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PERI-PARTUM SEPSIS INDUCED CARDIOMYOPATHY: CASE REPORT WITH LITERATURE REVIEW

Anaesthesiology

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ABSTRACT

Heart failure can very rarely be caused by postpartum cardiomyopathy. Heart failure symptoms frequently resemble those of a typical pregnancy, delaying diagnosis and leading to avoidable complications. 24-year-old previously healthy pregnant women developed Sepsis induced peripartum cardiomyopathy. In this case report we discuss peripartum cardiomyopathy, mechanism, clinical diagnosis, imaging modalities, methods for management and advise for the patient. The present case report concludes that the diagnosis of peripartum cardiomyopathy should be considered in any pregnant or postpartum woman with symptoms concerning for heart failure. Most patients get successfully recovered if diagnosed in early stages and treatment received on time.

KEYWORDS

INTRODUCTION

The definition of peripartum cardiomyopathy has been widened because women who presented before the last month of gestation were found to be clinically indistinguishable from patients with classically-defined peripartum cardiomyopathy. When no other cause is apparent, peripartum cardiomyopathy should be taken into consideration as a diagnosis of exclusion in women who present with heart failure brought on by left ventricular systolic dysfunction. Previously, peripartum cardiomyopathy was defined as symptomatic heart failure presenting in the last month of pregnancy and up to 5 months postpartum[(1)]. The 2010 Heart Failure Association of the European Society of Cardiology Working Group therefore revised the definition of Peripartum cardiomyopathy to "an idiopathic cardiomyopathy presenting with heart failure secondary to left ventricular systolic dysfunction towards the end of pregnancy or in the months following delivery, where no other cause of heart failure is found". The diagnostic criteria indicate that left ventricular ejection fraction is <45% and there may or may not be ventricular dilatation[(2)]. Heart failure can very rarely be caused by postpartum cardiomyopathy. The first description of heart failure during pregnancy dates back to 1849. The most recent diagnostic standards for peripartum cardiomyopathy include cardiac failure in a previously healthy woman in the final month of pregnancy or within five months of delivery, the absence of a known cause for the failure, the absence of demonstrable cardiac disease prior to the final month of pregnancy, and echocardiographic evidence of decreased left ventricular systolic function.

African-American women are more likely to experience it, as are women who are older at conception, have hypertensive issues during pregnancy, or have repeated gestations. Heart failure symptoms frequently resemble those of a typical pregnancy, delaying diagnosis and leading to avoidable complications.

Medical treatment is comparable to that given to heart failure caused by other etiologies with lower ejection fraction, although modifications are required during pregnancy to protect the fetus. Complete recovery, prolonged heart failure, arrhythmias, thromboembolic events, and death are some of the possible outcomes. A subsequent pregnancy carries a significant risk of recurrence and, in the event that myocardial repair is not complete, even death[(3)].

Here we present a case of 24 years old previously healthy pregnant women developed sepsis induced peripartum cardiomyopathy.

Case presentation:

A 24-year-old multipara was admitted to labour room for normal vaginal delivery at 37 weeks. Her vitals on admission were as follow, heart rate 120/min, blood pressure 96/50 mm Hg, SpO2 98 % on room air and respiratory rate 22/min. She was febrile with tympanic temperature of 38.5 degree celsius. Her CBC showed WBC 13300 cells per cubic milimeter, hemoglobin 10.5 gm /dl, Plat 202000 cells per cubic millimeter, and CRP 16.6 mg/dl. She requested labour epidural analgesia but ongoing fever and high WBC count - explained the epidural analgesia procedure and associated risks and benefits to the patient and relatives. Subsequently, patient refused for labour epidural analgesia and opted for other mode of labour pain management. Early morning (four hours after first contact of counselling for pain management); patient had normal vaginal delivery which was complicated by primary PPH. She bled approximately 800 ml as per surgeon in labour room. Surgeon pushed the patient in OR as emergency class 1 for Examination under anesthesia for on-going bleeding. Patient was conscious and oriented. Her preoperative vitals were as follow, heart rate 135 /min with low volume pulse , blood pressure 90/45 mmHg , Spo2 95 % on room air and respiratory rate 30/min. She was looking dehydrated by her physical signs and symptoms.

There was no time for resuscitation so immediately planned for general anaesthesia with RSI and associated risk and benefits with GA were explained to the patient and relatives. After pre-oxygenation for 3 min with 100 % oxygen, rapid sequence induction was performed with Propofol 100 mg, Fentanyl 100 mcg and Rocuronium 50 mg IV. Her airway was managed with cuffed ETT size of 7.0 and kept on SIMV-PCV mode during intra-operatively. Intra-op her vitals were as follows, heat rate 135 /min, systolic blood pressure 86-96 mmHg and SpO2 97-99 % with 45 % Fio2. She required 3 bolus of phenylephrine 50 mcg to maintain her MBP > 65 mmHg. The procedure was finished in half an hour with further blood loss of 200 ml and emergence was smooth.

The patient was transferred to recovery room with vitals of HR 135 /min , BP 92/50 mmHg and SpO2 97 % with Oxygen 4 lit /min via face mask. During her stay in the post-operative care unit, her condition getting deteriorated. Her Spo2 slowly started falling and it reached 87% with Oxygen 4 lit /min. To improve her oxygenation status, high flow nasal cannula with 10-12 lit /min oxygen was initiated and her SpO2 improved and reached 96 - 97%. Her arterial blood gas report in PACU showed metabolic acidosis compensated with respiratory alkalosis with PaO2 of 60%. Her BP and HR were 86/50 mmHg and 135 / min subsequently. The Chest examination revealed B/L basal crepitation with respiratory rate of 30-34 /min and Chest X ray report showed B/L infrahilar airspace opacification, significant in right side.

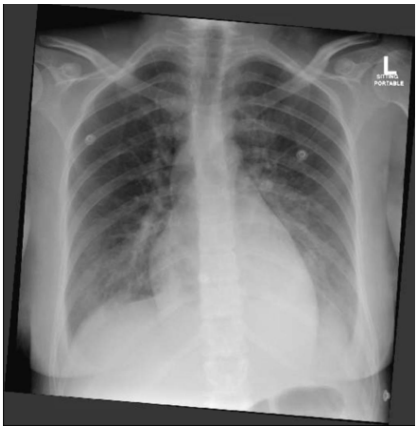

Figure 1: Chest X-ray: Infrahilar airspace opacification

Patient was shifted to ICU for further evaluation and management. In ICU, Her blood pressure started to drop down (BP 82 /40 mmhg). Therefore, Nor-epinephrine (vasopressor) infusion was initiated and she responded well with it. During her ICU stayed: CRP was raised from 16.3 mg/lit > 61.9 mg/lit > 119.6 mg/lit and Pro calcitonin was > 100 ng/lit. Therefore, Tazocine and Augmentine I.V antibiotics were started in the ICU. Her Chest auscultation revealed bilateral basal crepitation along with raised jugular venous pressure of +3. In addition, her echocardiogram report showed severe left ventricular systolic dysfunction especially at apex area with ejection fraction (EF) of 25-30% and moderate mitral regurgitation. With the above clinical picture along with raised cardiac enzyme (Trop T 422ng/lit), she was diagnosed as peri-partum cardiomyopathy and started treatment with Beta blocker and Angiotensin converting enzyme inhibitors in ICU. Subsequently, her condition was improved and wean off from Nor-epinephrine on 3rd day of ICU stay. Her vitals were as follows: blood pressure 110/68 mmHg, heart rate 88/min, respiratory rate 22 /min and SpO2 98 % with oxygen of 4 lit /min with face mask. In addition, her repeated echocardiogram report showed ejection fraction 30-35 % with improved contractility of left ventricle. Overall, her general condition was improved, and she was transferred to the ward on 4th postoperative day, where antibiotics, beta blockers and ACEI were continued. Three days later, she was discharged from ward with follow-up advice to the cardiology clinic. 22 /min. Repeat Echo showed EF 30-35 %, improved contractility in LV Apex. So, she transfers to the ward where antibiotics, beta blockers and ACEI were continued.

DISCUSSION

Global estimates of the prevalence of peripartum cardiomyopathy vary by geographical area, with estimates ranging from 1 in 100 births in Nigeria[4] and 1 in 300 births in Haiti[5] to 1 in 20,000 births in Japan[6]. The reported incidence rates may be rising as a result of rise in maternal age, an increase in multifetal pregnancies brought on by modern reproductive treatments, and possibly to increased recognition of the disease. Additionally, many cases can go unrecognized; as a result, the true incidence is unknown. Peripartum cardiomyopathy has been linked to a number of risk factors. African-American ancestry, pre-eclampsia, hypertension, multiple pregnancies, older maternal age, and familial history of cardiomyopathy are a few of them. Closer monitoring of pregnant women who are at most risk could result in earlier diagnosis and treatment. The etiology of peripartum cardiomyopathy is not fully understood and is likely multifactorial. Nutritional deficits[7], viral myocarditis[8], and autoimmune processes[9] have all been proposed as potential but unproven reasons for the development of peripartum cardiomyopathy. Pregnancy-related hemodynamic stress has been proposed as a potential cause. Most peripartum cardiomyopathy cases in women are discovered after delivery, usually during the first month following delivery[10]. Due to the disease's under-recognition and the similarities between heart failure symptoms and those of a healthy pregnancy, there are frequently delays in diagnosis.

Unfortunately, delayed diagnosis is associated with worse outcomes and a higher incidence of avoidable complications. A majority of women exhibit the classic signs and symptoms of heart failure, such as shortness of breath on effort, tiredness, orthopnea, paroxysmal nocturnal dyspnea, edema, and tightness in the chest. Tachycardia, pulmonary rales, increased jugular venous pressure, tachypnea, and peripheral edema are frequently found during a physical examination. Only a small percentage of patients present with thromboembolic complications, severe arrhythmias, and cardiogenic shock. Contrary to a typical pregnancy, peripartum cardiomyopathy typically is associated with higher levels of B-type natriuretic peptide and N-terminal pro-brain natriuretic peptide (NT-proBNP). The left bundle branch block (LBBB) pattern, sinus tachycardia and interventricular delay is seen on an electrocardiogram. On a chest radiograph, pulmonary edema is often seen, and the cardiac silhouette may appear enlarged or there may be pleural effusions (or both). It can be distinguished from other causes by echocardiography, which typically reveals right ventricular and bi-atrial enlargement, mitral and tricuspid regurgitation, pulmonary hypertension, and intracardiac thrombus in addition to left ventricle dilatation of varying degrees and left ventricle systolic dysfunction. Ejection fraction less than 45%, end-diastolic diameter more than 2.7 cm/m^2, and/or M-mode fractional shortening less than 30% are all echocardiography criteria's required for the diagnosis of peripartum cardiomyopathy. When a precise calculation of the ejection fraction (EF) is required, cardiac magnetic resonance imaging can also be employed in the diagnosis. Unless another cause of heart failure (such as cardiac sarcoidosis, giant cell myocarditis, etc.) is suspected, a cardiac biopsy is typically not recommended[11]. The diagnosis of peripartum cardiomyopathy is one of exclusion. It's crucial to pay close attention to any potential pre-existing heart illness, such as cardiomyopathies and valve disease, in order to prevent over diagnosis. Severe pre-eclampsia can result in heart failure linked to diastolic dysfunction. However, peripartum cardiomyopathy is only identified when there is systolic dysfunction.

The typical course of treatment focuses on symptomatic management for heart failure[12]. The patient's volume status is optimized using standard heart failure therapy. Angiotensin converting enzyme inhibitors (ACEIs) and beta 1 selective blockers are the two most often prescribed medications that have been found to reduce mortality[13]. However, pregnant individuals should not take an ACEI. Diuretics are frequently used to relieve heart failure symptoms. The symptoms of heart failure in cardiomyopathies caused by pregnancy have been found to be improved by new anti-heart failure drugs such sacubitril/valsartan[14]. According to recent research, the peripartum period's increased oxidative stress boosts the production of aberrant 16-kDa prolactin, which has harmful effects on cardiac myocytes. In small studies, bromocriptine, a dopamine receptor agonist with prolactin-blocking capabilities, has been linked to better results. It lessens the impact of 16-kDa prolactin on cardiac myocytes[15]. When pharmacological therapy alone is inadequate, cardiac resyn chronization therapy has also been demonstrated to increase ejection fraction and outcomes[16].

Heart failure, cardiogenic shock, arrhythmias, thromboembolic events such as stroke, left ventricle apical clots, and transient ischemic attack (TIA) are all complications of peripartum cardiomyopathy. Based on racial groups, geographic location, and length of follow-up, mortality estimates vary dramatically. In the IPAC research (Investigations of Pregnancy Associated Cardiomyopathy), mortality rates at 1-year follow-up varied from 4% to 11% in a group of 96% African-American women[17].

The left ventricular ejection fraction at the time of diagnosis is the most accurate predictor of adverse outcomes or long-term recovery among the different prognostic variables that have been researched. Lower rates of recovery and a higher risk of unfavorable outcomes were found in the IPAC cohort when the left ventricular ejection fraction was less than 30% and the left ventricular size was greater than 6 cm. Some women will recover despite having substantial left ventricular dysfunction at the time of diagnosis, hence the initial left ventricular ejection fraction alone cannot predict when advanced therapy may be necessary. Obesity left ventricular thrombus, right ventricular systolic dysfunction, and left ventricular dilatation are additional indicators of a poorer outcome. Lower recovery rates, longer recovery times, more negative outcomes, and higher mortality rates are all substantially connected with being of African-American origin. Biomarkers associated with adverse outcomes include troponin, NT-proBNP, and sFlt1 (soluble fms-like tyrosine kinase 1)[18].

Compared to other types of heart failure with lower left ventricle ejection fraction, peripartum cardiomyopathy has been linked to a higher rate of recovery, and recovery frequently happens within the first 3 to 6 months. Also possible is a delayed recovery, even up to two

years after diagnosis[19]. Few studies indicate that breastfeeding is safe. Women who are acutely unwell should be treated by specialized interdisciplinary teams and might need advanced heart failure therapies. Women considering a subsequent pregnancy should be counseled and monitored by physicians familiar with peripartum cardiomyopathy. Long-term monitoring is crucial.

CONCLUSION:

The present case report concludes that the diagnosis of peripartum cardiomyopathy should be considered in any pregnant or postpartum woman with symptoms concerning for heart failure. An echo cardiography should always be performed after a high BNP level to check for systolic dysfunction. Early diagnosis and administration of pregnancy- and lactation-specific medicines may help to avoid negative effects.

REFERENCES

1. Elkayam U, Akhter MW, Singh H, Khan S, Bitar F, Hameed A, et al. Pregnancy-associated cardiomyopathy: clinical characteristics and a comparison between early and late presentation. Circulation. 2005 Apr 26;111(16):2050–5.
2. Sliwa K, Hilfiker-Kleiner D, Petrie MC, Mebazaa A, Pieske B, Buchmann E, et al. Current state of knowledge on aetiology, diagnosis, management, and therapy of peripartum cardiomyopathy: a position statement from the Heart Failure Association of the European Society of Cardiology Working Group on peripartum cardiomyopathy. Eur J Heart Fail. 2010 Aug;12(8):767–78.
3. Brar SS, Khan SS, Sandhu GK, Jorgensen MB, Parikh N, Hsu JWY, et al. Incidence, mortality, and racial differences in peripartum cardiomyopathy. Am J Cardiol. 2007 Jul 15;100(2):302–4.
4. Isezuo SA, Abubakar SA. Epidemiologic profile of peripartum cardiomyopathy in a tertiary care hospital. Ethn Dis. 2007;17(2):228–33.
5. Fett JD, Christie LG, Carraway RD, Murphy JG. Five-year prospective study of the incidence and prognosis of peripartum cardiomyopathy at a single institution. Mayo Clin Proc. 2005 Dec;80(12):1602–6.
6. Kamiya CA, Kitakaze M, Ishibashi-Ueda H, Nakatani S, Murohara T, Tomoike H, et al. Different characteristics of peripartum cardiomyopathy between patients complicated with and without hypertensive disorders. -Results from the Japanese Nationwide survey of peripartum cardiomyopathy-. Circ J Off J Jpn Circ Soc. 2011;75(8):1975–81.
7. Fett JD, Ansari AA, Sundstrom JB, Combs GF. Peripartum cardiomyopathy: a selenium disconnection and an autoimmune connection. Int J Cardiol. 2002 Dec;86(2–3):311–6.
8. Bültmann BD, Klingel K, Näbauer M, Wallwiener D, Kandolf R. High prevalence of viral genomes and inflammation in peripartum cardiomyopathy. Am J Obstet Gynecol. 2005 Aug;193(2):363–5.
9. Ansari AA, Fett JD, Carraway RE, Mayne AE, Onlamoon N, Sundstrom JB. Autoimmune mechanisms as the basis for human peripartum cardiomyopathy. Clin Rev Allergy Immunol. 2002 Dec;23(3):301–24.
10. Fett JD. Earlier detection can help avoid many serious complications of peripartum cardiomyopathy. Future Cardiol. 2013 Nov;9(6):809–16.
11. Hibbard JU, Lindheimer M, Lang RM. A modified definition for peripartum cardiomyopathy and prognosis based on echocardiography. Obstet Gynecol. 1999 Aug;94(2):311–6.
12. Khan A, Paré E, Shah S. Peripartum Cardiomyopathy: a Review for the Clinician. Curr Treat Options Cardiovasc Med. 2018 Sep 29;20(11):91.
13. Ma GG, Fang Q, Wang FX. The effect of beta-blockers on mortality in patients with heart failure and atrial fibrillation: A meta-analysis of observational cohort and randomized controlled studies. Cardiol J. 2019;26(6):744–52.
14. Gaddipati VC, Patel AA, Cohen AJ. The Use of a Novel Heart Failure Agent in the Treatment of Pregnancy-Associated Cardiomyopathy. Case Rep Cardiol. 2017; 2017: 9561405.
15. Sliwa K, Blauwet L, Tibazarwa K, Libhaber E, Smedema JP, Becker A, et al. Evaluation of bromocriptine in the treatment of acute severe peripartum cardiomyopathy: a proof-of-concept pilot study. Circulation. 2010 Apr 6;121(13):1465–73.
16. Mouquet F, Mostefa Kara M, Lamblin N, Coulon C, Langlois S, Marquie C, et al. Unexpected and rapid recovery of left ventricular function in patients with peripartum cardiomyopathy: impact of cardiac resynchronization therapy. Eur J Heart Fail. 2012 May;14(5):526–9.
17. McNamara DM, Elkayam U, Alharethi R, Damp J, Hsich E, Ewald G, et al. Clinical Outcomes for Peripartum Cardiomyopathy in North America: Results of the IPAC Study (Investigations of Pregnancy-Associated Cardiomyopathy). J Am Coll Cardiol. 2015 Aug 25;66(8):905–14.
18. Habli M, O'Brien T, Nowack E, Khoury S, Barton JR, Sibai B. Peripartum cardiomyopathy: prognostic factors for long-term maternal outcome. Am J Obstet Gynecol. 2008 Oct;199(4):415.e1-5.
19. Fett JD, Sannon H, Thélisma E, Sprunger T, Suresh V. Recovery from severe heart failure following peripartum cardiomyopathy. Int J Gynaecol Obstet Off Organ Int Fed Gynaecol Obstet. 2009 Feb;104(2):125–7.