CASE REPORTS

PERI-OPERATIVE PULMONARY EDEMA A MANIFESTATION OF PERIPARTUM CARDIOMYOPATHY

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INTRODUCTION

Pulmonary edema encountered due to peripartum cardiomyopathy is a less common form of edema in perioperative settings. Peripartum cardiomyopathy is a rare form of cardiomyopathy, of unknown etiology, which is associated with a significant morbidity and mortality.

Age more than 30 years, lower socio-economic status with poor nutritional status, smoking, tobacco eating, toxemia of pregnancy, long term tocolysis, twin pregnancies, poor ejection fraction and increased left ventricular end-diastolic dimension are poor prognostic factors [1,2].

Pulmonary edema leads to such an insufficient gas exchange that both the oxygenation of blood and elimination of CO_2 from the body can become inadequate. In addition to other measures, mechanical ventilation is considered if the supplemental O_2 mask fails to maintain O_2 tension up to 60 mm Hg. Institution of intermittent positive pressure ventilation (IPPV) and positive end expiratory pressure (PEEP) causes preferential shift of edema fluid in the lungs and thus improve oxygenation.

CASE REPORT

A 20 years primigravida of 70 kg, non-smoker and normotensive, presented in emergency with history of pulmonary edema after caesarean hysterectomy under spinal anesthesia, complicated by cardiac arrest on 5th July 2005. She was revived and transferred to intensive care unit of POF hospital on assisted ambu ventilation after tracheal intubation. She had a history of profuse bleeding due to uterine atony during surgery and received six units of whole blood transfusion. On examination, the patient was conscious, anxious and restless, pale, cyanosed, had edema face and feet, distended neck veins and was orthopnoeic. She was having laborious breathing with respiratory rate 42/min, blood pressure 135/80 mmHg, pulse rate 84/min and oxygen saturation of blood less than 70%. Blood stained froth was visible through the endo tracheal tube. Her chest was full of crackles all over.

X-ray chest showed diffuse homogenous opacities in both lung fields. A provisional diagnosis of over transfusion pulmonary edema was made.

She was placed on ventilatory support on synchronized intermittent mandatory ventilation (SIMV) along with positive end expiratory pressure (PEEP).

Her endotracheal suction had to be performed quite frequently due to exuberant amount of edema fluid. She was given diuretics, antibiotics and analgesics. Monitoring during this period included pulse oximetry, ECG, non-invasive blood pressure, end tidal CO_2, and central venous pressure. Her blood picture, LFTs, Renal functions, coagulation profile was within normal limits. ECG showed sinus tachycardia and serum troponin-T was negative.

Next morning she was fully conscious, oxygen saturation was > 94% on air and the chest clear on auscultation and was weaned off the ventilator. The administration of intravenous fluids was kept guarded.

Next day, she once again became orthopnoeic. She was found to have elevated

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central venous pressure; S3 gallop rhythm and crackles in both the lungs. Her oxygen saturation fell down to 70%. Oxygen was given by mask and injection furosemide 100 mg repeated twice over a period of one hour. Her condition improved again.

Echocardiography was now performed, which showed global hypokinesia, valves were normal with increased left ventricular end diastolic diameter (LVEDD) 57.5 mm, an ejection fraction (EF) of 35%, and no pericardial effusion or left ventricular clots. A diagnosis of peripartum cardiomyopathy was made. Patient was digitalized and subsequently maintained on 0.25 mg digoxin once daily, captopril 6.25 mg 8 hourly, Low molecular weight heparin 40 mg 12 hourly and spironolactone 100 mg daily.

She was discharged on 10th day on captopril 12.5 mg 8 hourly, furosemide 20 mg daily, spiranolacton 50 mg once daily, digoxin 0.25 mg once daily, metoprolol 50 mg once daily, clopidogrel 75 mg once daily and is being followed in out patient department.

DISCUSSION

Perioperative pulmonary edema in obstetric patient can result from a hemodynamic cause (permeability edema), prolonged severe airway obstruction (post obstructive pulmonary edema) or severe pulmonary hypertension (neurogenic pulmonary edema) [3].

The management of pulmonary edema and consequent respiratory failure is directed to support pulmonary functions until the lungs recover from the insult. The therapeutic measures include administration of supplemental oxygen, diuretics, tracheal intubation, mechanical ventilation, positive end expiratory pressure (PEEP), facilitation of removal of secretions from the airways and control of infection [4].

Peripartum cardiomyopathy is a known cause of congestive cardiac failure (CCF) in the last trimester of pregnancy or within six months after parturition. Signs and symptoms of CCF can commonly be mistaken for normal manifestations of pregnancy or associated cardiac lesions like mitral stenosis, mitral insufficiency, aortic insufficiency, left atrial myxoma, pericardial disease and congenital heart disease [5].

It is essentially a diagnosis of exclusion, which can only be made in the absence of other demonstrable cause and is confirmed by echocardiography. The investigations like chest radiograph and ECG are non-specific [6].

In 50% of the patients cardiac functions recover within six months of parturition. The other fifty percent show variable clinical deterioration with thirty-five percent mortality in 5 years. With the impaired cardiac function, subsequent pregnancy is associated with recurrence of peripartum cardiomyopathy in 50% of patients and possesses 50% to 100% mortality [7].

For most of the cardiac diseases, no single anesthetic technique is exclusively indicated or contra indicated. Epidural or subarachnoid blocks have the edge over general anesthesia so that early recognition of symptoms is possible. The primary concern is to minimize increase in cardiac output that ordinarily occurs during parturition. This is best accomplished by continuous lumber epidural anesthesia [5].

It is concluded that peripartum cardiomypathy has high morbidity and mortality therefore subsequent pregnancy should be avoided. The management may be aimed at decreasing pulmonary capillary pressure and maintaining adequate oxygenation alongwith hemodynamic, inotropic and ventilatory support.

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