Original Research Article

Assessment of cardiovascular hemodynamics in gestational hypertension and preeclampsia

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Abstract

Preeclampsia is a multisystem disease complicating 5-10% of pregnancies and remains in the top three causes of maternal morbidity and mortality globally. During pregnancy mean arterial pressure and vascular resistance decrease, while blood volume and basal metabolic rate increase resulting in increased cardiac output In hypertensive disorders of pregnancy there is currently no consensus on the systolic and diastolic parameters of cardiac function and the literature is conflicting regarding whether there is increased, decreased or any change in cardiac output. Women with a history of preeclampsia/eclampsia have approximately double the risk of early cardiac, cerebrovascular, and peripheral arterial disease, and cardiovascular mortality. This study was undertaken to evaluate cardiovascular hemodynamic alterations in hypertensive disorders of pregnancy in comparison with appropriately age, parity and gestational age matched control normotensive pregnancies. In women with preeclampsia cardiac work index and left ventricular mass index are increased as a result of increased workload on heart to maintain cardiac output against increased after load. Systolic function is well preserved. Diastolic function is reduced and those with global diastolic function are at increased risk of developing pulmonary edema. Advanced techniques like speckle tracking echocardiography can better identify those with compromised cardiovascular function.

Key words

BP, MAP, BSA, CWI, LVMI, PE, E wave, A wave, IVS, ILVPW, LVESD, LVEDD.

Introduction

Cardiac function in hypertensive disorders of pregnancy

In preeclampsia disturbances in cardiovascular system are related to increased cardiac after load caused by hypertension leading to reduced cardiac output. Cardiac preload affected by pathologically diminished hypervolemia of pregnancy. Extravasation of intravascular fluid into extracellular space compounded by decreased oncotic pressure from a low serum albumin concentration leading to pulmonary oedema [1].

A wide range of hemodynamic conditions have been described in preeclampsia with no agreement as to the overall cardiac function. There are studies that have reported that women with severe preeclampsia have lower cardiac outputs than healthy pregnant women [2, 3] and also studies that show preservation of left ventricular systolic function at term in women with preeclampsia [4].

The hypothesis that preeclampsia may be a hyperdynamic state prior to diagnosis is supported by studies by Easterling and Bosio [5, 6]. These studies demonstrate that there may be a greater rise in cardiac output prior to clinical manifestations of preeclampsia than the cardiac output rise that may occur in healthy women. The data describing the ventricular diastolic function in preeclampsia are contradictory [7, 8].

Preeclampsia and future cardiovascular risk

Many studies have found that women with a history of preeclampsia have a 2- to 4-fold increased risk of developing hypertension, coronary artery disease, or stroke and venous thromboembolism up to 14 years after the index pregnancy [9]; among women in their fifties, the risk of hypertension 25 years after pregnancy has been shown to be considerably higher (48.5%) for those who had preeclamptic pregnancies than for those who had normotensive pregnancies (22%) [10]. The increase in mortality from cardiovascular disease generally becomes evident 2 to 3 decades after pregnancy in women with a history of non-recurring preeclampsia, substantially later than women who have had preeclampsia more than once [11] dices suggest that left ventricular remodeling was an adaptive response to maintain myocardial contractility with preeclampsia at term. Systolic function is preserved in women with preeclampsia.

Aim and objectives

To study cardiovascular hemodynamic alterations in hypertensive disorders of pregnancy in comparison with appropriately matched control normotensive pregnancies.

Material and methods

This was a prospective case control study carried out in the Department of Obstetrics and Gynaecology, KGH during the period of November 2012-September 2014. 50 pregnant women admitted with the diagnosis of gestational hypertension/ preeclampsia/ eclampsia were recruited as cases by simple random sampling. An equal number of age ($\pm$2 years) and gestation ($\pm$1 week) matched healthy normotensive pregnant woman attending the antenatal OP during the study period constituted control group.

Inclusion criteria

- Gestational hypertension
  - systolic BP $\geq$ 140 mm Hg or diastolic BP $\geq$90 mm hg
  - no proteinuria
- Preeclampsia
  - systolic BP $\geq$ 140 mm Hg or diastolic BP $\geq$ 90 mmhg after 20 weeks
  - Proteinuria $\geq$ 300mg/24 hrs or $\geq$ 1+dipstick
- Eclampsia seizures that cannot be attributed to other causes in a woman with preeclampsia

Exclusion criteria

- Chronic hypertension (BP $\geq$ 140/90 mm Hg before pregnancy or diagnosed before 20 weeks of gestation)

- Heart disease
- Medical disorders like diabetes mellitus, Renal disease
- Moderate or severe anaemia
- Twin pregnancy

On admission cases were assessed clinically, appropriate biochemical tests were done, fetal well being was assessed. Controls were assessed clinically. The subjects were studied by standard 2-dimensional and doppler transthoracic echocardiography in the left lateral decubitus position and data acquired at end expiration from standard parasternal/ apical views. Cardiac indices were normalized for body surface area.

Heart remodeling: Chamber quantification and left ventricular geometric pattern were estimated using M-mode.

Left ventricular mass index: Left ventricular mass corrected for body surface area.

LV mass[g] = 1.04{[LVEDD+IVS+LVPW]^3 - LVEDD^3}

Systolic function: Derived from Simpsons' modified biplane method from apical 4-chamber and 2-chamber views.

Ejection fraction

EF= [LVEDD]^3 – [LVESD]^3/ [LVEDD]^3 X100%

Normal range 50-85%

Fractional shortening: % change in LV dimensions between systole and diastole.

FS = LVEDD-LVESD/ LVEDDX100%

Normal range 30-45%

Diastolic function: E-wave is the result of passive early diastolic LV filling [0.70 ± 0.16 m/s] % change in left ventricular volume between systole and diastole. A-wave represents active late diastolic LV filling due to LA contraction [0.72±0.18 m/s]. E: A ratio = 1.03±0.34

Diastolic dysfunction is indicated by decrease in E-wave, increase in A-wave size, reduced E: A ratio.

Calculation of echocardiographic indices

- Stroke volume (SV) was calculated as the product of aortic Doppler flow velocity time integral (VTI) and cross-sectional area of the left ventricle outflow tract (CSA).
- Cardiac output (CO) was obtained as the product of stroke volume and heart rate (HR) derived from ECG monitoring.
- Cardiac work (CW) was calculated using the formula: CW=CO x MAP.

Results

Mean arterial pressure was 118 mm of Hg in cases and 87 mm of Hg in controls. 50 (100%) of controls were in the range of 71-105. 41 (82%) of cases are in the range of 106-125. In 9 (18%) of cases MAP was of ≥126 as per **Table - 1**.

CWI was increased in cases compared to controls. Mean CWI in cases was 386 and in controls were 287. In 25 (50%) controls CWI was in the range of 191-290. In 26 (52%) cases and 25 (50%) of controls CWI was in the range of 291-390. In 24 (48%) of cases\ CWI was in the above normal range of more than 390 as per **Table - 2**.

Mean Inter ventricular septum thickness in cases was 1 and in controls was 0.8. Mean left ventricular posterior wall thickness in cases was 1 and in controls was 0.8. Both were increased in cases compared to controls as per **Table - 3**.

LVMI was increased in cases compared to controls. Mean LVMI in cases was 80 gm/m^2 and in controls was 70 gm/m^2 in 1 (2%) case and 14 (28%) of controls. LVMI was in the range of 56-65. In 33 (66%) cases and 27 (54%) controls LVMI was in the range of 66-75. In 11 (22%) cases and 9 (18%) controls LVMI was in the range of 76-85. In 5 (10%) cases LVMI was in the range of 86-95 as per **Table - 4**.

Systolic function was preserved in cases. Mean cardiac index in cases and controls was 3.3. Mean Ejection fraction in cases was 66 and in

controls was 68. Mean Fractional shortening in cases was 37 as per **Table – 5**.

In controls E wave was reduced in cases which were statistically not significant. Mean E wave velocity in cases was 0.80 m/s and in controls were 0.82 m/s. A wave is increased in cases which is statistically significant. Mean A wave velocity in cases was 0.65 m/s and in controls was 0.60 m/s. E/A ratio was reduced in cases which is statistically significant. Mean E/A ratio in cases were 1.23 and in controls is 1.34 as per **Table - 6**.

Table – 1: Distribution of mean arterial pressure.

Mean arterial pressure	71-105	106-125	≥126
Cases 118 (106-140)	_	41 (82%)	9 (18%)
Controls 87 (73-96)	50 (100%)		

Table – 2: Distribution of cardiac work index ($p<0.05$)
Normal value in non pregnant women is 157-307 mm of hg X lit/min/m². Normal value in pregnant women is 208-380 mm of hg X lit/min/m².

Cardiac work index (mm of hg X lit/min/m²)	191-290	291-390	391-490
Cases 386.7 (311-455)	---	26 (52%)	24 (48%)
Controls 287.5 (197-344)	25 (50%)	25 (50%)	---

Table – 3: Left ventricular remodeling parameters.

	Cases	Controls	
Inter ventricular septum thickness (cm)	0.96 (0.8-1.1)	0.8 (0.7-0.9)	P<0.05
Left ventricular posterior wall thickness (cm)	0.98 (0.8-1.1)	0.8 (0.7-0.9)	P<0.05

Table – 4: Distribution of left ventricular mass index. ($p<0.05$)
Normal value in non pregnant woman is 43-95 gm/m². Normal value in pregnant women is 66-100 gm/m².

LVMI (gm/m²)	56-65	66-75	76-85	86-95
Cases 74.5 (60-94)	1 (2%)	33 (66%)	11 (22%)	5 (10%)
Controls 70 (57-83)	14 (28%)	27 (54%)	9 (18%)	

Discussion

Left ventricular (LV) geometry and function were compared between non-pregnant controls (*n* = 12) and normotensive (*n* = 44) and preeclamptic (*n* =15) pregnant women using echocardiography. Their conclusions regarding cardiovascular function in preeclampsia compared to normotensive controls are: Systemic vascular resistance is significantly greater in

preeclamptic women. Cardiac index did not differ between normotensive and preeclamptic women. Cardiac work index is increased in preeclamptic women. LV mass is significantly greater in preeclampsia consistent with concentric hypertrophy. Systolic function is preserved in preeclampsia. In women with preeclampsia, the E wave velocity was greater than in normotensive pregnancy, Peak A wave velocity and duration were greater in preeclamptic women [12, 13]. One study was designed to evaluate cardiac function and remodeling in preeclampsia occurring at term. This was a prospective case– control study of 50 term preeclampsia and 50 normal pregnancies assessed by echocardiography and tissue Doppler analysis. The results of this study are Global diastolic dysfunction was observed more frequently in preeclampsia versus control pregnancies (40% versus 14%, P<0.007) [14, 15].

Table – 5: Systolic function parameters.

	Cases	**Controls**	
Cardiac index l/min/m2	3.3 (2.6-3.8)	3.3 (2.4-3.8)	P<0.05
Ejection fraction	66.6 (53-79)	67.9 (55-80)	P<0.05
Fractional shortening	37 (27-48)	36 (28-45)	P<0.05

Table – 6: Diastolic function parameters.

	Cases	**Controls**	
Peak early wave of mitral flow (m/s) E wave	0.80 (0.73-0.95)	0.82 (0.72-0.94)	P<0.05
Peak atrial wave of mitral flow (m/s) A wave	0.64 (0.55-0.76)	0.60 (0.55-0.67)	P<0.05
E/A ratio	1.23(1.04-1.43)	1.34 (1.25-1.50)	P<0.05

Present study was a preliminary study undertaken to evaluate cardiovascular function in hypertensive disorders of pregnancy in comparison with normotensive controls Variation in results of diastolic function in comparison with other studies who found diastolic dysfunction in significant proportion of cases might be due to inadequacy of conventional indices used in the present study to identify subtle changes in preeclampsia. Further studies can be carried out to evaluate cardiovascular function in hypertensive disorders of pregnancy, diastolic function in particular taking into consideration numerous other parameters and advanced techniques like speckle tracking echocardiography are extremely helpful. LV myocardial relaxation is paradoxically an energy-dependent process that results in a rapid decrease in LV pressure after the end of contraction and during early diastole. Hence, the process of myocardial relaxation is more vulnerable than contraction and is apparently compromised in both early-stage cardiovascular disorders and in PE. Evaluation of cardiovascular function can have significant clinical implications for peripartum intravascular volume management, because the women with global diastolic dysfunction are the ones most likely to sustain acute cardiopulmonary morbidity, most commonly from pulmonary edema. The latter may be better predicted by the early diastolic mitral wave velocity/average lateral and septal diastolic myocardial velocities ratio in patients with global diastolic dysfunction, because it is a better indirect index of pulmonary capillary wedge pressure than central venous pressure. The

study findings also demonstrate that PE is associated with heart remodeling and significant changes in cardiac function. Many of the altered Tissue doppler indices are known to be independently related to the long-term risk of cardiovascular morbidity in non-pregnant subjects. A better understanding of the relationship of these indices and subsequent morbidity in the context of pregnancy may provide an opportunity for early cardiovascular risk stratification and the introduction of prophylactic strategies.

Conclusion

Preeclampsia is a multisystem disease complicating 5-10% of pregnancies and remains in the top three causes of maternal morbidity and mortality globally. During pregnancy mean arterial pressure and vascular resistance decrease, while blood volume and basal metabolic rate increase resulting in increased cardiac output. In hypertensive disorders of pregnancy there is currently no consensus on the systolic and diastolic parameters of cardiac function and the literature is conflicting regarding whether there is increased, decreased or any change in cardiac output. Women with a history of preeclampsia/eclampsia have approximately double the risk of early cardiac, cerebrovascular, and peripheral arterial disease, and cardiovascular mortality. This study was undertaken to evaluate cardiovascular hemodynamic alterations in hypertensive disorders of pregnancy in comparison with appropriately age, parity and gestational age matched control normotensive pregnancies. In women with preeclampsia cardiac work index and left ventricular mass index are increased as a result of increased workload on heart to maintain cardiac output against increased after load. Systolic function is well preserved. Diastolic function is reduced and those with global diastolic function are at increased risk of developing pulmonary edema. Advanced techniques like speckle tracking echocardiography can better identify those with compromised cardiovascular function.

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