International Journal of Reproduction, Contraception, Obstetrics and Gynecology
Joshi AV et al. Int J Reprod Contracept Obstet Gynecol. 2017 Feb;6(2):523-526
www.ijrcog.org

pISSN 2320-1770 | eISSN 2320-1789

DOI: http://dx.doi.org/10.18203/2320-1770.ijrcog20170374

Original Research Article

A study of peripartum cardiomyopathy in a tertiary care center in India

Aditi V. Joshi*, Michelle N. Fonseca, Deepali S. Kharat-Kapote

Department of Obstetrics and Gynecology, Lokmanya Tilak Municipal General Hospital, Sion, Mumbai, Maharashtra, India

Received: 06 November 2016
Revised: 08 November 2016
Accepted: 13 December 2016

***Correspondence:**
Dr. Aditi V. Joshi,
E-mail: avj111@gmail.com

ABSTRACT

Background: Peripartum cardiomyopathy is an idiopathic and reversible form of dilated cardiomyopathy. The aim of the study was to study the mean age of presentation of peripartum cardiomyopathy in pregnant patients over a period of 1 year in a tertiary health care and study the maternal and fetal outcome of patients with peripartum cardiomyopathy.
Methods: A retrospective observational study with total of 22 patients with peripartum cardiomyopathy was diagnosed in a total of 10,279 deliveries conducted at LTMMC, Sion hospital between September 2014 to February 2016.
Results: Our study revealed that most of the affected patients were young with the mean age at presentation being 26.8±4.29 years. Most of the patients were diagnosed in the postpartum period (63.6%). the most common risk factor was pre-eclampsia (22.7%) followed by anemia (18.2%). The mean Ejection fraction at the time of presentation was 25.3±9.8%. There were 2 (9.1%) intrauterine fetal deaths and 1 neonatal death.
Conclusions: There is a need for more multi-centric studies in order to understand the underlying pathogenesis and to determine the possible early interventions to help provide better pregnancy outcome.

Keywords: Induction of labour, Prolonged pregnancy, Perinatal morbidity, Ultrasound

INTRODUCTION

Peripartum cardiomyopathy is an idiopathic and reversible form of dilated cardiomyopathy. Its incidence varies with geography and ethnicity and ranges from 1 in 1300 to 1 in 15,000 pregnancies worldwide.[1] Although peripartum cardiomyopathy is a phenotype of dilated cardiomyopathy, its clinical course is highly variable. It is a rare but potentially life threatening form of cardiac failure with a high degree of morbidity and mortality. A rapid progression to end stage of heart failure can occur within days. On the other hand a spontaneous and complete recovery may also occur.[2] Both these features make it a distinct clinical entity. The rarity of the condition and lack of awareness of it among physicians and nurses/midwives often leads to late diagnosis and treatment.

Aim and objectives

- To study the mean age of presentation of peripartum cardiomyopathy in pregnant patients over a period of 1 year in a tertiary health care.
- To study the various aetiological factors associated with peripartum cardiomyopathy.
- To study the maternal and fetal outcome of patients with peripartum cardiomyopathy.

METHODS

A retrospective Observational Study with total of 22 patients with peripartum cardiomyopathy was diagnosed in a total of 10,279 deliveries conducted at LTMMC, Sion hospital between September 2014 to February 2016.

A detailed history, physical examination, echocardiography, mode of delivery, complications and maternal as well as fetal outcome was recorded. Statistical analysis was done using SPSS.

RESULTS

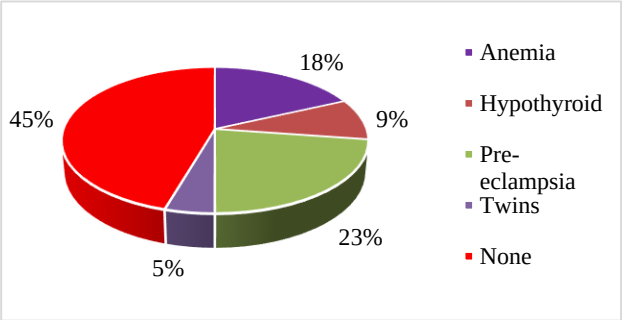

Figure 1: Antenatal risk factors encountered in the study group.

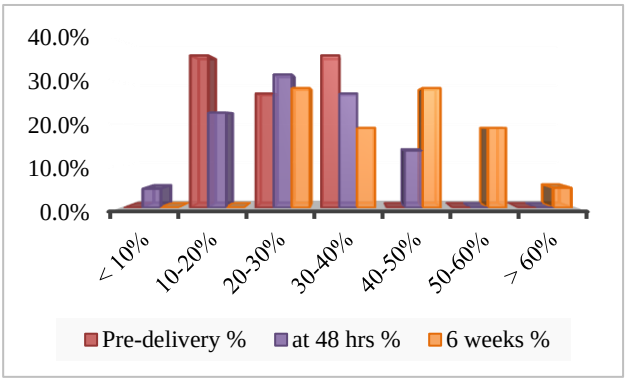

Figure 2: Comparison of 2D ECHO values pre-delivery, at 48 hours and at 6 weeks.

In a study by Elkayam et al the mean age of presentation was 31±6 years. Our study revealed that most of the affected patients were young with the mean age at presentation being 26.8±4.29 years. Most of the patients were diagnosed in the postpartum period (63.6%).

There were 4 patients who had a prior history of peripartum cardiomyopathy in the previous pregnancy. In the study by Elkayam et al the commonly found antenatal risk factors were gestational hypertension (43%), tocolytic therapy (19%), and twin pregnancy (13%). In our study, the most common risk factor was pre-eclampsia (22.7%) followed by anemia (18.2%). The mean Ejection fraction at the time of presentation was 25.3±9.8%.

At the end of 6 weeks only one patient (4.8%) showed an improved ejection fraction of >60% ($p<0.05$). 1 patient expired. Rest of them did not have an improvement of Ejection fraction; however none of them deteriorated either. Similarly, in a study conducted at D. Y. Patil Hospital at Kolhapur by Prasad et al, 61% (8/13) patients recovered completely. There was one mortality. 19 out of the 22 neonates had a good outcome (86.4%). There were 2 (9.1%) intrauterine fetal deaths and 1 neonatal death.

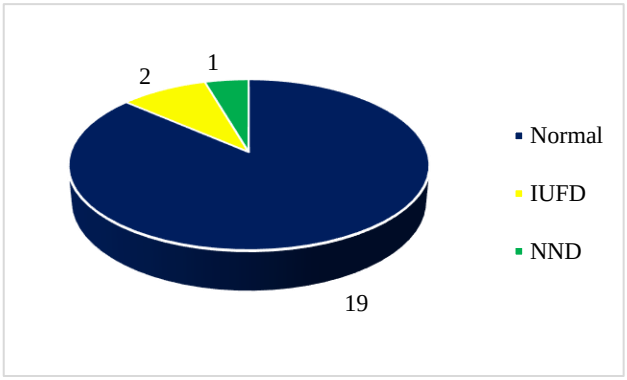

Figure 3: Neonatal outcome.

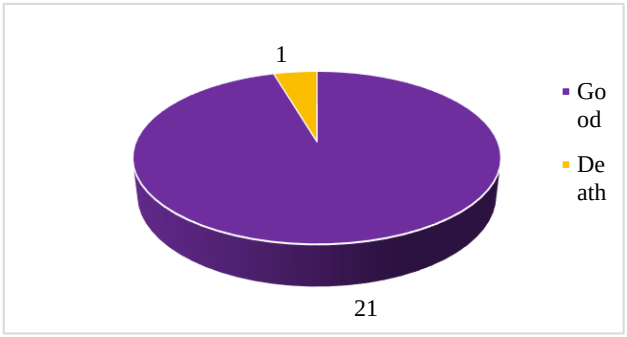

Figure 4: Maternal outcome.

DISCUSSION

Peripartum cardiomyopathy is a rare form of dilated cardiomyopathy with unknown aetiology. The modified definition of which includes:

- Development of heart failure in the last trimester or within six months postpartum
- Absence of any identifiable cause
- Absence of any recognizable heart disease before the last trimester of pregnancy
- Echocardiography criteria: ejection fraction <45%, left ventricular fractional shortening <30% or left ventricular end diastolic dimension >2.7 cm/m2 of body surface area.[3]

Peripartum cardiomyopathy has a very variable clinical course. Its diagnosis remains a challenge. The major risk factors include multiparity and advanced maternal age. Others include obesity, history of cardiac disorders like myocarditis, smoking, alcohol, use of certain drugs, preeclampsia and black race.

A study conducted in the United States observed a strong association with hypertension. This creates a dispute as to whether increased blood pressure may be a cause of heart failure seen in patients with peripartum cardiomyopathy.

However additional studies in women with preeclampsia have revealed no change in systolic left ventricular function.[4]

The proposed mechanisms include immunological, abnormal inflammatory changes, myocarditis, cytokine production and genetic predisposition. A recent study suggests that an unbalanced oxidative stress caused by prolactin sub-fragments may have a significant role [5.] Particular attention is currently given to this concept of increased oxidative stress inducing production of proapoptotic, angiostatic and proinflammatory mediators. Plasma markers of inflammation like CRP, IL-6, TNF-α were significantly elevated in these subset of patients and correlated with increased LV dimensions and lower EF at presentation.[6]

The clinical presentation and haemodynamic changes are similar to any other form of dilated cardiomyopathy which includes new or rapid onset of dyspnea, cough, chest pain, palpitations, fatigue etc. Physical examination will reveal tachycardia, decreased pulse oximetry, peripheral oedema, pulmonary rales, ascites and hepatomegaly. Arrhythmias are a common phenomenon which is responsible for the embolic episodes.

A multidisciplinary team approach is vital. Early diagnosis and prompt treatment are the keys to optimize pregnancy outcome. The choice of treatment is determined by the clinical presentation of the patient. The principles of management include continuous hemodynamic monitoring and prevention of fluid overload. The mainstay of medical therapy involves the use of afterload reducing agents like Hydralazine in combination with Nitroglycerine or Amlodipine. These are preferred in the antepartum period due to its non-teratogenic effects on the foetus. Diuretics are used for pre-load reduction and treating pulmonary congestion. Inotropic agents like digoxin is given in low output failures. The goal is to maintain the lowest dose possible such that serum digoxin levels are<1.0ng/ml. Beta blockers are recommended since they improve symptoms, ejection fraction and survival.[7]

Anti-coagulation is recommended in patients who are bedridden or have ejection fraction of <35%, or with atrial fibrillation, or obese patients. Therapy is usually continued until normal left ventricular function returns. In patients with persistent severe left ventricular (LV) dysfunction, advanced therapies like mechanical support and heart transplantation should be considered.

Upcoming modalities of therapy include pentoxifylline (inhibits tumor necrosis factor), bromocriptine, and cabergoline (antagonist of prolactin) along with newer interventions such as plasmapheresis, immunoadsorption, ventricular assist devices and heart transplantation which hold a promising future.[8] The dilemma regarding the optimum time and mode of delivery still continues. Spontaneous labour and vaginal delivery is generally acceptable in stable patients. Cesarean delivery is generally reserved for obstetrical indications. However, planned caesarean section is usually the preferred delivery mode in unstable patients who are critically ill and in need of an inotropic therapy or mechanical support.[9]

Prognosis depends on the degree of cardiomegaly and left ventricular size at presentation and in the following 6 months. Hence it is essential to maintain long-term follow up of these patients regardless of initial recovery of LV function. The treatment is usually tapered over a period of 6-12 months. A rapid recovery of EF is often seen in patients after initial diagnosis and diuresis. An EF > 45% at 2 months after diagnosis predicts full functional recovery in 75% of women.[10]

Subsequent pregnancies should be avoided in patients with persistent ventricular dysfunction. Women with recovery from a prior cardiomyopathy need to be aware of the possible recurrence in next pregnancy and need to be counselled likewise. Normalization of left ventricular function after a prior peripartum cardiomyopathy history does not guarantee an uncomplicated subsequent pregnancy; approximately 20% of such patients are also at risk of moderate to severe deterioration of LV function, which persists after delivery in 20% to 50% of patients. Early antenatal registration will enable us to diagnose these patients before acute clinical deterioration.[11] In a study conducted by Bitekar et al, Early recovery was observed only in six patients (30%) within six months of diagnosis, whereas delayed recovery, was observed in 14 out of 20 patients (70%) beyond 6 month.

Peripartum cardiomyopathy remains a perplexing condition to diagnose and treat. Changing definitions, strict time frame for definitions and echocardiographic cut-offs for diagnosis, have probably led to the condition being overlooked or misdiagnosed.

CONCLUSION

Early antenatal registration, patient education, physician alertness and awareness of this clinical entity and regular antenatal visits play a vital role in the outcome of these patients. Hence; there is a need for more multi-centric studies in order to understand the underlying pathogenesis and to determine the possible early interventions to help provide better pregnancy outcome.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. Fennira S, Demiraj A, Khouaja A, Boujnah MR. Peripartum cardiomyopathy. Annales de Cardiologie et D'angéiologie. 2006;55(5):271-5.

2. Pandit V, Shetty S, Kumar A. Incidence and out come ofperipartum Cardiomyopathy from a tertiary hospitalin South India.Trop Doct. 2009;39:168-9.
3. Pearson GD, Veille JC, Rahimttola S. PeripartumCardiomyopathy; National Heart, Lung and Blood Institute and Office of Rare Diseases (National Institute of Health) workshoprecommendation and review. JAMA. 2000;283:1183-88.
4. 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;111(16):2050.
5. Sliwa K, Hilfiker-Kleiner D, Petrie MC. 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. European Journal of Heart Failure, 12(8), 767–78.
6. Sarojini A, Sai Ravi Shanker A, Anitha, M. Inflammatory Markers-Serum Level of C-Reactive Protein, Tumor Necrotic Factor-α, and Interleukin-6 as Predictors of Outcome for Peripartum Cardiomyopathy. Journal of Obstetrics and Gynaecology of India. 2013;63(4):234-9.
7. Bhattacharyya A, Basra SS, Sen P. Peripartum cardiomyopathy: a review. Texas Heart Institute Journal/from the Texas Heart Institute of St. Luke's Episcopal Hospital, Texas Children's Hospital. 2012;39(1):8-16.
8. Mishra VN, Mishra, N, Devanshi. Peripartum cardiomyopathy. The Journal of the Association of Physicians of India. 2013;61(4):268-73.
9. Davis M, Duvernoy C. Peripartum cardiomyopathy: current knowledge and future directions. Women's Health (London, England). 2015;11(4):565-73.
10. Peripartum Cardiomyopathy: A Current Review KatieM. Twomley and Gretchen L.Wells: Hindawi Publishing Corporation Journal of Pregnancy. 2010; Article ID 149127.
11. 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;12(8):767-78.

Cite this article as: Joshi AV, Fonseca M, Kharat-Kapote D. A study of peripartum cardiomyopathy in a tertiary care center in India. Int J Reprod Contracept Obstet Gynecol 2017;6:523-6.