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RESEARCH ARTICLE

HEMODYNAMICS IN PRIMIGRAVIDA WITH PRE-ECLAMPSIA

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ABSTRACT

Objective: To determine the hemodynamic changes in primigravida with pre-eclampsia.
Study design: Prospective cross-sectional study.
Methodology: Forty-four primigravida women with pre-eclampsia and 44 normal pregnant (NP) woman matched for gestational age were recruited. Cross-sectional array echocardiographic Doppler system were used to obtain the hemodynamic measurements. The height, weight and blood pressure too were recorded. Results are expressed as the mean and standard error (SE) of the mean. Students 't' test, Pearson's correlation and regression analysis were used to analyze the data. A probability of < 0.05 was considered as significant.
Results: The women in the pre-eclampsia and NP group had comparable clinical characteristics with respect to age (27.2 [0.83] / 26.7 [0.71] years), weight and height at the respective values in the NP were 120/75 (1.9/0.71), 81 (1.4), 21.38 (0.34) and 3.93 (0.07). The stroke volume (SV) was significantly lower (77.57 (2.61) ml) in pre-eclampsia than in NP (84.26 [1.92/ml]. The cardiac output in subjects with pre-eclampsia too (6.04 [0.23] l /min] was lower than in NP (6.79 [0.16] l / min). The mean total peripheral vascular resistance (TPVR) in the pre-eclampsia was 1625 (88.9) and in the NP was 1003 (26.6). In pre-eclampsia, TPVR showed a significant negative correlation with cardiac output (-0.83), SV (-0.56) and AD (-0.64). However, no significant correlation was observed with flow velocity integral (-0.17).
Comments: It is important to know the correct hemodynamic state of a pre-eclampsia patients because it may have important therapeutic implications. The untreated pre-eclampsia is characterized by a reduced circulation volume, low cardiac output and high peripheral resistance.

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INTRODUCTION

Hemodynamics is the relationship between the motion of blood and the factors that influence that motion. In normal pregnancy there is an increase in cardiac output which is 30% greater than the non-pregnant value. This is associated with a fall in the blood pressure during the first and second trimester of the pregnancy and a fall in the total peripheral vascular resistance, (Duvekot and Peeters, 1994). The hemodynamics profile of a women with pre-eclampsia is variable. Although the blood pressure is increased, the reported cardiac output is variable. It appears that severe pre-eclampsia has a hemodynamic expression that varies between the extremes of a high output low resistance and a low output high resistance state (Dennis *et al*., 2012). However, no data hemodynamics in untreated primigravida with pre-eclampsia of varying, severity with matched control group is available. The hemodynamic data is important to understand the pathophysiology of the disease and rational basis for treatment of pre-eclampsia, (Visser and Wallenburg, 1995). In view of the risk and complications, the use of Direct Fick and pulmonary artery catheter methods to estimate the cardiac output is only limited to women with severe pre-eclampsia. Their use is not justified in normal pregnancy. A combination of echocardiography and Doppler ultrasound is a reliable non-invasive test for estimation of cardiac output and this technique had been thoroughly validated use in pregnancy, (Robson *et al*., 1987). The aims were to study the hemodynamics in a group of untreated primigravida with varying disease severity of pre-eclampsia and compare the results with a group of matched normotensive primigravida. Secondly, the hemodynamic measurements were compared with laboratory indices of disease severity.

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MATERIALS AND METHODS

Forty-four primigravida women with pre-eclampsia were recruited for the study. None received fluids, antihypertensives or anticonvulsant medication. Pre-eclampsia was diagnosed if the diastolic blood pressure was >90mmHg. On 2 occasions 24 hours apart having been within normal limits prior to 20 weeks

gestation. Proteinuria was a quantified on a 24 hour collection and considered to be significant if more than 0.5g protein were excreted. If a 24 hour collection was not available, then more than 1+ on stick testing of the urine was considered to be clinically significant. Fourty-four normotensive healthy primigravida matched for maternal age (± 2 years) and gestational age (± week) at the time of measurements were selected from the antenatal clinic.

All investigations were performed in a warm quiet room in the left semilateral position after a minimum of 15 minutes rest. Heart rate was measurement every 5 minutes and investigation commenced when 3 consecutive measurements were ± beats/min. Blood pressure was measured by a standard mercury sphygmo-manometer. Diastolic blood pressure was taken as Korot Koff IV. Cross-sectional phased array Doppler system was used to obtain all ECG measurements. Measurement of cardiac output was performed using the method described previously, (Robson *et al*., 1987). Briefly, the system utilized a 3.5 MHz phased array and pulsed Doppler transducer and a 2.5 MHz continuous wave transducer. The flow velocity integral (FVI) at the aortic root was recorded at the suprasternal notch with continuous wave Doppler on a freeze frame. Diameter at the aortic root was measured from cross-sectional ECG using a freeze-frame facility and calipers. The systolic velocity integral (VI) and aortic diameter were measured at systole on 3 consecutive cycles and the average was taken as the final. The heart rate was obtained from the ECG.

The stroke volume (SV), aortic cross-sectional area (CSA), cardiac output, mean arterial pressure (MAP) and total peripheral vascular resistance (TPVR) were calculated with formula indicated below.

Results are expressed as the mean and standard error of the mean (SE). The statistical tests carried were Student's 't' test having established that the data were normally distributed, Pearson's correlation and regression analysis. A probability of < 0.05 was considered as significant.

- Stroke volume (ml) = CSA (cm^2) x FVI (cm^2)
- Cross-sectional area (cm^2) = 11 $(0/2)^2$
- Cardiac output (ml/min) = 0.07850^2 x FVI x HR
- Mean arterial pressure (MAP) = (SBP + 2 x DBP) / 3
- TPVR (dyne/s/cm^5) = MAP x 80/cardiac output

RESULTS

Demographic data are shown in Table 1. The pre-eclampsia clinic characteristics with respect to age (27.2 [0.83] vs. 26.7 - 0.71] years), weight and height at booking (Table 1). The hemodynamic results are shown in table 2. Though the heart rate, VI and aortic area were all lower in the patients with pre-eclampsia, none of them reached statistical significance. However, the SV in pre-eclampsia were lower than in normotensive subjects (77.57 [2.61] vs. 84.26 [1.92] ml; p = 0.042). In pre-eclamptics, the cardiac output was also lower (6.04 [0.23] vs 6.79 [0.160 l/min; p = 0.011). The total peripheral vascular resistance (TPVR) in pre-eclampsia was higher (1625 [88.9] vs 1003 [26.6]; p = 0.000]. There was a significant correlation between cardiac output with diastolic blood pressure. TPVR, platelet count and birth weight. The arbitrary division of the pre-eclampsia group into subjects with a blood pressure exceeding 100mmHg and < 100mmHg showed a lower cardiac output (5.50 [0.33] vs 6.54 (0.290; p = 0.02) and higher TPVR (1887 [169] vs 1449 [48]; p = 0.02) in subjects with a diastolic blood pressure exceeding 100mmHg.

Table 1. Demographic details

Variable	Pre-eclamptic mean (SE)	Normotensive mean (SE)
Age (years)	27.2 (0.83)	26.7 (0.71)
Weight (kg)	63. (1.6)	62.5 (1.1)
Height (cm)	161 (0.9)	163 (0.7)
Gestational age (days)	250 (3.2)	255 (4.2)
Delivery gestation (days)	260 (2.1)	279 (1.1)
Birth weight (kg)	2.413 (0.821)	3.428 (0.494)
24 hour proteinuria (g/dl)	2.87 (0.69)	Nil
Platelet count (mm^3)	187 (10.3)	200 (12.5)
Urate (mmol/l)	0.45 (0.01)	0.40 (0.01)

Table 2. Hemodynamic Results

Variable mean (SE)	Pre-eclamptic Mean (SE)	Normotensive	p value	Significance
Heart rate (bpm)	78 (2.0)	81 (1.4)	0.19	NS
Systolic velicity integral (cm)	20.56 (0.45)	21.38 (0.34)	0.15	NS
Aortic area (cm)	3.77 (0.08)	3.93 (0.07)	0.14	NS
Stroke volume (ml)	77.57 (2.61)	84.26 (1.92)	0.04	SG
Cardiac output (l/ml)	6.04 (0.23)	6.79 (0.16)	0.01	SG
Systolic pressure (mmHg)	152 (1.9)	114 (1.4)	0	SG
Diastolic pressure (mmHg)	100 (1.1)	70 (1.7)	0	SG
Peripheral vascular resistance	1625 (89)	1003 (27)	0	SG

bpm = beats per minute; SG = significant; NS = Non-significant

Comments

The results of cardiac output in normotensive group are comparable to those previously reported. The SV and cardiac output observed in patients with pre-eclampsia were significantly lower than in normotensive primigravida. It is important to note that there was no significant difference in the weight of subjects in the 2 groups because cardiac output is closely related to body weight (Oppen *et al*., 1995). Results to this study are compatible with that reported by Khalil *et al*., 2014; Visser, 1991and Belfort *et al*., 1986), Easterling *et al*, (1979) in their longitudinal study, where Doppler technique was used reported an elevated cardiac output in patients with pre-eclampsia.

However, there were only 9 pre-eclamptic subjects who completed the study when compared to 89 in the control group. Majority of the women did not have severe disease. Though Lim and Walters (Lim *et al*., 1990 reported similar results in a group of primigravida and multigravida with mild

hypertension no mention was made to the presence of proteinuria. In 10 severe pre-eclamptics, (Benedetti *et al*., 1980), reported a high cardiac output than in normotensives. However, the studies have been performed during labour, delivery and early puerperium. Phelan *et al* (1982) too reported similar results but some of the patients received medications such as hydralazine and magnesium sulphate. Cotton *et al*, (1988), in 45 women with severe pre-eclampsia reported that the SV and cardiac output were not appreciably different to the values reported for normal pregnancy. However, there was no control group and few of the subjects were either in labour or received intravenous fluids and magnesium sulphate. In a similar study by Mabie *et al*. (1989), the results were as above.

Pre-eclampsia poses a threat to both the mother and the fetus. As demonstrated in the study, high resistance and low cardiac output is associated with an increased incidence of intrauterine growth retardation, (Easterling *et al*., 1991). The only effective treatment is delivery. However, for different reasons, antihypertensive treatment is instituted to protect the mother and prolong the pregnancy. How will the 'low output high resistance circulation' detected in the study respond hemodynamically to anti-hypertensive vasodilator treatment? As detected in the study in severe pre-eclampsia the reduction of cardiac output is greater than and also appears to be a more intensely vasoconstricted state than in mild pre-eclampsia.[16] It is not clear whether plasma volume contraction and decreased cardiac output is as a result of vasoconstriction or whether vasoconstriction is due to insufficient physiological expansion of plasma volume and low cardiac output. If the decrease of cardiac output is the primary event, the increase of peripheral vascular resistance may be secondary or compensatory.

This may have important implications in the management as it may be necessary to restore the blood volume and cardiac output before attempting to reduce blood pressure. Instead a reduction in vascular resistance alone may further reduce cardiac output resulting in a decrease in renal and uterine blood flow. Therefore, volume expansion prior to or with the antihypertensive drugs may be beneficial because this will increase the cardiac output while reducing the blood pressure and vascular resistance, without adversely affecting uteroplacental or umbilical blood flow (Belfort *et al*., 1994). (Belfort *et al*., 1989 and Visser *et al*., 1991) justified this approach. However, caution must be exercised in the administration of intravenous fluids and this should be done only by personnel with an understanding of underlying hemodynamics and access to monitoring facilities to prevent pulmonary edema.

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