause, but a review of perioperative period of our patient revealed no significant fluid administration. Unfortunately we did not have a record of her oral fluid intake in the postoperative period during her

staying in private clinic and also at home. Sciscione et al[14] reviewed 10 years of pregnancy data to examine pulmonary edema in pregnancy. Of the 20 patients in their study with postpartum pulmonary edema, the most common attributable cause was iatrogenic fluid overload (patient had a positive fluid balance of 6022 mL, in the preceding 48-hour before diagnosis). Although a record of true intake-output of fluid was not kept for this patient, we could not rule out iatrogenic fluid overload as a contributing factor. However, the absence of tocolytic therapy and her presentation 5 days postpartum reduce the likelihood that volume overload was the sole source of her pathology.

One episode of hypertension in the immediate postoperative period and subsequent pulmonary edema in the 5$^{th}$ postoperative day is an uncommon clinical scenario. Initial resuscitation and investigations should be taken into consideration for multiple possibilities. Positive pressure ventilation and symptomatic support will often allow enough time to identify a probable cause. In our case, postpartum hypertension causing acute MI led to left heart failure and pulmonary edema.

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