Peripartum Cardiomyopathy- A Case Report

[1] S. Sreelatha, [2] M. Shilpashree, [2] M. Ashwini Rani, [3] Renuka Ramaiah

[1]Associate Professor, [2]Post graduates, [3]Professor

Department of OBG, ESICMC & PGIMSR, Rajajinagar, Bangalore, Karnataka, India.

Abstract

Peripartum cardiomyopathy (PPCM) is a dilated cardiomyopathy defined as systolic cardiac heart failure in the last month of pregnancy or within five months of delivery. Its diagnosis is often delayed because its symptoms closely resemble those within the normal spectrum of pregnancy and the postpartum period. When PPCM is misdiagnosed or its diagnosis is delayed, the consequences for patients are deadly. The disorder carries a high mortality rate. We report a case of a 27 year old female who delivered by caesarean section and developed acute onset of breathlessness on post operative day 1. On further evaluation, ECG and ECHO changes revealed cardiomegaly with reduced ejection fraction. A diagnosis of peripartum cardiomyopathy was made and the patient was treated accordingly.

Key words: Peripartum cardiomyopathy, pregnancy

Introduction

Peripartum cardiomyopathy (PPCM) is a rare and life-threatening cardiomyopathy of unknown etiology that affects women in the last month of pregnancy or in the first five months postpartum [1]. The incidence of PPCM ranges from 1:300 to 1:15,000 live births. The definition of PPCM includes four criteria: 1) development of cardiac failure in the last month of pregnancy or within five months of delivery, 2) absence of an identifiable cause for the cardiac failure, 3) absence of recognizable heart disease before the last month of pregnancy, and 4) left ventricular (LV) dysfunction (ejection fraction of less than 45% or reduced shortening fraction) [2,3]. Risk factors include multiparity, black race, older maternal age, pre-eclampsia, and gestational hypertension [4]. Symptoms of PPCM, which include fatigue, edema, and dyspnea, are similar to those for the normal spectrum of peripartum states and pregnancy comorbidities such as pulmonary emboli and eclampsia. Therefore, diagnosis is often delayed and the disorder is under recognized, with devastating consequences. Mortality is as high as 20% to 50% [5].

Case report

A 27year old lady with $G_2P_1L_1$ with term gestation with previous lower segment caesarean section (LSCS) presented with c/o pain abdomen since one day. On admission, the pulse rate was 104/min, BP 110/70 mmHg. There was no pallor. On per abdomen and pelvic examination patient was in labour. Fetal heart rate was 140 bpm. There was no scar tenderness. Non stress testing (NST) was non-reassuring. Emergency LSCS was done and extracted a single alive male baby of 2.3kgs with two loops of cord around the neck. There were no intra operative complications. The patient developed acute onset of breathlessness 8 hours post operatively. On examination, pulse rate-134/min, blood presure 100/60 mmHg , respiration rate -32/min. On RS examination, bilateral crackles were heard and oxygen saturation was 89%.The patient was shifted to the ICU and treated with oxygen and diuretics. After three hours she developed orthopnea and PR was 160/min and RR 40/min. Cardiac consultation was

Address for correspondence
Dr. Sreelatha S, Associate Professor Dept. of OBG
ESICMC and PGIMSR Rajajinagar, Bangalore Karnataka India
E- mail: dr.sreelatha2011@gmail.com

sought and X ray showed cardiomegaly with nonhomogenous opacities in the lower zone. ECG showed sinus tachycardia. 2D ECHO showed reduced left ventricular function with ejection fraction (EF) 37%. The patient was diagnosed as peripartum cardiomyopathy.The patient was treated with diuretics, digoxin, anticoagulants and dobutamine infusion. Patient's condition improved after 48hrs and was shifted out of intensive care unit and the patient was discharged on tenth post operative day. At the time of discharge ECHO revealed an EF of 65% and X ray showed clearance of opacities with cardiomegaly.

Discussion

A possible relationship between pregnancy and dilated cardiomyopathy was recognized as early as the1870s and was classified as a distinct clinical entity in the 1930s. Yet the cause of PPCM is still unknown. Most postulate that it is related to the cardiovascular stress of pregnancy (increased fluid load). Other researchers further postulate that PPCM may be an inflammatory response in pregnancy, citing an elevation of tumor necrosis factor-alpha and interleukin-6 levels [6,7]. Multiparity, advanced maternal age, multifetal pregnancy, pre-eclampsia, gestational hypertension and African ethnicity are risk factors. Other risk factors include association with maternal cocaine abuse or selenium deficiency, long-term (more than four weeks) oral tocolytic therapy with beta adrenergic agonists such as terbutaline. However, the disease can occur in women without any risk factors.

Clinical features of PPCM include symptoms of congestive heart failure and chest pain. Signs can include tachycardia, tachypnea, pulmonary rales, an enlarged heart, and third heart sound. Such signs and symptoms overlap with those of many other conditions, ranging from normal pregnancy to pulmonary emboli and upper respiratory infection. Diagnosis of PPCM includes the four criteria described at the beginning of this report.

There are no specific laboratory abnormalities for PPCM. However, other exclusionary laboratory studies should also be considered, including cardiac enzymes assessment and a pre-eclampsia workup. Electrocardiographic findings are often normal but can include sinus tachycardia, nonspecific ST- and T-wave abnormalities, and voltage abnormalities [8]. Chest radiographs can show signs of pulmonary congestion, cardiac enlargement, and even pleural effusions in some cases. Echocardiograms usually show decreased contractility and LV enlargement without hypertrophy [9].

The treatment for PPCM is the same as for other forms of congestive heart failure (fluid and salt restriction, β-blocker, diuretic, and digoxin), except for angiotensin converting enzyme inhibitors and angiotensin-receptor blockers, which are contraindicated in pregnancy [10]. Hydralazine can be used during pregnancy to reduce afterload. Patients with PPCM are also at high risk for thrombus formation; thus, anticoagulation should be considered especially for high-risk patients with severe LV dysfunction. In addition, physical activity should be encouraged according to patients' tolerance of symptoms. Immunosuppressive therapy has no clear cut efficacy and, therefore, is not currently recommended. Intravenous immune globulin may play a role in women with PPCM. Women who fail maximal medical management may be candidates for cardiac transplantation.

Regardless of recovery, however, a second pregnancy is usually not recommended for these patients because PPCM recurs in more than 30% of subsequent pregnancies, which puts both mother and baby at great risk. The prognosis for women with PPCM appears to depend on the normalization of left ventricular size and function within six months after delivery. About half of the patients of PPCM recover without any complications. Most hearts that are destined to recover normal function probably do so within six months from the time of diagnosis.

The persistence of cardiac dysfunction 6 to 12 months after the initial diagnosis of PPCM usually indicates an irreversible problem and almost always represents an absolute contraindication to a subsequent pregnancy. The data have been conflicting in patients with PPCM in whom left ventricular function recovers. But these women appear to have some risk for recurrence. Women with PPCM who have regained normal resting left ventricular size and performance have decreased

contractile reserve. Therefore, subsequent pregnancies, if they cannot be avoided, should be managed in collaboration with a high risk perinatal centre.

Bromocriptine

The ergot alkaloid bromocriptine mesylate is a dopamine agonist that suppresses prolactin secretion. Following the concept that the cleaved form of the prolactin initiates and drives PPCM, it has been hypothesized that early blockade of prolactin with bromocriptine may improve the condition of patients with acute onset of PPCM before irreversible damage occurs.The beneficial effect results from eliminating the detrimental 16kDa prolactin form. Bromocriptine was also shown to increase oxygen consumption and resting energy expenditure. Anticoagulation therapy is strongly recommended as this has been associated with several reports of myocardial infarction [11].

Conclusion

Peripartum cardiomyopathy is a rare disease of unknown cause that strikes women in the child bearing years. Diagnosis of PPCM is challenging and requires vigilance. Once PPCM is identified, the primary goal of therapy is to alleviate symptoms of congestive heart failure.

References

1. Demakis JG, Rahimtoola SH. Peripartum cardiomyopathy. Circulation 1971;44(5):964-8.

2. Demakis JG, Rahimtoola SH, Sutton GC, et al. Natural course of peripartum cardiomyopathy. Circulation 1971 Dec;44(6):1053–61.

3. Pearson GO, Veille JC, Rahimtoola S, et al. Peripartum cardiomyopathy: National Heart, Lung, and Blood Institute and Office of Rare Diseases (National Institutes of Health) workshop recommendations and review. JAMA 2000 Mar 1;283(9):1183–8.

4. Marx JA, Hockberger RS, Walls RM, eds. Rosen's emergency medicine: concepts and clinical practice. 6th ed. Philadelphia, PA: Elsevier Health Sciences; 2006.

5. Abboud J, Murad Y, Chen-Scarabelli C, Saravolatz L, Scarabelli TM. Peripartum cardiomyopathy: a comprehensive review. Int J Cardiol 2007 Jun 12;118(3):295–30.

6. Sliwa K, Skudicky D, Bergemann A, Candy G, Puren A Sareli P. Peripartum cardiomyopathy: analysis of clinical outcome, left ventricular function, plasma levels of cytokines and Fas/APO-1. J Am Coll Cardiol 2000 Mar 1;35(3):701–5.

7. Sliwa K, Förster O, Libhaber E, et al. Peripartum cardiomyopathy: inflammatory markers as predictors of outcome in 100 prospectively studied patients. Eur Heart J 2006 Feb;27(4):411–6.

8. Davidson NM, Parry EH. The etiology of peripartum cardiac failure. Am Heart J 1979 Apr;97(4):535–6.

9. Lampert MB, Lang RM. Peripartum cardiomyopathy. Am Heart J 1995 Oct;130(4):860–70.

10. Libby P, Bonow RO, Mann DL, Zipes DP, eds. Braunwald's heart disease: a textbook of cardiovascular medicine. 8th ed. Philadelphia, PA: Saunders; 2007.

11. Hopp L, Harder B, Iffy L. Myocardial Infarction postpartum in patients taking bromocriptine for the prevention of of breast engorgement. Int J cardiol 1996;57:227-32.

How to cite this article: Sreelatha S, Shilpashree M, Ashwini Rani M, Ramaiah R. Peripartum cardiomyopathy - A case report Med Inn 2013;2:120-22
Source of funding - Nil
Conflict of interest - None declared