Morbidly Obese Complex Obstetrical Patient with Undiagnosed Peripartum Cardiomyopathy and Development of Flash Pulmonary Edema in PACU

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Introduction

Peripartum cardiomyopathy (PPCM) is a rare disorder of uncertain etiology. Virchow and Porack first recognized the relationship between heart failure and pregnancy in the 1870s when they noted myocardial degeneration in patients who died in the postpartum period. PPCM was first described as a distinctive cardiomyopathy in 1937 by Gouley et al. Since then, much has been learned about this disease process, and better treatment options now exist. Incidence varies greatly worldwide. Reports suggest an incidence of 1 case per 299 live births in Haiti, 1 per 1000 in South Africa, and 1 per 3000-4000 in the United States. Reported mortality rates are between 18% and 56%. A latent form of PPCM has also been described. Here we describe a case of latent PPCM in a morbidly obese patient who developed dramatic flash pulmonary edema in the postanesthesia care unit (PACU).

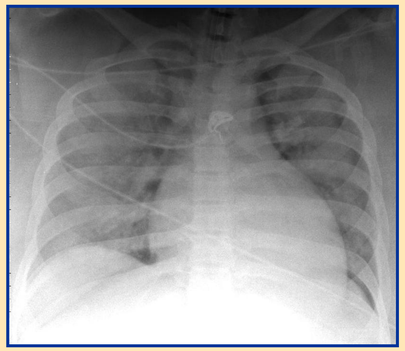
Case Report

A 31-year-old morbidly obese (BMI 53), African American female G3P2002 at 37 weeks' gestation, with a history of insulin-dependent diabetes mellitus, hypertension, hypothyroidism, gastric esophageal reflux disease, chronic focal segmental glomerular sclerosis, hypothyroidism and hyperlipidemia, vaginally delivered twins under epidural analgesia. Patient was stable after delivery. Epidural anesthesia was also used the next day for tubal ligation. Intraoperative course was uneventful.

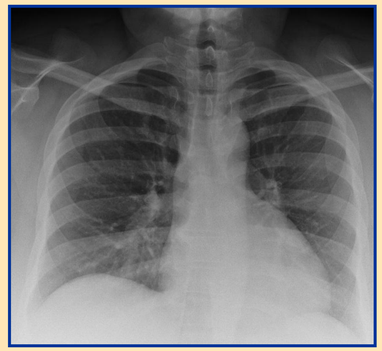
Patient was stable in PACU, but soon developed dyspnea with some wheezing. Albuterol jet nebulizer and 100% non-rebreather oxygen face mask did not help. Patient suddenly progressed into flash pulmonary edema, requiring emergency intubation. Patient was transferred to ICU and placed on a ventilator with 100% FiO_2 and high PEEP. Postintubation chest x-ray revealed pulmonary edema and cardiomegaly; arterial blood gas showed metabolic and respiratory acidosis. Immediate cardiac consultation was obtained. Electrocardiogram showed sinus tachycardia but was otherwise normal. Transthoracic echocardiogram, technically difficult due to patient's body habitus, demonstrated severely decreased left ventricular systolic function. Left ventricular ejection fraction (LVEF) was 20-25% and left ventricular chamber size was moderately dilated with LVIDd 6.28 cm (range, 3.8-5.7 cm).

After excluding other causes for flash pulmonary edema, diagnosis of peripartum cardiomyopathy was made. Patient also developed acute renal insufficiency for a short period but recovered quickly. Patient was aggressively treated in ICU and extubated on Postoperative Day 2. Patient was discharged home in stable condition after 4 days, and was doing well at 3 mo follow-up. Repeat echocardiogram showed moderately reduced LV function with (improved) LVEF 35%. LVIDd improved to 5.52 cm. She reported being asymptomatic and walking a mile without symptoms.

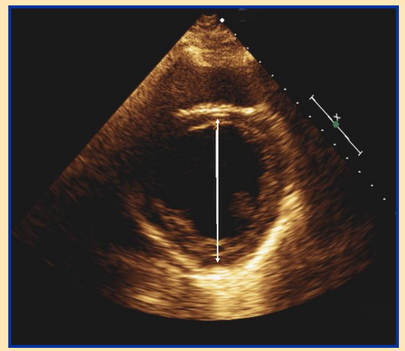
Figures



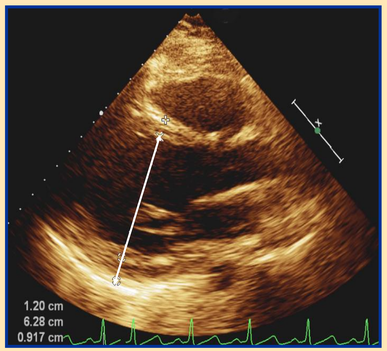
CX-Ray: Pulmonary Edema & Cardiomegaly



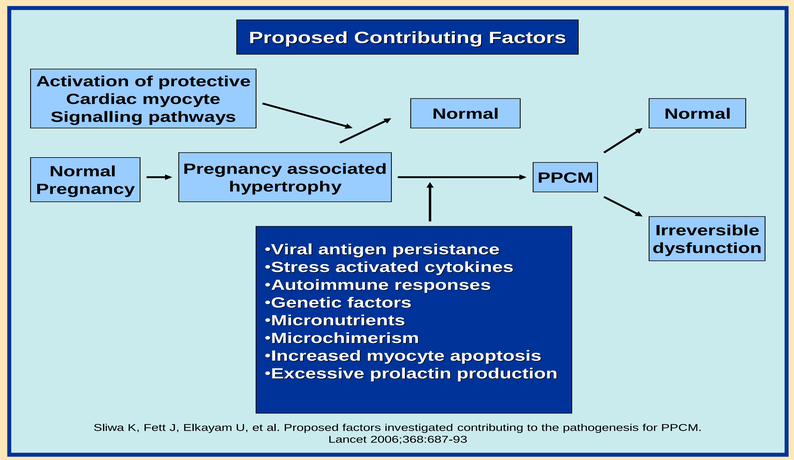
Normal Chest X-Ray on Follow-up



ECHO: Dilated Left Ventricle Chamber



ECHO: Enlarged LVIDd 6.28 cm



Sliwa K, Fett J, Elkayam U, et al. Proposed factors investigated contributing to the pathogenesis for PPCM. Lancet 2006;368:687-93

Discussion

PPCM is a form of dilated cardiomyopathy in which other causes of heart dysfunction are excluded. Risk factors include advanced maternal age (>30 yrs), multiparity, multiple gestation, obesity, nutritional disorder, prolonged tocolysis, preeclampsia, chronic hypertension and African American race.

PPCM is diagnosed by: (1) development of cardiac failure in the last month of pregnancy or within 5 mos of delivery; (2) lack of identifiable cause for cardiac failure; (3) lack of recognizable heart disease prior to the last month of pregnancy; and (4) left ventricular systolic dysfunction (LVSD), demonstrated as a depressed ejection fraction. The causes and pathogenesis of PPCM remain poorly understood. Proposed etiologies include myocarditis, abnormal immune response to pregnancy, viral infections, maladaptive response to the hemodynamic stresses of pregnancy, genetic factors, stress-activated inflammatory cytokines, excessive prolactin production, and autoantibodies against myocardial proteins.

Management goals include preload and afterload reduction and increased contractility. Diuretics, digoxin, beta-blockers and vasodilators (e.g., hydralazine, nitrates) are therapy mainstays. ACE inhibitors and ARBs are used postpartum (teratogenic). Inotropes (e.g., dobutamine, dopamine, milrinone) are considered. Newer treatments with immunosuppressants, immunoglobulin and bromocriptine are promising. Anticoagulation should be considered; serious thromboembolism is associated with PPCM. Prognosis depends on recovery of LV function, usually occurring 6-12 mos after delivery. Counseling is required about risk of heart failure in subsequent pregnancy, occurring in 44% with prior LVSD, but in 21% with normal LV systolic function. Patients failing medical management may be considered for heart transplant. LVAD or IABP are bridges to transplant. Defibrillator is considered for ventricular arrhythmias.

Summary

The diagnosis of PPCM should be considered whenever a patient presents with heart failure during the peripartum period. PPCM is a diagnosis of exclusion, distinguished by rapid onset and occurrence in the peripartum period. Effective treatment reduces mortality and increases full recovery of left ventricular function. Significant improvement is seen in up to 50% of affected women.

References

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