Medical Science

pISSN 2321–7359; eISSN 2321–7367

A near miss case of PPCM a diagnostic dilemma and an intriguing challenge - a case report

Mahak Choudhary[1], **Kamlesh Chaudhari**[2], **Neeta Verma**[3], **Sanket Bakshi**[4*]

To Cite:
Choudhary M, Chaudhari K, Verma N, Bakshi S. A near miss case of PPCM a diagnostic dilemma and an intriguing challenge a case report. Medical Science 2022; 26:ms450e2101.
doi: https://doi.org/10.54905/disssi/v26i129/ms450e2101

Authors' Affiliation:
[1]Medical Student, Jawaharlal Nehru Medical College, Datta Meghe Institute of Medical Sciences (Deemed to be University), Sawangi (Meghe), Wardha – 442001, Maharashtra, India
[2]Professor, Department of Obstetrics and Gynaecology, Datta Meghe institute of Medical Sciences (Deemed to be University), Sawangi (Meghe), Wardha – 442001, Maharashtra, India
[3]Associate Professor, Department of Anaesthesiology, Datta Meghe institute of Medical Sciences (Deemed to be University), Sawangi (Meghe), Wardha – 442001, Maharashtra, India
[4]MBBS Intern, Jawaharlal Nehru Medical College, Datta Meghe institute of Medical Sciences (Deemed to be University), Sawangi (Meghe), Wardha – 442001, Maharashtra, India

[*]**Corresponding author**
MBBS Intern, Jawaharlal Nehru Medical College, Datta Meghe institute of Medical Sciences (Deemed to be University), Sawangi (Meghe), Wardha – 442001,
Maharashtra, India
Email: sanketbakshi25@gmail.com

Peer-Review History
Received: 12 February 2022
Reviewed & Revised: 14/February/2022 to 26/October/2022
Accepted: 01 November 2022
Published: 05 November 2022

Peer-review Method
External peer-review was done through double-blind method.

URL: https://www.discoveryjournals.org/medicalscience

ABSTRACT

The period of pregnancy is quite vulnerable for the female, taking into consideration her health and the changing physiological conditions in her body, the adaptations that are made to adjust to the pregnant condition, sometimes may put a burden over her body. Amongst the morbid conditions, cardiovascular abnormalities have a higher rate and chances to have a dreadful impact on the pregnant situation. Due to the increased load and change in the body physiology. Postpartum Cardiomyopathy (PPCM) is the foremost cause of an unsuccessful pregnancy. Because of the degrading impacton the cardio vascular system of the mother, postpartum cardiomyopathy stands to be one of the leading causes of morbidity during the pregnancy period.

Keywords: Peripartum Cardiomyopathy, Heart Failure ejection fraction, Echocardiography.

1. INTRODUCTION

Consequent change in the physiology of the female during her pregnancy, exposes her to an arena of various disorders, leading in them is cardiovascular pathologies. Nearly 5 % of the diseases do not lead to fatality and the outcome is positive, the average occurrence of the feto mortality rate is seen risen in the cases of Cardiomyopathy associated with pregnancy (Karafiatova et al., 2017; Wang et al., 2009). Although the disease of peripartum cardiomyopathy proves to be of danger associated with high mortality rates and mis diagnosed cases, the clear knowledge regarding the aetiology, pathogenesis, treatment medications and further outcomes have not been yet researched in depth. The patterns of peripartum cardiomyopathies are generally seen associated with the other cardiac events as well. Leading among these events are the cardiac vascular insufficiencies, due to the vascular disturbances caused, the effective Blood flow to the growing foetus could be compromised sometimes in severe condition leading to the feto maternal mortalities. Idiopathic heart failure is also often seen associated with the progression of the pathology of PPCM (Libby et al., 2007); Without any pre disposing cardiac abnormalities, Acute

heart failure is presented in the last 5 weeks of the third trimester (Demakis et al., 1971); according to the surveys the pathologies of the Left ventricular dysfunction have a basis of the idiopathic cardiomyopathies (Pearson et al., 2000).

The condition of Cardiomyopathy in the peripartum period is associated with the conditions of left ventricular abnormalities and the heart failure leading further to the hemodynamic changes hindering the growth of the foetus and in severe conditions leading to the feto maternal fatalities. The female who become pregnant for the very first time without having any known history of cardiac abnormalities, they have a comparatively higher incidence rate of this condition (Marx et al., 2006). Modern day medicine and the constant evolution in the medical science have made the prognosis of this condition much better. But in certain conditions death is seen post surgically in the presentation of myocardial infarction, and other cardiac events (Abboud et al., 2007; Simon et al., 1990). Medical definition of peripartum cardiomyopathy strictly states the presence of signs and symptoms in partum without any previous presence of cardiac abnormalities. Left ventricular dysfunction and the associated electro cardiogram signs are necessary to make the diagnosis of this pathology. Taking into consideration the other cardiac investigations, ejection fraction being less than 45% and the end diastolic dimension index of more than 2.7 cm/m2 is also being noted (Sliwa et al., 2006).

2. CASE REPORT

A 24 year old presented to the casualty of Acharya Vinoba Bhave Rural Hospital in July, on day 9 post partum, with an unremitting fever and was responsive only to deep stimuli. She was referred from rural hospital in the periphery with high grade temperature, not relieved on basic first line management of antipyretics and antibiotics, where she had undergone her primary caesarean section 9 days back. She had no antenatal or immediate postnatal complications. Her medical history was unremarkable, there was no history of exposure to any cardiotoxic drugs or any intake or exposure to infectious agent, apart from a history of low appetite since childhood resulting in an undernourished poor built. During her general examination in our hospital, the patient was noted to be febrile [101 F] and her blood pressure of 80/60 mmHg, a pulse rate of 150/beats per minute, a respiratory rate of 28/cycles per minute, and an oxygen saturation of 99% on room air. Her breast examination revealed the occurrence of engorged breast, an immediate manual expression of milk with cold fermentation was given. Her Cardiovascular and Respiratory System examination findings were normal. Her abdominal examination had uterus well contracted and on per vaginal examination there was no fresh bleeding or foul-smelling lochia. Her basic investigations were sent, and she was sent to Medicine ICU for further management. A central line was inserted in situ.

Her investigation on the same day were, Hb of 8.1 gm/dl, WBC count of 8700/ cmm3, CRP count raised, platelet count of 2.12 lakhs, PT came to be 11.9 sec, APTT was 29.5 and ESR was 65 and the urine examination came to be normal. Her ABG analysis came back normal. Her reports for Malaria, Dengue, Scrub typhus, Leptospira were negative. Her ECG showed sinus tachycardia and nonspecific ST-T wave changes, her bedside USG revealed an involuting uterus with minimal endometrial collection. Xray chest and Xray abdomen were within normal limits. A blood transfusion was done following low Haemoglobin [8.1gm/dl]. Her blood culture reports revealed a growth of Acinetobacter species and vaginal swab of E. coli. She was then started with higher antibiotics but continued having fever for 2 more days.

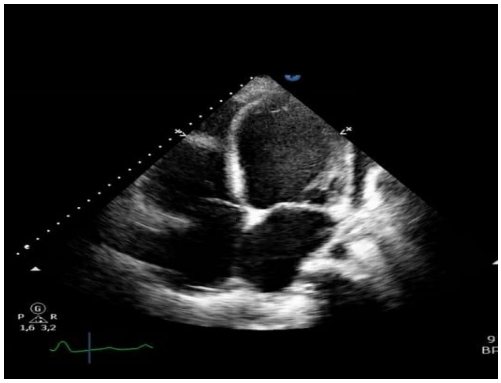

Figure 1a Ultra Sonography of the case

Taking in to consideration her septicaemia may be originating from her engorged breast, and therefore breast feeding was stopped with tablet cabergoline, as she was admitted in ICU, but continued having high grade fever for 2 more days. A blood transfusion was given. A repeat complete blood count was done after 24 hours of management and the values came out to WBC count was 24000/cmm3 confirming the septicaemic theory. Ultra-sonography revealed no relevant findings (Fig 1a). On the third

day of her admission (day 11 of her Caesarean section), she complained of severe chest pain and breathlessness later in the night. An urgent ECG revealed a sinus bradycardia (56 beats per minute). 2D echo was done which revealed a diagnosis of Global Left Ventricle Hypokinesia with Ejection Fraction - 40% suggestive of either Myocarditis or Peripartum Cardiomyopathy (Fig 1b). Her CPKMB and Troponin levels came within normal limits.

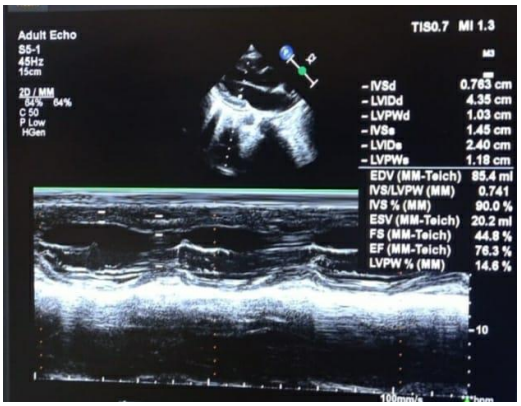

Figure 1b 2D echo findings of the case

A cardiac consultation was done for the management of heart failure and was advised a tablet Larilactone which alleviated her symptoms and injection atropine SOS in case of heart rate less than 60 beats per minute. Later she was monitored during hospitalization, where in during one instance her heart rate dropped to 54 bpm, for which she was given injection atropine. Her potassium level was 2.5mmol/litre and corrections were given for the same. Her repeat 2D showed an improvement in her left ventricular function, where her ejection fraction increased from 40 to 55%. Patient during her stay also had an outbreak of or labial herpes, which was managed conservatively. During the later days of her admission, patient gradually progressed and showed symptomatic improvement and was finally discharged after 10 days, with 2D echo showing LVEF 0f 50%. She was asked to follow - up within a week and regular follow up in cardiology OPD. She was also explained about the importance of 2D echo scan, 6 months later to check for any residual heart failure. On follow-up after a week, she was comfortable with HR – 80/bpm, CVS – normal, BP – 120/80 mm/hg, ECG was also normal.

3. DISCUSSION

Peri partum cardiomyopathy is acardiovascular pathology related to the occurrence of cardiac events in pre partum or post partum period. Particularly pregnancy associated cardiac disorders have a comparatively blood prognosis, leading to less fatality, but if the disease of per partum cardiomyopathy progress to the severe condition, the fatality rates are high including the feto maternal deaths. The diagnosis of this condition is made in the pregnant women having no previous history of any cardiac abnormalities. The findings on the electrocardiogram are a key presentation to make the diagnosis. Along with the ECG findings, the reduction the ejection fraction and the left ventricular systolic dysfunction are the characteristics of this pathology. Certain guidelines have been put forward to make the management of this condition easy. Rather than a surgical way out, medical management is given a preference. The medical treatment options include the modalities of bromocriptine therapy in combination with the anti coagulants which ultimately leads to the inhibition of the prolactin from the higher centres. From the many studies conducted, prolactin has been found to be an efficient trigger for the causation of this disease. Also with this the administration of low molecular weight heparin in the pre partum period and warfarin in the post partum period are a better option to reduce the severity of this disease. It is a must to avoid the usage of warfarin in the ante partum period, looking to the teratogenic effects it causes.

To further narrow down the diagnosis of this condition, the American Heart Association has given some clinical guidelines which state that onset of symptoms of heart failure pre partum or a few days post partum, alongside the presence of left ventricular dysfunction would indicate the presence of peripartum cardiomyopathies (Cemin et al., 2009). The medical management must be advised by keeping the safety of the growing foetus at the centre point. The class of drugs that could be generally prescribed during the pregnancy is diuretics, hydralazine and beta blockers. ACE inhibitors and the angiotensin receptor blockers are contraindicated to use during the pregnancy, but after the term the newer drugs in these groups could be prescribed to enhance the cardiac efficiency, such as enalapril and spironolactone. The American Heart Association suggest the use of advanced drugs such as Low Molecular Weight Heparin if the ejection fraction goes further below 30% apart from the low molecular weight heparins (Ansari et al., 2002) the drug warfarin is strictly avoided during the pregnancy due to the teratogenic effects it causes. As far as the advanced

therapeutic measures are concerned, the role of bromocriptine is currently experimental, but as it is pro thrombotic agent, its combination with certain anti coagulation agents is currently being considered. If the condition of the patient ids stable, it is advised that the delivery to be done vaginally, unless there is any obstetric indication to perform an LSCS. Post partum care is also needed to rule out the decompensation as lactation has been advised to be avoided in the condition if the patient further deteriorates. After a thorough literature search, it is evident that the current therapeutical advancements medical management of PPCM has been further classified in to following groups (Fett et al., 2002).

As the trails have concluded, after the effective therapy a bed rest of 12 months approximately proves to be helpful in reducing the incidence of cardiomegaly, but as per the newer guidelines this prolonged bed rest is not necessary, on the other hand it pre disposes the patient to the presentation of deep venous thrombosis, which further leads to pulmonary embolism. Pregnancy, which is one of the most delicate circumstances for women and the fetus she is carrying, puts physiological stress on her body, can occasionally cause the unforeseen occurrence of certain systemic problems, further complicating the pregnancy process. Major population morbidity is caused by cardio vascular diseases linked to pregnancy. One of the main factors contributing to the rise in morbidity and mortality among pregnant women is peripartum cardiomyopathy (PPCM), a disorder linked to cardiac dysfunction during pregnancy (Kothari et al., 1997). It has been the main contributor to maternal mortality among women who are not pregnant. Feto maternal mortality is a common occurrence in the prognosis of this disorder due to the burden on the cardio vascular system if the body in the milleu becomes damaged.

In addition to familial differences, some studies have also found regional variations in the incidence of cardiomyopathies during the peripartum period. Although this illness presents relatively frequently, more research in this area of pathology is required to avoid the ghastly prognosis of this pathology. To accurately diagnose the illness, the electrograms must show a peculiar presentation (Kuhl et al., 2005).

4. CONCLUSION

This condition's treatment choices lean heavily toward medicinal management. Various new medications have been entering clinical trials based on their efficacy rates. A good treatment plan includes bromocriptine therapy, a neo adjuvant combination of anti coagulant medications, and non pharmacological methods to help prevent the pathology from progressing. This article has examined the understanding of the aetiology, elements contributing to the severity, pathophysiology, along with the treatment options and the specific outcomes of the therapy.

Acknowledgments

We thank the participants who contributed to this case study. We also thank our project guide Dr Kamlesh Chaudhari.

Informed Consent

Written and oral consent was obtained from all the individual participants included in the study. Additional informed consent was obtained from all the individual participants for whom identifying information is included in this manuscript.

Funding

This study has not received any external funding.

Conflicts of interest

The authors declare that there are no conflicts of interests.

Data and materials availability

All data associated with this study are present in the paper.

REFERENCES AND NOTES

1. Abboud J, Murad Y, Chen Scarabelli C, Saravolatz L, Scarabelli TM. Peripartum cardiomyopathy: A comprehensive review. Int J Cardiol 2007; 118(3):295-303. doi: 10.1016/j.ijcard.2006.08.005.
2. Ansari AA, Fett JD, Carraway RE, Mayne AE, Onlamoon N, Sundstrom JB. Auto immune mechanisms as the basis for human peripartum cardiomyopathy. Clin Rev Allergy Immunol 2002; 23(3):301-24. doi: 10.1385/CRIAI:23:3:301.

3. Cemin R, Janardhanan R, Daves M. Peripartum cardiomyopathy: An intriguing challenge. Case report with literature review. Curr Cardiol Rev 2009; 5(4):268-272. doi: 10.2174/157340309789317896.
4. Demakis JG, Rahimtoola SH. Peripartum cardiomyopathy. Circulation 1971; 44(5):964-8. doi: 10.1161/01.cir.44.5.964.
5. Fett JD, Ansari AA, Sundstrom JB, Combs GF. Peripartum cardiomyopathy: A selenium disconnection and an auto immune connection. Int J Cardiol 2002; 86(2-3):311-6. doi: 10.1016/s0167-5273(02)00359-5.
6. Kothari SS. Aetiopathogenesis of peripartum cardiomyopathy: Prolactin selenium interaction? Int J Cardiol 1997; 60(1):111-4. doi: 10.1016/s0167-5273(96)02906-3.
7. Kühl U, Pauschinger M, Seeberg B, Lassner D, Noutsias M, Poller W, Schultheiss HP. Viral persistence in the myocardium is associated with progressive cardiac dysfunction. Circulation 2005; 112(13):1965-70. doi: 10.1161/CIRCULATIONAHA.105.548156.
8. Libby P, Bonow RO, Mann DL, Zipes DP, editors. Braunwald's heart disease: A textbook of cardio vascular medicine. 8th ed Philadelphia, PA: Saunders 2007.
9. Lucie Karafiatova, Marie Lazarova, Milos Taborsky, Peripartum cardiomyopathy. A case report and concise review. Cor et Vasa 2017; e272-e276. doi: 10.1016/j.crvasa.2016.12.013.
10. Marx JA, Hockberger RS, Walls RM, editors. Rosen's emergency medicine: Concepts and clinical practice. 6th ed. Philadelphia, PA: Elsevier Health Sciences 2006.
11. Pearson GD, Veille JC, Rahimtoola S, Hsia J, Oakley CM, Hosenpud JD, Ansari A, Baughman KL. Peripartum cardiomyopathy: National Heart, Lung, and Blood Institute and Office of Rare Diseases (National Institutes of Health) workshop recommendations and review. JAMA 2000; 283(9):1183-8. doi: 10.1001/jama.283.9.1183.
12. Simon PM, Schwartzstein RM, Weiss JW, Fencl V, Teghtsoonian M, Weinberger SE. Distinguishable types of dyspnea in patients with shortness of breath. Am Rev Respir Dis 1990; 142(5):1009-14. doi: 10.1164/ajrccm/142.5.1009.
13. Sliwa K, Fett J, Elkayam U. Peripartum cardiomyopathy. Lancet 2006; 368(9536):687-93. doi: 10.1016/S0140-6736(06)69253-2.
14. Wang M. Peripartum cardiomyopathy: Case reports Perm J 2009; 13(4):42-45. doi: 10.7812/TPP/08-079.