



A PROSPECTIVE STUDY TO ACCESS RELATION BETWEEN PERIPARTUM CARDIOMYOPATHY AND ASCITES, MULTIPARITY AND TWIN GESTATION.

Obstetrics & Gynaecology

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ABSTRACT

Peripartum cardiomyopathy is a rare complication in pregnancy but is more commonly found in the patients of preeclampsia and eclampsia. The purpose of study is to evaluate incidence of peripartum cardiomyopathy among cases of preeclampsia and eclampsia. This is a prospective observational study. A total of 190 cases of preeclampsia and eclampsia were studied for features of PPCM and subjected to echocardiography. Out of 190 subjects, 3.7% had findings suggestive of PPCM. Subjects were accessed for relation between peripartum cardiomyopathy and ascites, multiparity and twin gestation.

KEYWORDS

peripartum cardiomyopathy, preeclampsia, eclampsia, ascites, multiparity, twin gestation.

INTRODUCTION

PPCM is a rare form of dilated cardiomyopathy of unknown origin, that is unique to the pregnant women of all reproductive ages.^{1,2,3} The real incidence is unknown. It affects previously healthy pregnant women with a low incidence of 0.1% of pregnancies but has a high morbidity and mortality rate ranging from 7% to 50%.^{3,4,5} The precise incidence in India is not known, an incidence of one case per 1374 live births has been reported from a tertiary care hospital from South India.⁶ Reported incidences range from 1 in 299 live births in Haiti,⁷ to 1 in 100 in Northern parts of Nigeria,^{7,8} to 1 case per 6000 live births in Japan,⁹ to 1 in 1000 in South Africa,¹⁰ to 1 in 2229 live births in Southern California,¹¹ to 1 in 4000 live births in the United State.¹² There is wide variation in the incidence of PPCM because the diagnosis is not always consistent and a comparison with age-matched non-pregnant women does not exist.^{9,12,13}

Peripartum cardiomyopathy (PPCM) is defined as the development of cardiac failure between the last month of pregnancy and 6 months postpartum, the absence of an identifiable cause, the absence of recognizable heart disease prior to the last month of pregnancy, and left ventricular systolic dysfunction demonstrated by classic echocardiographic criteria.^{4,15,12,16,17,18}

Modified PPCM diagnostic criteria includes 3 clinical and 1 echocardiographic criteria. These are –

Clinical Criteria

1. The development of heart failure in the last month of pregnancy or first 6 months postpartum.
2. The absence of a identifiable cause for cardiac failure.
3. The absence of recognizable heart disease prior to last month of pregnancy.

Echocardiographic Criteria

1. LVEF(Left Ventricular Ejection Fraction) <45%
2. FS(Fractional shortening) <22%
3. LVEDD(Left Ventricular End Diastolic Dimension) >2.7 cm/m² BSA or >5.5 cm.

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.

PPCM is more frequent in women at the extremes of childbearing ages and in older women of higher parity.^{12,19} PPCM has been reported in 24-37% of young primigravidae.^{20,20}

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.

The pathophysiology is still controversial. The basis of human PPCM

cannot be explained by a single etiology, thus, the disease has multifactorial origin.¹⁸

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, peripheral oedema, pulmonary edema, ascites and hepatomegaly. Arrhythmias are a common phenomenon which is responsible for the embolic episodes. In some patients, the clinical and echocardiographic status improve rapidly and may return to normal while for others it may progress and the clinical condition rapidly worsens, even with medical therapy to chronic cardiac failure and sudden cardiac death.^{12,21}

In acute cases, treatment may involve the use of intravenous vasodilatation, inotropic medications, an intra-aortic balloon pump, ventricular-assist devices, and extracorporeal membrane oxygenation. In severe cases, women experience a rapid deterioration in health without improvement even with medical therapy, and may require cardiac transplantation or die of heart failure, thrombo-embolic events and cardiac arrhythmias. However, the initial severity of left ventricular dysfunction or dilatation is not necessarily predictive of long-term functional outcome.²¹ Survivors of PPCM often recover from the left ventricular dysfunction, however, they may be at risk of recurrence for heart failure and death in subsequent pregnancies.

This Study Was Carried Out

- To find out patients having clinical features suggestive of PPCM.
- To find out how many of these cases have deranged myocardial function which is suggestive of peripartum cardiomyopathy thereby finding out the incidence of these cases in preeclampsia and eclampsia.
- To find out relation between PPCM and ascites, multiparity, twin gestation.

Methods

The present study was a prospective observational study carried out between 1st march 2017 to 31st august 2018 in the department of obstetrics & gynaecology and non invasive echocardiography lab Netaji Subhash Chandra Bose Medical College and Hospital Jabalpur after obtaining ethical committee approval. All the women admitted with diagnosis of preeclampsia and eclampsia was taken for study. Out of 800 admitted preeclampsia and eclampsia patients 190 cases selected by taking every 4th case as study subjects through systematic sample selection. Echocardiography of all patients was done either antepartum or postpartum before discharge. If patient was very critical on ventilator then echocardiography was done after she is weaned. Patients were accessed for symptoms and signs of peripartum cardiomyopathy. Patient's necessary sociodemographic details, vitals, symptoms and sign of impending eclampsia, blood investigation, obstetric history, gestational age at the time of admission, mode of delivery, maternal outcome and neonatal outcome also recorded in proforma. All these data was then entered in a structured schedule in

microsoft excel sheet. All the records rechecked for completeness and consistencies. Non numeric entries were coded into nominal/ordinal distribution before analysis. Categorical variables were summarized in frequency and percent distribution and Chi square test was performed as appropriate and Fischer's Exact test was applied where frequency was < 5. Correlation coefficient analysis was applied to measure strength of association. Total 7 cases of PPCM diagnosed during period of study.

RESULTS

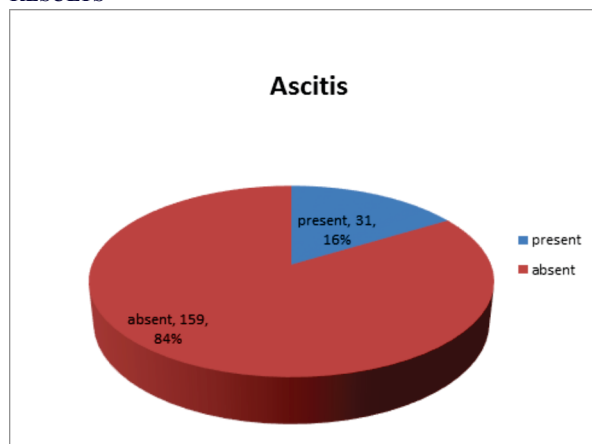


Figure No. 1: Distribution Of Study Subjects According To Ascites

Figure No. 2: Distribution Of Study Subjects Echo Findings According To Ascites

Ascites	Present	Absent	Total
PPCM Found	5(71.4%)	2(28.6%)	7
PPCM Not found	29(15.9%)	154(84.1%)	183

Chi square = 14.18, P value = 0.002 (fischer's exact was applied as frequency was less than 5).

Figure no. 2 shows that 71.4% PPCM populations had ascites while ascites is present in only 15.9% of rest population.

Figure No. 3: Distribution Of Study Subjects According To Parity

Parity	Frequency	Percent
Primiparous	120	63.2
Multiparous	70	36.8
Total	190	100.0

Figure no. 3 depicts the parity status of study subjects shows that majority of women (63.2%) were primiparous followed by multiparous(36.8%).

Figure No. 4: Distributions Of Study Subjects Echo Findings According To Parity

Parity	Primiparous	Multiparous	Total
PPCM found	5(71.4%)	2(28.6%)	7
PPCM not found	115(62.8%)	68(37.2%)	183
	120	70	190

Chi square = 0.2, P value = 1.0

Figure no.4: shows that in 7 cases in which PPCM diagnosed 2(28.6%) were multiparous and 5(71.4%) were primiparous.

Figure No.5: Distribution Of Study Subjects According To Twin Gestation

Twins	Frequency	Percent
No	178	93.7
Yes	12	6.3
Total	190	100

Figure No.6: Distribution Of Study Subjects According To Echocardiographic Finding And Twin Gestation

Twins	Yes	No	Total
PPCM found	1(14.3%)	6(85.7%)	7
PPCMnot found	11(6.01%)	172(93.99%)	183
Total	12	178	190

Chi square = 0.78, P value = 0.371

Figure no.5 shows that majority of pregnancies were singleton (93.7%) followed by twins pregnancies(6.3%).

Figure no.6 shows that out of 12 twin gestation only 1 had PPCM while in 178 singleton pregnancies 6 had PPCM.

DISCUSSION

In our study Ascites was noted in 16.3 % subjects(figure no. 1).

In the 11 year study conducted by Cong KJ and Weng TT (Complication of Ascites in pregnancy induced hypertension) in 1994 comprising 23 pregnancy induced hypertension patient with ascites reported that incidence of ascites in severe PIH was 21.6 / 1000 during antenatal period²².

In study conducted by Vijayanath et al (massive ascites in severe preeclampsia: a rare complication) in 2002 reported that incidence of ascites was 8 in 1000 patients²³.

In our study 71.4% (5 cases) of PPCM patients had ascites (figure no. 2).

Chi square = 14.18 with p value of 0.002. So there is significant correlation between ascites and PPCM.

In our study 63.2% (120 cases) were primiparous and 36.8 % (70 cases) were multiparous. Hypertensive disorders of pregnancy are more common in primiparous (figure no.3).

In our study 71.4% (5 cases) PPCM patients were primiparous and 28.6% (2 cases) were multiparous (figure no.4).

Chi square = 0.21 and p value is 1.0. There is no significant correlation of PPCM with parity.

A study conducted by Yang et al peripartum cardiomyopathy - report of 16 cases of PPCM in 2002 in which 27 % cases were multiparous.

Another study conducted by Shani et al in 2008 PPCM – risk factor and prognosis 36 % PPCM patients were multiparous.

A prospective study conducted by GS Prasad et al on peripartum cardiomyopathy from January 2006 to December 2012 stated that 56 % case study were primigravidae²⁴.

In our study 6.3% (12 cases) pregnancies were twin pregnancy and 14.3% (1 case) PPCM subjects had twin pregnancy (figure no. 5).

Chi square is 0.78 with p value 0.371. The number of cases of multifetal pregnancy in our study were not sufficient in both the groups i.e. PPCM and Non PPCM group and we did not get a significant difference in the incidence.

A study conducted by Chapa et al in prognostic value of echocardiography in peripartum cardiomyopathy in 2005 13% of PPCM patient had twin pregnancy²⁵.

Other study was conducted by Elkayam et al pregnancy associated cardiomyopathy: clinical characteristics and a comparison between early and late presentation in 2005, 13% patients had twin pregnancy².

Other case and control study conducted by Hawani et al in Hasan sadikin hospital Bandung Indonesia from 1st Jan 2011 until 31 Dec 2014 multiple fetal pregnancy in PPCM patients were 8.3%.

CONCLUSION

Out of 190 selected subjects, seven cases were diagnosed with PPCM. We found a significant correlation between PPCM and ascites i.e. ascites was more common in PPCM patients than in non PPCM patients. There was no correlation with multiparity and twin gestation between cases with cardiomyopathy and those without cardiomyopathy. In support of our study there are no previous studies available at present. Various studies have been done to implicate preeclampsia as a risk factor for PPCM. Similar studies can be conducted at other health institute to obtain more data regarding this subject.

REFERENCES

1. Nelson-Piercy C. Heart disease in pregnancy. In: Edmonds DK, editor. Dewhurst's Textbook of Obstetrics And Gynaecology. 7th ed. UK: Oxford, Blackwell Science Ltd;

2007. pp. 236–45.
2. Hilfiger-Kleiner D, Sliwa K, Drexler H. Peripartum cardiomyopathy: Recent insights in its pathophysiology. *Trends Cardiovasc Med*. 2008;18:173–9.
3. Sovndal S, Tabas JA. Cardiovascular disorders in pregnancy. In: Pearlman MD, Tintinalli JE, Dyne PL, editors. *Obstetric and Gynecologic Emergencies: Diagnosis and Management*. 1st ed. New York: McGraw-Hill Medical Publishing Division; 2004. pp. 300–9.
4. Tidswell M. Peripartum cardiomyopathy. *Crit Care Clin*. 2004;20:777–88.
5. Sliwa K, Skudieck 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;35:701–5.
6. Pandit V, Shetty S, Kumar A et al; Incidence and outcome of peripartum Cardiomyopathy from a tertiary hospital in South India. *Trop Doct* 2009;39:168–9.
7. Sanderson JE, Adesanya CO, Anjorin FI, Parry EH. Postpartum cardiac failure: Heart failure due to volume overload? *Am Heart J*. 1979;97:613–21.
8. Davidson NM, Parry EH. Peri-partum cardiac failure. *Q J Med*. 1978;47:431–61.
9. Sliwa K, Fett J, Elkayam U. Peripartum cardiomyopathy. *Lancet*. 2006;368:687–93.
10. Desai D, Moodley J, Naidoo D. Peripartum cardiomyopathy: Experiences at King Edward VIII Hospital, Durban, South Africa and a review of the literature. *Trop Doct*. 1995;25:118–23.
11. Mielniczuk LM, Williams K, Davis DR, Tang AS, Lemery R, Green MS, et al. Frequency of peripartum cardiomyopathy. *Am J Cardiol*. 2006;97:1765–8.
12. Pearson GD, Veille JC, Rahimtoola S, Hsia J, Oakley CM, Hosenpud JD, 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;283:1183–8.
13. James PR. A review of peripartum cardiomyopathy. *Int J Clin Pract*. 2004;58:363–5.
14. Sliwa K, Fett J, Elkayam U. Peripartum cardiomyopathy. *Lancet*. 2006;368:687–93.
15. Nelson-Piercy C. Cardiac disease. In: Luesley DM, Baker PN, editors. *Obstetrics and Gynaecology: An Evidence-Based Text For MRCOG*. 1st ed. London: Arnold; 2004. pp. 54–8.
16. Nelson-Piercy C. Heart disease in pregnancy. In: Edmonds DK, editor. *Dewhurst's Textbook of Obstetrics And Gynaecology*. 7th ed. UK: Oxford, Blackwell Science Ltd; 2007. pp. 236–45.
17. Hibbard J U, Lindheimer M, Lang R M. A modified definition for peripartum cardiomyopathy and prognosis based on echocardiography. *Obstet Gynecol*. 1999;4:311–5.
18. Johnson-Coyle L, Jensen L, Sobey A. American College of Cardiology Foundation, American Heart Association. Peripartum cardiomyopathy: Review and practice guidelines. *Am J Crit Care*. 2012;21:89–98.
19. Ramaraj R, Sorrell VL. Peripartum cardiomyopathy: Causes, diagnosis, and treatment. *Cleve Clin J Med*. 2009;76:289–96.
20. 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.
21. Elkayam U, Tummala PP, Rao K, Akhter MW, Karaalp IS, Wani OR, et al. Maternal and fetal outcomes of subsequent pregnancies in women with peripartum cardiomyopathy. *N Engl J Med*. 2001;344:1567–71.
22. Vaijyanath AM, Nayar B, Malhotra N, Deka D. massive ascites in severe preeclampsia: a rare complication. *J Obstet Gynaecol Res*. 2002;28:199–202.
23. Cong KJ, wang TT. Complication of ascites in pregnancy induced hypertension. *Zhonghua Fu Chan Ke Za Zhi*. 1994;29:7–9.
24. Prasad G S, Bhupali A, Prasad S, Patil A N., Deka Y. peripartum cardiomyopathy – case series.
25. Chapa JB, Heiberger HB, Weinert L, Decara J, Lang RM, Hibbard JU. Prognostic value of echocardiography in peripartum cardiomyopathy. *Obstet Gynecol*. 2005;105:1303–8.