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CASE REPORT

Peripartum cardiomyopathy co-existing with severe preeclampsia complicated by AKI

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ABSTRACT

Peripartum cardiomyopathy is a rare idiopathic cardiac disease occurring in pregnancy and puerperium. Studies have shown that women with preeclampsia have a higher risk of developing peripartum cardiomyopathy. In this report we present a case of a 3rd trimester pregnancy with severe preeclampsia with acute kidney injury who was diagnosed with peripartum cardiomyopathy. Supportive management was done with a multidisciplinary approach. Caesarean section was done, an IUGR but healthy baby was delivered and the patient recovered gradually.

Keywords: Cardiomyopathy, pregnancy, idiopathic, preeclampsia, acute kidney injury, multidisciplinary.

Peripartum cardiomyopathy (PPCM) is a type of dilated cardiomyopathy presenting with heart failure secondary to left ventricular (LV) systolic dysfunction towards the end of pregnancy or in the puerperium period (usually within 5 months), where no other cause of heart failure is found (American Heart Association, European Society of Cardiology). Echocardiographic findings include ejection fraction (EF) of below 45% and/or fractional shortening of less than 30% or end-diastolic dimension of more than 2.7 cm/m2 with or without LV dilatation. The overall incidence of PPCM is around 1 in 2000 [1] with mortality of more than 50%. Common risk factors for PPCM are advanced maternal age, gestational hypertension, preeclampsia, multiparity, multiple gestations, obesity, diabetes, substance and tobacco abuse and family history[2]. Complications include thromboembolism, arrhythmias and myocardial infarction.

Preeclampsia is a multiorgan disease, which is considered to be severe when BP is greater than 160/110 mm Hg, proteinuria is greater than 5 gm or signs and symptoms are consistent with end organ damage such as headache, visual disturbances, impaired coagulation, impaired hepatic function, impaired renal function or pulmonary edema. It has been observed that PPCM is seen more in parturients with preeclampsia, incidence ranging 2-68% [3, 4], but the reason is not known.

Acute kidney injury (AKI) in pregnancy is a clinical challenge with significant morbidity and mortality. Studies suggest an overall incidence of AKI in

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preeclampsia is 1.5– 2%[5] with maternal mortality rates of 0 –10%, and perinatal mortality rates of 34–41% and short-term dialysis rates of 10–50%. Managing PPCM with severe preeclampsia is very challenging and needs combined effort of cardiologist, obstetrician, intensivist, anesthesiologist, and neonatologist.

Case report

A 34 year old nullipara with history of 1 spontaneous abortion was referred from a peripheral centre at 35 weeks with post IUI pregnancy with breathing difficulty for 1week and uncontrolled BP. She was diagnosed with PIH 3weeks back and was on tablet amlodipine 5mg. She has been on thyroxine 75 mcg for last 10 years due to hypothyroidism. On admission, edema was present, with pulse rate of 100bpm, respiratory rate of 18/min and BP of 170/120mmHg. On obstetric examination fundal height was 32weeks and FHR was regular and reactive. On chest auscultation occasional fine crepitations were present with SpO_2 of 95%. As advised by Cardiologist, she was started with nitroglycerine infusion, furosemide injection, ipratropium nebulisation along with tablet amlodipine. Loading dose of Inj.$MgSO_4$ was given and Inj. Betnesol 12gm IM given for fetal lung maturity. O_2 inhalation started and patient was kept in propped up position.

Investigations revealed that her creatinine was 3.11mg/dl with hyperkalemia and elevated liver enzymes (Table I). Gastroenterology consultation was taken. Patient was then shifted to ICU due to uncontrolled BP and multiorgan involvement. Labetalol infusion was started and correction of hyperkalemia was done with insulin-dextrose infusion, calcium gluconate injection and salbutamol nebulisation. Her respiratory difficulty was

Table 1: Investigations of the patient

Days	**Serum creatinine mg/dl**	**Blood urea in mg/dl**	**Serum Na+ in mmol/L**	**Serum K+ in mmol/L**	**SGOT (U/L)**	**SGPT (U/L)**	**Hb in gm/dl**	**Others**
Day 1	2.98	65	145	5.2	419	544	9.9	ALKP- 241U/L PT-10.4 sec Serum bilirubin - 0.71mg/dl Platelet - 2.15lacs/mm3 Urine albumin- 2+
Day 2	3.11			5.9	420	674		USG - hepatomegaly, bilateral raised renal cortical echoes
Day 3				6.1	476	822		
Day 4	3.45	106		5.9	398	774	10.2	Serum uric acid- 12mg/dl
Day 5	3.35	106	142	4.6	430	686		PT- 10.7 sec
From day 6 till onset of labour parameters were almost plateau								
On day of operation	3.74	127	141	5	260	567	8.8	Serum albumin- 1.9gm/dl BT,CT.PT were normal
Patient had oliguria and acute LVF prior to operation and developed cardiogenic shock and acidosis after operation. Hemodialysis was done								
Post op day 2	4.04	131	141	6.7	574	578	8	
PPH occurred. Oxytocin infusion given.								
Post op day 3	5.83	162	149	4.9	293	414	6.7 (PRBC & FFP given)	PT-11.2sec
Post op day 5	5.3	180	147	4.7	126	382	8	
Serum creatinine and liver enzymes were gradually declining.								
Before discharge	4.6	121	142	3.8	32	146		Urine albumin- trace

relieved to some extent and on 3rd admission day her BP was under control and antihypertensive infusions were stopped. Orally nifedipine and labetalol were continued. But her creatinine and liver enzymes were elevated further, with slight fall in potassium level. Echocardiography showed moderate LV systolic dysfunction, EF of around 35% with moderate to severe MR, thus suggesting PPCM. Fluids were restricted to 1.5 litres/day. CPAP was started from 4th day of admission. Creatinine levels were gradually declining.

During her stay fetal monitoring was done using frequent FHR auscultation and alternate day doppler study. On 10th day of admission, she went into labour. She developed acute respiratory distress with anuria and was diagnosed with acute LVF. Emergency LSCS was done under general anaesthesia with cardiac monitoring. A 1.9kg IUGR female baby was delivered. Patient was kept under mechanical ventilation postoperatively.

Patient went into cardiogenic shock after 2 hours of operation for which PRBC transfusion and ionotropes(dopamine & noradrenaline) were started. Hemodialysis was done 12 hours after delivery as the patient was oliguric and developed acidosis. BP again went up to 180/120mmHg on 2nd postoperative day and nitroglycerine infusion was started. She had an episode of PPH on the 2nd day which was managed with oxytocics, PRBC & FFP transfusions. Urine output and acidosis were improving and potassium level was corrected but creatinine level increased. She was extubated on 2nd postoperative day. Labetalol, inapure and amlodipine were started. Nebulisation and diuretics were continued.

Patient was improving clinically with gradual control of BP and maintaining O_2 saturation. Liver enzymes levels were also declining. Gradually from 4th postoperative day creatinine levels started falling. Urinary albumin was trace. Baby was discharged from NICU and she was discharged on 10th postoperative day with amlodipine, furosemide and ursodeoxycholic acid and followed up by cardiologist, gastroenterologist and nephrologist in OPD.

Discussion

Bello N et al [3] found 4 times increased risk of PPCM in preeclamptic patients. Also, hypertension might increase the severity of PPCM [6]. Early symptoms of PPCM such as fatigue, dyspnea, and edema which are seen in later half of pregnancy may be confused with normal pregnancy manifestations. Later symptoms are identical to those of congestive heart failure [2]. Additionally, preeclampsia may manifest with symptoms of respiratory distress due to pulmonary edema [7]. Our case also presented with respiratory distress with uncontrolled BP.

Treatment of PPCM includes stabilising hemodynamics, relief of symptoms, and treatment of precipitating factors [2]. Judicious use of diuretics to relieve pulmonary edema, afterload reduction to decrease the work load of the heart, and control of hypertension [2] are necessary. In our case we used nitroglycerine infusion mostly 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 was also used. Afterload reduction has the risk of FHR deterioration. Strict fetal monitoring is thus recommended. Clinical recovery is considered with symptomatic relief and when all circulatory support drugs have been tapered. Recovery usually takes up to 2 months. However, it can take 6–12 months for complete recovery [8].

In preeclampsia 70% patients develop the pathognomic renal lesion i.e. glomeruloendotheliosis. Also, there is an overall decrease in GFR and effective renal plasma flow in preeclampsia by 32% and 24% respectively as compared with normal pregnancy. Additionally, preeclampsia can also predispose to AKI through secondary effects of relative intravascular volume depletion, vasoconstriction, and activation of the inflammatory and coagulation cascades. In preeclampsia related ARF, the primary pathologic process is acute tubular necrosis, with the most severe cases developing renal cortical necrosis[9].

In the case of preeclampsia related AKI, delivery is indicated, as termination of pregnancy is the only effective treatment of preeclampsia. If the conceptus is mature, it should be delivered as soon as the mother's condition has been stabilized. Prognosis of the fetus is usually worse than that of the mother. Mode of delivery depends on various clinical factors. AKI management is primarily supportive and includes maintenance of

intravascular volume and electrolyte balance and renal replacement therapy as needed. 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.

Our 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[10].

Conclusion

A high index of suspicion is required to diagnose PPCM, specially in cases of preeclampsia as it is difficult to diagnose whether the patient with dyspnea or edema has heart failure or not. Early diagnosis, intervention and treatment may prevent or lessen symptoms of PPCM and improve maternal and fetal outcomes. A cardiac evaluation is very much necessary even in slightest suspicion in these cases.

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