Case Report

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Successful Use of Non-invasive Positive Pressure Ventilation in a Patient with Peripartum Cardiomyopathy: A Case Report

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Abstract

Peripartum cardiomyopathy (PPCM) occurs in approximately 1/3000-1/10,000 deliveries and may result in severe ventricular dysfunction during last month of pregnancy or early puerperium. The clinical presentation and the management of the disease are similar to that of dilated cardiomyopathy due to any other cause. Non-invasive positive pressure ventilation (NPPV) delivers mechanically assisted ventilation to the lungs, without the use of an invasive endotracheal airway. We report a case of a 22-year-old woman, with full-term gestation, in the active stage of labor with PPCM, in decompensated heart failure and pulmonary edema. She was successfully managed with NPPV without any adverse outcome for mother and child.

Key words: Echocardiography, Heart failure, Non-invasive positive pressure ventilation, Peripartum cardiomyopathy

INTRODUCTION

Peripartum cardiomyopathy (PPCM) is an idiopathic cardiomyopathy, characterized by heart failure (HF) and left ventricular systolic dysfunction towards the end of the pregnancy or within 5 months of delivery, when no other cause of HF is identified.[1] The clinical presentation and the management of the disease are similar to that of dilated cardiomyopathy due to any other cause. Along with intensive care management, these patients may also require anesthetic management during vaginal or operative delivery. Non-invasive positive pressure ventilation (NPPV) delivers mechanically assisted ventilation to the lungs, without the use of an invasive endotracheal airway. It reduces the need for invasive mechanical ventilation and the complications associated with it. The length of stay in the intensive care unit and the mortality in selected patients is also reduced.[2]

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CASE REPORT

A 22-year-old primigravida with full-term gestation in the active stage of labor presented with sudden onset of dyspnea, tachypnea, tachycardia and low oxygen saturation. Her heart rate was 120 beats/min; BP was 140/90 mmHg; respiratory rate was 40/min, and SpO_2 was 80% with O_2 @ 6 L/min with face mask. On auscultation, bilateral crepitations were heard all over the lung fields, and normal heart sounds were heard with S3 gallop.

Hematological, renal, liver and clotting parameters are normal. ABG showed pH of 7.38, PCO_2 27 cmH_2O, PO_2 58 cm H_2O, HCO_3^- 14 meq/L, SO_2 82%. Brain natriuretic peptide levels were 890 pg/ml. 2D echocardiography revealed global hypokinesia with left ventricular function (LVEF) 28%, fractional shortening of 20%, LV end-diastolic dimension of 3.2 cm/m^2, normal RA and RV with normal valves and no evidence of pulmonary hypertension.

Patient was started on NPPV in 45° propped up position with P_{insp} 16 cmH_2O, PEEP 6 cm H_2O, FiO_2 100%. Injection furesomide 40 mg intraveuons (IV) was given. Injection dobutamine infusion was started with 3 mcg kg min. Patient improved gradually after increasing P_{insp} to 20 cm H_2O, PEEP to 8 cm H_2O, and

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injection Furosemide 20 mg was repeated. Injection dobutamine infusion was increased to 5 mcg/kg/min. FiO_2 slowly reduced to 60%. Emergency cesarean section was planned meanwhile she delivered by normal vaginal delivery. Baby cried immediately after birth with APGAR score of 7 and 9 at 1 and 5 min respectively.

Patient was shifted to cardiac ICU with NPPV support. Patient improved clinically. So, NPPV support was reduced gradually and removed after 12 h and switched over to non-re-breathing oxygen mask for next 48 h. Injection dobutamine was tapered slowly and stopped. She was started on digoxin, carvedilol, diuretics and LMWH. With this treatment, there was a significant improvement in the LVEF (LVEF – 28% → 38%). Pulmonary edema was regressed. After 1 week, the patient was discharged from hospital for out-patient treatment. Patient was asked to come for follow-up every month for up to 6 months.

DISCUSSION

In this case, PPCM was diagnosed upon exclusion of other causes for the HF, according to the diagnostic criteria of PPCM. The diagnosis is confirmed on the basis of diagnostic criteria:[3] (a) development of the HF during the last month of pregnancy or within 5 months of delivery; (b) absence of an identifiable cause for the HF; (c) absence of recognizable heart disease prior to the last month of pregnancy; (d) left ventricular dysfunction determined during echocardiography with ejection fraction <45%, fractional shortening of <30% on an M-mode echocardiographic scan, or both, and a left ventricular end-diastolic dimension of >2.7 cm/m^2 of body-surface area.[4,5]

There is an increased incidence with multiple gestation, preeclampsia, obesity, advanced maternal age, African descent and prolonged use of tocolytics.[6] These risk factors are not present in our patient. Although historically PPCM risk factors occur in elder women and African women, contemporary trends show that there is an increasing incidence (24-37%) in young primigravid and white patients.[7]

The etiology of PPCM is uncertain; viral,[8] autoimmune, genetic and idiopathic causes have been considered.

The treatment of PPCM is not different from acute and chronic HF. The combination of digoxin, diuretics and sodium restriction, anticoagulation, b-blockers and afterload reduction forms the cornerstone of therapy.[9] Same treatment modalities were given to our patient, to which she responded well. Dobutamine infusion was tapered slowly and was started on Digoxin, in addition to Carvedilol, Diuretics and Warfarin.

Newer drugs like Milrinone, Levosimendan and Bromocriptine, are also being used for PPCM. Levosimendan is a calcium sensitizer that has positive inotropic action and vasodilatation property. Levosimendan is effective in improving the cardiac output and decreasing the mortality.[10]

Mechanical cardiovascular support with an intra-aortic balloon pump or ventricular assist devices may be required if medical therapy is unsuccessful in women with PPCM. Left ventricular assist devices can act as a bridge to recovery or transplantation.[11] Use of short-term extracorporeal membrane oxygenation has also been of benefit in women with PPCM whose HF was refractory to medical therapy and who had persistent pulmonary edema with hypoxemia. Extracorporeal membrane oxygenation can also serve as a bridge to left ventricular assist devices in patients with refractory cardiogenic shock despite use of an intra-aortic balloon pump and full inotropic support.[11] Cardiac transplantation is indicated if supportive treatment fails.

NPPV rapidly improves oxygenation by re-expanding flooded alveoli, increasing functional residual capacity, and thereby more favorably positioning the lung on its compliance curve. This will reduce the work of breathing.[12] Afterload will be decreased by increasing the pericardial pressure and decreasing the trans-mural pressure. This will improve the cardiac performance.[13] There will be an augmentation of stroke volume[14] and reduction in the cardiac sympathetic activity[15] in patients with HF and elevated left ventricular end-diastolic pressure. Increase in the intra-thoracic pressure reduces right and left ventricular end-diastolic volume, thereby decreasing the preload.[16] This favorable effect occurs when filling pressures are high and ventricular performance is poor.

Improved inotropic function of the left ventricle or reduced left ventricular afterload with NPPV is evidenced by increase in stroke volume without a change in pulmonary capillary wedge pressure.

Discontinuation of the therapy is recommended only in case of recovery of the left ventricular function, by monitoring the cardiac function with repeated 2D cardiac ultrasound imaging.[17] Recovery of the left ventricular function usually occurs within 2-6 months after diagnosis.[7] The mortality rate of PPCM is 30-60% and may be caused by severe pulmonary congestion, arrhythmias and thromboembolic events.[18] In future pregnancies there are 50-80% chances of developing cardiac failure, with 60% mortality rate.[19]

CONCLUSION

PPCM is a rare disease of unknown cause. Diagnosis is difficult and requires vigilance. The main objective of

treatment is to reduce the symptoms of congestive HF. High degree of clinical suspicion supported by the early echocardiography is important to diagnose this entity that can have a poor outcome despite optimal medical management. NPPV may offer an adjunct to medical therapy in improving left ventricular function and augmenting cardiac performance in a subset of patients with PPCM.

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