

Echocardiographic evaluation of cardiovascular hemodynamic in preeclampsia

Shripad Vithalrao Dhanorkar¹, Balasaheb Eknathrao Karad^{2*}

¹Associate Professor, ²Professor and Head, Department of Medicine, Gouri Devi Institute of medical sciences, Rajbandh, Durgapur, West Bengal, INDIA.

Email: karadbalsasheb@gmail.com, svdhanorkar@gmail.com

Abstract

Background: Pregnancy is associated with volume overload producing significant vascular and hemodynamic adaptations in cardiovascular physiology. **Aims and Objectives:** To study Echocardiographic evaluation of cardiovascular hemodynamic in preeclampsia. **Methodology:** After approval from institutional ethical committee this cross-sectional study was carried out in the patients of Preeclampsia referred for the Cardiovascular hemodynamic at tertiary health care centre. All the patients with the written explained consent were enrolled for the study during the one year duration i.e. January 2016 to January 2017. During the one year 35 random patients with preeclampsia and 35 random patients with normal pregnancy were enrolled into the study. The statistical analysis done by unpaired t-test and calculated by SPSS 19 version software. **Result :** Systolic parameters were LV ESV (ml) -28.21 ± 1.7 and 32.02 ± 11.23 ($p > 0.92$), LV EDV (ml) -105.32 ± 3.32 and 110.12 ± 19.21 ($p > 0.80$), SV (ml) -69.51 ± 5.12 and 72.45 ± 9.21 ($p > 0.92$); CO (ml/min) -42.23 ± 3.41 and 70.23 ± 5.1 ($p > 0.001$) LVM Diastolic (g) -101.71 ± 24.52 and 137.11 ± 17.12 ($p > 0.01$), Systolic (g) -81.23 ± 19.34 and 91.52 ± 5.67 ($p > 0.02$), ARD (cm) -2.02 ± 0.115 and 3.12 ± 0.382 ($p > 0.01$), LVOT (cm) -1.62 ± 0.12 and 1.92 ± 0.12 ($p > 0.13$), TVR (dyne, Sec cm^5) -1195.12 ± 71.18 and 1387.80 ± 160.23 ($p > 0.01$) respectively in Normotensive and hypertensive. The diastolic parameters like E wave, m/s were 0.512 ± 0.112 and 1.823 ± 0.112 ($p > 0.01$), A Wave, m/s -0.612 ± 0.112 and 0.881 ± 0.121 ($p > 0.001$), E/A ratio -1.62 ± 0.132 and 1.9234 ± 0.571 ($p > 0.52$), E Dec time, ms -129 ± 9.12 and 195.21 ± 50.12 ($p > 0.05$), IVRT, ms -85.72 ± 6.12 and 98.11 ± 7.82 ($p > 0.05$), E VTI, ms -12.32 ± 1.94 and 98.11 ± 7.82 ($p > 0.05$), A VTI, ms -3.82 ± 0.72 and 7.12 ± 2.11 ($p > 0.05$). **Conclusion:** It can be concluded from our study that significantly there were changes in systolic and diastolic parameters of the heart of preeclampsia patients as compared to normal pregnancy hence only blood pressure monitoring is not sufficient it should be accompanied with 2 D echo for monitoring and intervention

Key Words: hemodynamic in preeclampsia, Eclampsia, Echocardiography.

* Address for Correspondence:

Dr. Balasaheb Eknathrao Karad, Professor, Department of Medicine, Gouri Devi Institute of medical sciences, Rajbandh, Durgapur, West Bengal, INDIA.

Email: svdhanorkar@gmail.com

Received Date: 19/01/2018 Revised Date: 15/02/2018 Accepted Date: 23/03/2018

DOI: <https://doi.org/10.26611/1021615>

| Access this article online | |
|---|--|
| Quick Response Code: | Website: www.medpulse.in |
|  | Accessed Date: 09 April 2018 |

INTRODUCTION

Pregnancy is associated with volume overload producing significant vascular and hemodynamic adaptations in

cardiovascular physiology.¹ They are necessary for successful pregnancy, but they impose further load on the heart. Moreover, heart disease is the leading cause of non-obstetric mortality during pregnancy, and the number of pregnant women at risk for cardiovascular complications is on the rise.^{2,3} Therefore, understanding of maternal cardiac function allows clinicians to detect and manage cardiac disease during early stages. In earlier days, gas exchange techniques were used to determine cardiac output. With development of cardiac catheterization, studies were performed using right heart hemodynamics. The advent of echocardiography enabled serial noninvasive cardiovascular monitoring throughout pregnancy. Initially, investigators used M-mode but perhaps due to its limitations, results were not uniform.⁴

Recently, Doppler echocardiography has been used which is reproducible and non-invasive technique suitable for pregnant women.⁵

MATERIAL AND METHODS

After approval from institutional ethical committee this cross-sectional study was carried out in the patients of Preeclampsia referred for the Cardiovascular hemodynamic at tertiary health care centre. All the patients with the written explained consent were enrolled for the study during the one year duration i.e. January 2016 to January 2017. During the one year 35 random patients with preeclampsia and 35 random patients with normal pregnancy were enrolled into the study. All the patients under gone 2-D echocardiographic evaluation thoroughly. The statistical analysis done by unpaired t-test and calculated by SPSS 19 version software.

RESULT

Table 1: Distribution of the patients as per the baseline parameters

| Parameter | Normotensive (n = 35) | Hypertensive (n = 35) | p-value |
|------------|-----------------------|-----------------------|---------|
| Age (Yrs.) | 27.32 ± 4.32 | 26.21 ± 5.21 | p>0.05 |
| BMI | 23.21 ± 2.32 | 24.52 ± 3.78 | p>0.05 |

The average age was 27.32 ± 4.32 and 26.21 ± 5.21 Yrs. and BMI was 23.21 ± 2.32 and 24.52 ± 3.78 which was comparable with each other (p>0.05).

Table 2: Distribution of the patients as per the systolic parameters

| Parameter | Normotensive (n = 35) | Hypertensive (n = 35) | P-Value |
|----------------------------------|-----------------------|-----------------------|---------|
| LV ESV (ml) | 28.21 ± 1.7 | 32.02 ± 11.23 | 0.92 |
| LV EDV (ml) | 105.32 ± 3.32 | 110.12 ± 19.21 | 0.80 |
| SV (ml) | 69.51 ± 5.12 | 72.45 ± 9.21 | 0.92 |
| CO (ml/min) | 42.23 ± 3.41 | 70.23 ± 5.1 | 0.001 |
| LVM Diastolic (g) | 101.71 ± 24.52 | 137.11 ± 17.12 | 0.01 |
| Systolic (g) | 81.23 ± 19.34 | 91.52 ± 5.67 | 0.02 |
| ARD (cm) | 2.02 ± 0.115 | 3.12 ± 0.382 | 0.01 |
| LVOT (cm) | 1.62 ± 0.12 | 1.92 ± 0.12 | 0.13 |
| TVR (dyne, Sec cm ⁵) | 1195.12 ± 71.18 | 1387.80 ± 160.23 | 0.01 |

Systolic parameters were LV ESV (ml) -28.21 ± 1.7 and 32.02 ± 11.23 (p>0.92), LV EDV (ml)-105.32 ± 3.32 and 110.12 ± 19.21 (p>0.80), SV (ml)-69.51 ± 5.12 and 72.45 ± 9.21 (p>0.92); CO (ml/min)-42.23 ± 3.41 and 70.23 ± 5.1 (p>0.001) LVM Diastolic (g)- 101.71 ± 24.52 and 137.11 ± 17.12 (p>0.01), Systolic (g)- 81.23 ± 19.34 and 91.52 ± 5.67 (p>0.02), ARD (cm)-2.02 ± 0.115 and 3.12 ± 0.382 (p>0.01), LVOT (cm)-1.62 ± 0.12 and 1.92 ± 0.12 (p>0.13), TVR (dyne, Sec cm⁵) - 1195.12 ± 71.18 and 1387.80 ± 160.23 (p>0.01) respectively in Normotensive and hypertensive.

Table 3: Distribution of the patients as per the diastolic parameters

| Parameter | Normotensive (n = 35) | Hypertensive (n = 35) | P value |
|----------------|-----------------------|-----------------------|---------|
| E wave, m/s | 0.512 ± 0.112 | 1.823 ± 0.112 | 0.01 |
| A Wave, m/s | 0.612 ± 0.112 | 0.881 ± 0.121 | 0.001 |
| E/A ratio | 1.62 ± 0.132 | 1.9234 ± 0.571 | 0.52 |
| E Dec time, ms | 129 ± 9.12 | 195.21 ± 50.12 | 0.05 |
| IVRT, ms | 85.72 ± 6.12 | 98.11 ± 7.82 | 0.05 |
| E VTI, ms | 12.32 ± 1.94 | 13.56 ± 5.12 | 0.72 |
| A VTI, ms | 3.82 ± 0.72 | 7.12 ± 2.11 | 0.05 |

The diastolic parameters like E wave, m/s were 0.512 ± 0.112 and 1.823 ± 0.112 (p>0.01), A Wave, m/s - 0.612 ± 0.112 and 0.881 ± 0.121 (p>0.001), E/A ratio-1.62 ± 0.132 and 1.9234 ± 0.571 (p>0.52), E Dec time, ms -129 ± 9.12 and 195.21 ± 50.12 (p>0.05), IVRT, ms-85.72 ± 6.12 and 98.11 ± 7.82 (p>0.05), E VTI, ms-12.32 ± 1.94 and 13.56 ± 5.12 (p>0.05), A VTI, ms-3.82 ± 0.72 and 7.12 ± 2.11 (p>0.05).

DISCUSSION

Pre-eclampsia is a multisystem disorder that occurs after 20 weeks of pregnancy. The incidence of pre-eclampsia in nulliparous women ranges from 3 to 10%⁶. Acute preeclampsia is associated with significantly higher prevalence of asymptomatic abnormal global left ventricular (LV) abnormal function/geometry and myocardial injury⁶. It is also associated with significantly higher risk of subsequent heart failure, ischaemic and hypertensive heart diseases, and related mortality compared with uneventful pregnancy in later life^{7,8}. The hemodynamics of preeclampsia is a subject of controversies. Cross-sectional studies of women with preeclampsia have revealed diverse hemodynamic findings such as elevated cardiac output^{9,10}, high vascular resistance and reduced cardiac output¹¹ and reduced myocardial contractility¹². In our study we have found that The average age was 27.32 ± 4.32 and 26.21 ± 5.21 Yrs. and BMI was 23.21 ± 2.32 and 24.52 ± 3.78 which was comparable with each other (p>0.05). Systolic parameters were LV ESV (ml) -28.21 ± 1.7 and 32.02 ± 11.23 (p>0.92), LV EDV (ml)-105.32 ± 3.32 and 110.12 ± 19.21 (p>0.80), SV (ml)-69.51 ± 5.12 and 72.45 ± 9.21 (p>0.92); CO (ml/min)-42.23 ± 3.41 and 70.23 ± 5.1 (p>0.001) LVM Diastolic (g)- 101.71 ± 24.52 and 137.11 ± 17.12 (p>0.01), Systolic (g)- 81.23 ± 19.34 and 91.52 ± 5.67 (p>0.02), ARD (cm)-2.02 ± 0.115 and 3.12 ± 0.382 (p>0.01), LVOT (cm)-1.62 ± 0.12 and 1.92 ± 0.12 (p>0.13), TVR (dyne, Sec cm⁵) - 1195.12 ± 71.18 and 1387.80 ± 160.23 (p>0.01) respectively in Normotensive and hypertensive. The diastolic parameters like E wave, m/s were 0.512 ± 0.112 and 1.823 ± 0.112 (p>0.01), A Wave, m/s - 0.612 ± 0.112

and 0.881 ± 0.121 ($p < 0.001$), E/A ratio- 1.62 ± 0.132 and 1.9234 ± 0.571 ($p > 0.52$), E Dec time, ms -129 ± 9.12 and 195.21 ± 50.12 ($p > 0.05$), IVRT, ms- 85.72 ± 6.12 and 98.11 ± 7.82 ($p > 0.05$), E VTI, ms- 12.32 ± 1.94 and 98.11 ± 7.82 ($p > 0.05$), A VTI, ms- 3.82 ± 0.72 and 7.12 ± 2.11 ($p > 0.05$). These findings are similar to Solanki Rizwana¹³ found parameters higher in pre-eclamptic subjects as compared to normotensive controls- mean cardiac output (66.85 ± 4.56 ml/min vs. 56.1 ± 1.77 ml/min); mean LV diastolic mass (131.15 ± 16.85 vs. 104.90 ± 23.17 g); systolic mass (88.5 ± 7.34 vs. 83.33 ± 23.84 g); total vascular resistance (1396.85 ± 150.2 vs. 1204.5 ± 71.182 dyne, s cm⁵). Women with preeclampsia delivered smaller babies (2410 ± 426.16 g) as compared to normotensive controls (2895 ± 276.20 g). Different mechanisms have been proposed to explain these changes. Nitric oxide (NO), the vascular smooth muscle relaxing substance acts through cGMP mediated vasodilatation. Enzyme endothelium derived nitric oxide synthase (eNOS) plays important role in its production. Biologically it is present in two isoforms i.e. Ca^{++} dependent and Ca^{++} independent. Increase in estradiol levels during pregnancy increases activity of Ca^{++} dependent eNOS.¹³ Also high levels of estrogen and progesterone metabolites (5- α dihydroprogesteron) during pregnancy increases arterial refractoriness to angiotensin II. Increased secretions of ANP by heart in response to atrial distention contribute to vasodilatation which is also mediated by cGMP.¹⁴ In normal pregnancy vascular relaxation in peripheral artery and enhanced arterial compliance in conduit arteries plays crucial role in accomodating increased intravascular volume without increase in blood pressure.¹⁵

CONCLUSION

It can be concluded from our study that significantly there were changes in systolic and diastolic parameters of the heart of preeclampsia patients as compared to normal pregnancy hence only blood pressure monitoring is not sufficient it should be accompanied with 2 D echo for monitoring and intervention.

REFERENCES

- Schrier RW. Pathogenesis of sodium and water retention in high output and low output cardiac failure, nephrotic syndrome and pregnancy. *N Engl J Med.* 1989; 319:1127-34.
- Presbitero P, Boccuzzi GG, Groot CJM, RoosHesselink JW. ESC textbook of cardiovascular medicine. Oxford: Oxford University Press. 2009.
- Siu SC, Sermer M, Colman JM, Alvarez AN, Mercier LA, Morton BC et al. Prospective multicenter study of pregnancy outcomes in women with heart disease. *Circulation.* 2001; 104:515–21.
- Vered Z, Poler SM, Gibson P, Wlody D, Wrez J. Noninvasive Detection of the morphologic and hemodynamic changes during normal pregnancy. *Clin Cardiol.* 1991; 14:327-34.
- Robson SC, Hunter S, Boys RJ and Dunlop W. Serial study of factors influencing changes in cardiac output during human pregnancy. *Am J Physiol.* 1989; 256:1060-5.
- Melchiorre K, Sutherland GR, Baltabaeva A, et al. Maternal cardiac dysfunction and remodeling in women with preeclampsia at term. *Hypertension.* 2011; 57:85–93.
- Lykke JA, Langhoff-Roos J, Sibai BM, et al. Hypertensive pregnancy disorders and subsequent cardiovascular morbidity and type 2 diabetes mellitus in the mother. *Hypertension.* 2009; 53: 944–51.
- Mongraw-Chaffin ML, Cirillo PM, Cohn BA. Preeclampsia and cardiovascular disease death: prospective evidence from the child health and development studies cohort. *Hypertension.* 2010; 56: 166–71.
- Easterling TR, Benedetti TJ, Schmucker BC, et al. Maternal hemodynamics in normal and pre-eclamptic pregnancies: a longitudinal study. *Obstet Gynecol.* 1990; 76(6):1061–9.
- Escudero EM, Favalaro LE, Moreira C, et al. Study of left ventricular function in pregnancy induced hypertension. *Clin Cardiol.* 1988; 11:329–33.
- Spinelli L, Ferro G, Nappi C, et al. Early diastolic time intervals during hypertensive pregnancy. *Clin Cardiol.* 1987; 10:567–72.
- Moran AM, Colan SD, Mauer MB, et al. Adaptive mechanisms of left ventricular diastolic function to the physiologic load of pregnancy. *Clin Cardiol.* 2002; 25:124–31.
- Weiner CP, Lizasoain I, Baylis SA, Knowles RG, Charles IG. Induction of calcium dependent nitric oxide synthases by sex hormone. *Proc Natl Acad Sci.* 1994; 91:5212-6.
- Cunningham FG, Leveno KJ, Bloom SL et al. In Maternal Physiology. Williams Obstetrics 22nd edn. Mc Graw-Hill. 2005; 129:135-6.
- Poppas A, Shroff SG, Korcarz CE, Hibbard JU. Serial assessment of cardiovascular system in normal pregnancy. *Circulation.* 1997; 95:2407-15.

Source of Support: None Declared
Conflict of Interest: None Declared