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A case of Peripartum Cardiomyopathy in Preeclamptic Toxemia

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INTRODUCTION

Peripartum cardiomyopathy is a erratic type of increased cardiomyopathy presenting with heart failure subordinate to left ventricular systolic dysfunction to the end of pregnancy or in the puerperium. The American heart association & European society of cardiology has defined this condition as considered it as a diagnosis of exclusion. Echocardiographic findings include ejection fraction(EF) of nearly below 45% and/or Fractional shortening of less than 30% or End-diastolic dimension of more than 2.7 cm/m2. Incidence of PPCM is around 1 in 2000 with a fatality rate of 20-50%. Known risk factors are advanced maternal age, gestational hypertension, preeclampsia, multiparity, multiple gestations, African American race, obesity, malnutrition , diabetes, substance and tobacco abuse and family history.

Preeclamptic toxemia or severe preeclampsia is a condition of severe hypertension(BP ≥ 160/100mmHg) in pregnancy associated with multiple organ dysfunction like impaired coagulation, impaired hepatic function, impaired renal function or pulmonary edema. Although the mechanism is not known, but various studies and case reports have shown a higher incidence of PPCM in preeclamsia, incidence ranging from 2-68%. Etiology of preeclampsia and its link with cardiovascular diseases are poorly understood, but there might be some common underlying mechanisms involved in the pathophysiology of preeclampsia and pregnancy-related cardiac problem. PPCM with severe preeclampsia is a big clinical challenge in terms of both diagnosis and management and must have combined efforts of cardiologist, obstetrician, intensivist, anesthesiologist, and neonatologist.

CASE REPORT:

A 34year old, post IUI (intrauterine insemination) pregnant patient, with past history of a 1st trimester abortion, was referred to our centre from a peripheral hospital at around 32weeks with complaints of dyspnoea for 1week and uncontrolled hypertension. She was a known case of gestational hypertension for last 3weeks and was on amlodipine 5mg tablet. She was also on thyroxine 75mcg for last 10years. On admission she had generalized edema, pallor, tachycardia(Pulse rate 100bpm), respiratory rate of 18/min, blood pressure(BP) of 170/120mmHg. Fine crepitations were present on chest auscultation with SpO2 95%. Fundal height of uterus was 32weeks and fetal heart rate(FHR) was regular and reactive. Patient kept in propped up position and oxygen inhalation was started. In consultation with cardiologist, nitroglycerine infusion, furosemide injection & ipratropium nebulisation were started. Also, Inj.MgSO4, loading dose was given to prevent eclampsia and Inj.Betnesol were given for fetal lung maturity.

Investigations revealed multiorgan involvement with high serum creatinine(3.11mg/dl), hyperkalemia(5.9mmol/L), very high liver enzymes(SGOT-419, SGPT-544 U/L) with 2+ urine albumin and serum uric acid of 12mg/dl. Ultrasonography showed hepatomegaly and bilateral renal cortical hyperechogenecity. Gastroenterology consultation was taken. Patient was managed in intensive care unit(ICU) due to uncontrolled BP and deranged parameters. Labetalol infusion was started and hyperkalemia corrected with insulin-dextrose infusion, calcium gluconate injection and salbutamol nebulisation. Her dyspnoea gradually improved and on 3rd day of admission her BP was under control. Antihypertensive infusions were stopped and oral nifedipine and labetalol were started. But creatinine and liver enzymes were continuously rising(day 5 creatinine- 4.6mg/dl, SGOT-430. SGPT-686 U/L).

Echocardiography showed moderate LV systolic dysfunction, EF of around 35% with moderate to severe mitral regurgitation, thus suggesting peripartum cardiomyopathy. Fluids were restricted to 1.5litres/day. CPAP be presented from 4th day of admission. In the ICU, fetal monitoring was continued using frequent intermittent FHR auscultation and alternate day Doppler study.

On day 10, patient went into labour and she developed acute LVF(left ventricular failure) with acute respiratory distress and anuria. Emergency LSCS was done in general anaesthesia with cardiac monitoring. A 1.9kg IUGR female baby was delivered. Patient kept under mechanical ventilation postoperatively.

2 hours after operation, she developed cardiogenic shock and thus ionotropes(dopamine & noradrenaline) were started. Hemodalysis was done due to oliguria and acidosis. BP became 180/120mmhg on 2nd postoperative day and nitroglycerine infusion was started. An episode of PPH occurred, which was managed with oxytocics, packed cells(PRBC) & FFP transfusions. She was extubated on 2nd day; urine output, saturation and electrolyte levels were improving.

From 4th postoperative day, BP was gradually controlled, liver enzymes started declining and plateau creatinine levels(on 5th post op day, creatinine- 4.7mg/dl, SGOT-126, SGPT- 382 U/L).

She was discharged on 10th day of LSCS with amlodipine, furosemide and ursodeoxycholic acid with advise of frequent follow ups.

DISCUSSION:

Bello N et al found 4 times increased risk of PPCM in preeclampsia patients. Demakis and Rahimtoola , reported that preeclamptic toxemia was detected in 22% of women with PPCM. Kai H et al[8] mentioned

that hypertension might increase the severity of PPCM. Diagnosis of PPCM is a challenge for various reasons. Early symptoms of PPCM like dyspnea, fatigue, and edema which are seen in later half of pregnancy may be confused with normal pregnancy symptoms. Late symptoms like paroxysmal nocturnal dyspnea, pulmonary edema, and distended neck veins are similar to those of congestive heart failure. Additionally, preeclampsia may manifest with edema, oliguria and symptoms of respiratory distress due to pulmonary edema. In our case also we did not suspect PPCM on the 1st day. However echocardiography findings with no other etiology brought us to the diagnosis.

Treatment of PPCM include relieving symptoms, stabilizing hemodynamics and treating precipitating factors like preeclampsia. Treatment is similar to acute heart failure like diuretics, afterload reduction and control of hypertension. In our case we used nitroglycerine infusion to control hypertension and to reduce afterload. Labetalol, which was administered to this patient, is commonly used to treat hypertension; however, it may worsen cardiac function and contribute to pulmonary edema, and thus was used cautiously. Loop diuretic(furosemide) was also used. Fetal surveillance, which was done in our case, is extremely important in these cases as afterload reduction may deteriorate FHR. Sometimes anticoagulants are given postpartum to reduce incidence of thrombosis.

When patient gets symptomatic relief with least circulatory support drugs, she is considered to have recovery. In PPCM patients, recovery of ventricular dysfunction has been defined as an LVEF greater than or equal to 50%ormore than 20%improvement, and left ventricular fractional shortening greater than or equal to 30% . Recovery usually takes up to 2 months. However, it can take 6–12 months to recover completely.

Our patient developed acute kidney injury(AKI) due to severe preeclampsia. AKI in pregnancy is an important clinical challenge with significant morbidity and mortality. Studies evaluating pregnancy related AKI suggest an overall incidence of AKI in preeclampsia of 1.5– 2% with maternal mortality rates of 0 –10%, and perinatal mortality rates of 34–41% and short-term dialysis rates of 10–50%. Preeclampsia causes AKI either due to glomeruloendotheliosis or secondary effects like relative intravascular volume depletion, vasoconstriction, and activation of the inflammatory and coagulation cascades. AKI management is primarily supportive and include replacement of blood products, maintenance of intravascular volume, and renal replacement therapy as needed. Maintenance of volume and electrolyte is the key component of the treatment. In our case, patient had hyperkalemia which was managed with appropriate medications. But after operation patient developed oliguria and acidosis for which hemodialysis was required.

However delivery of fetus is the only effective treatment in preeclamptic toxemia with multiorgan involvement. Prognosis of fetus in such cases is usually worse, though in our case the baby survived well. Presence of PPCM does not mean that delivery has to be done immediately. However a patient with complications as in our case necessitates preterm delivery to save both mother and baby. However our patient went into spontaneous labour, so, decision of pregnancy termination was easy.

The patient was also a known case of hypothyroidism, controlled on medication. Hypothyroidism also predisposes to heart failure which causes a hypodynamic cardiovascular state associated with reduced left ventricular systolic and diastolic function.

CONCLUSION:

PPCM is difficult to diagnose specially in cases with preeclampsia. intervention, Early diagnosis, and treatment may prevent or, at least, lessen symptoms of PPCM and improve fetaland maternal outcomes. Utmost caution is needed in treating such cases with severe manifestations. All peripartum women with suspicious cardiac symptoms and risk factors like preeclampsia should undergo a cardiac examination to rule out PPCM.