Acute Postpartum Pulmonary Edema: A Case Report

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Acute pulmonary edema after pregnancy is rare. Pulmonary embolism, pneumonia, aspiration and pulmonary edema are some of the potentially devastating causes that should be considered. We report a case of a previously healthy 45 year-old woman had pulmonary edema 3 hours after a normal vaginal delivery at a local clinic. Two days before admission, the patient had received tocolytic therapy to suppress premature labor. After medical treatment, her symptoms subsided and a chest radiograph showed resolution of pulmonary edema 12 hours later. She was discharged the next day in stable condition.

Key words: postpartum, acute pulmonary edema, tocolytic agent

Introduction

Postpartum pulmonary edema is a rare clinical entity. Pulmonary edema of cardiac origin is a common medical condition that can range in severity from chronic, to subclinical to acute, with accompanying severe respiratory compromise. In a young, previously healthy postpartum patient, the differential diagnosis must be expanded to include some less prevalent causes, such as peripartum cardiomyopathy and cardiac failure secondary to tocolysis¹. Herein, we report a case of postpartum noncardiogenic pulmonary edema with respiratory distress after use of a tocolytic agent.

Case Report

A 45-year-old woman G₂P₁ presented to the emergency department (ED) with progressive dyspnea 3 hours after normal vaginal delivery at a local clinic. She had no remarkable cardiac disease, hypertension, respiratory tract infection or significant allergies. She had no family history of cardiac or respiratory disease. Two days previously, the patient had received tocolytic therapy to suppress premature labor. Her baby had been delivered by spontaneous vaginal delivery at 37 weeks. The patient denied visual changes, headache, and hand and face swelling. No lower limb swelling was noted. On physical examination, she had marked respiratory distress and chest auscultation revealed basal crackles with reduced breath sounds bilaterally. The rest of the physical examination was unremarkable. Her blood pressure was 110/68 mmHg, heart rate 120 beats/ minute, respiratory rate 24 breaths/min, body temperature 37.4°C, and oxygen saturation on room air 89%. Electrocardiography showed sinus tachycardia. Her chest radiograph revealed increased infiltration bilaterally (Fig. 1), compatible with acute pulmonary edema. Laborotary data showed mild leukocytosis and cardiac makers, C-reactive protein and D-dimer were within the normal range. A cardiologist was consulted and immediate
echocardiography showed normal ventricular function with an ejection fraction of 66%, trace mitral and tricuspid regurgitation, normal wall motion and no pericardial effusion, clots or shunt. Arterial blood gas analysis showed the pH was 7.43, Pa CO$_2$ 38 mmHg, PaO$_2$ 59 mmHg and HCO$_3$ 28 mmol/L on 100% oxygen. Initial resuscitative measures in the ED included oxygen administration by nonrebreather mask, which increased her oxygen saturation to 95%. Furosemide was administered intravenously at a dose of 40 mg. After aggressive medical treatment, her symptoms gradually subsided and a repeat chest radiograph (Fig. 2) taken 12 hours later showed resolution of pulmonary edema. Urine output was 1700 mL seven hours after initial treatment and she showed considerable improvement in her symptoms. Urinalysis revealed no evidence of urinary tract infection. Blood cultures were negative. The patient received close observation in the ED overnight with monitoring of oxygen saturation, blood pressure and electrocardiography. The next day, 12 hours after arrival in the ED, her pulmonary edema was largely resolved and oxygen saturation was 98% on room air. She was discharged in stable condition. The patient was doing well at follow-up 9 months after discharge.

**Discussion**

*It has been estimated that 0.08% of pregnancies are complicated by acute pulmonary edema. Despite the low rate of occurrence, there is significant morbidity, and mortality has been reported with this diagnosis*.$^{(1)}$ The most common contributing factors include underlying cardiac disease, the use of tocolytic agents, iatrogenic fluid overload and preeclampsia.$^{(1,2)}$

According to Dunne et al$^1$, the differential diagnosis for postpartum dyspnea includes the following:

***without*** pulmonary edema
- pulmonary embolism, amniotic fluid embolism,
- pneumonia, foreign body aspiration, psychogenic dyspnea

***with*** pulmonary edema
- **cardiogenic**: peripartum cardiomyopathy,
- preeclampsia-related heart failure, underlying cardiac disease, myocardial ischemia and sepsis with poor cardiac output
- **noncardiogenic**: iatrogenic fluid overload, thyroid disease, tocolytic therapy or medication related
However, our investigation ruled out pulmonary embolism, amniotic fluid embolism, pneumonia and sepsis. Our patient was treated with a tocolytic agent (β-adrenergic agonist) for 2 days. According to a physician from the local clinic, iatrogenic fluid overload could be ruled out because there was no history of administration of large amounts of fluid before or during delivery. Three hours after a normal vaginal delivery, our patient suffered from progressive dyspnea. Cardiac disease was ruled out as there was no evidence of abnormal cardiac markers or abnormalities on the echocardiogram. Tocolytic agents, which include terbutaline, ritodrine, salbutamol, and isoxsuprine, suppress premature uterine contractions during pregnancy. These β-adrenergic agonists increase intracellular cyclic adenosine monophosphate levels, thus decreasing muscular contraction. Pulmonary edema has been reported in association with the short-term use of β-adrenergic agonists (average 54 hours) in late pregnancy with an incidence of approximately 0 to 4.4%\(^3,4\). Pulmonary edema occurs during current or recent (< 24 hours) usage or appears less than 12 hours postpartum when tocolytic therapy has failed\(^5\). In previous reports, using acute tocolysis therapy to prolong pregnancy in patients hospitalized with preterm labor at 32 to 34 weeks’ gestation was associated with improved neonatal outcomes\(^6\). At 32 weeks, tocolysis yielded the lowest total number of adverse maternal and neonatal events. At 36 weeks it is probably better not to use tocolysis\(^7\). Generally, tocolytic therapy is not recommended after 34 weeks’ gestation to avoid unnecessary complications, such as noncardiogenic pulmonary edema, as in our patient.

Clinical features and radiographic appearances are generally indistinguishable from other causes of pulmonary edema and adult respiratory distress syndrome. Typical manifestations include dyspnea, chest discomfort, tachypnea, and hypoxemia\(^5,8\). Unlike pulmonary edema due to congestive heart failure, cardiomegaly and pulmonary vascular redistribution are generally absent in cases that are drug-related\(^8\). Fluid overload purportedly occurs in 70% of patients. Rapid clinical improvement (< 24 hours) is normal, although a small percentage of patients may need mechanical ventilatory support. Mortality is low for both the mother and fetus\(^5\). These drugs may induce pulmonary edema in pregnant women, although this effect

Fig. 2 Twelve hours later, a chest radiograph shows resolution of pulmonary edema
has not been observed with treatment of asthma in the nonpregnant state. This condition appears to be a form of noncardiac pulmonary edema, possibly caused by drug-induced fluid retention, superimposed on that normally occurring in the gravid state. This syndrome is unassociated with evidence of myocardial dysfunction and responds readily to diuretics and oxygen\(^9\).

In conclusion, acute pulmonary edema in a previously healthy woman who has recently given birth is an uncommon clinical scenario with some life-threatening complications. No matter what the underlying pathology, prompt appropriate resuscitation is always the first priority.

References

產後發生急性肺水腫：病例報告

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產後發生急性肺水腫是臨床上罕見的疾病。有關潜在性危險的肺栓塞，肺炎，肺水腫等原因也應該要考慮的。我們報告一位45歲健康產婦自然生產後3小時發生急性肺水腫。該病患到院前2天在當地診所接受安胎劑治療。經治療後症狀改善，12小時後進胸部X光顯示肺水腫也消失。她第二天平安出院。

關鍵詞：產後，急性肺水腫，安胎劑