Assessment of cardiac hemodynamic changes during pregnancy in normal and hypertensive women

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ABSTRACT

Background: The hypertensive disorders of pregnancy (HDP) are the most common complication of pregnancy and are major cause of maternal and perinatal morbidity and mortality. Hypertension in pregnancy complicates about 10 % of all pregnancies worldwide.

Objective: This study aims to compare the hemodynamics of healthy pregnant women with hemodynamics of pregnant women with gestational hypertension (GH) or preeclampsia (PE).

Methods: A total of (120) women were included in this study; their ages range from (17 to 42) years; classified as follows: (60) normotensive pregnant women and (60) pregnant women with GH or PE. 2nd group is subdivided into: (30) hypertensive pregnant women who take antihypertensive treatment (methyldopa) and (30) those without antihypertensive medications. All women subjected to echocardiographic examination by experience specialist.

Results: Compared with healthy pregnant women, untreated pregnant women with GH or PE were associated with increase mean arterial pressure (MAP) (82.81±9.083 mm Hg vs. 113.66±7.327 mm Hg, p < 0.001), increase total peripheral vascular resistance (TPR) (1379.01±425.65 dyn.sec.cm-5 vs. 1733.99 ± 396.97 dyn.sec.cm-5, p<0.001), increased cardiac output (CO) (5.01 ± 1.100 L / min vs. 5.55 ±1.42 L/min, p = 0.04), increased ejection fraction (EF) (64.9 ± 4.9 % vs. 67.4± 6.561%, p=0.04) and fractional shortening (FS) (34.76±3.492% vs. 37.34±4.21%, p = 0.003), increased left ventricular mass (LVM) (136.41±29.22 gm vs. 174.16±41.04 gm, p<0.001) and decreased ratio of peak velocity of early transmitral flow to late transmitral flow (E/A ratio) (1.39±0.166 vs. 1.30±0.19, p=0.02).

The hypertensive pregnant patients treated with methyldopa; compared to non-treated group; showed a significant decreased in MAP (99.91±13.874 mm Hg vs. 113.66±7.325 mm Hg, p< 0.001, decreased TPR (1534.58 ± 349.47 dyn.sec.cm-5 vs. 1733.99 ± 396.97 dyn.sec.cm-5, p= 0.04), decreased EF (63.7±4.621% vs. 67.4±6.561%, p=0.014), decreased LVM (153.087±33.778 gm vs. 174.16±41.04 gm, p=0.03) and significant increase in E/A ratio (1.444±0.262 vs. 1.306±0.190, p=0.02).

Conclusion: Pregnant women with PE or GH have evidence of hemodynamic changes that justify routine echocardiographic assessment even in the absence of cardiac symptoms.

Keywords*:* Echocardiography, hypertension, pregnancy.

تقييم حركية الدم القلبية لدى النساء الحوامل واللاتي يعانين من ارتفاع ضغط الدم خلال الحمل

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الخلاصة

الخلفية: إضطرابات إرتفاع ضغط الدم بسبب الحمل هي المضاعفات الأكثر شيوعا خلال الحمل، وهي السبب الرئيسي لمرض ووفيات الأمهات والأطفال خلال الفترة المحيطة بالولادة. إرتفاع ضغط الدم في الحمل يشكل حوالي ١٠% من جميع حالات الحمل في جميع أنحاء العالم.

الهدف: هذه الدراسة تهدف الى مقارنة حركية الدم للنساء الحوامل الأصحاء مع حركية الدم للنساء الحوامل المصابات بإرتفاع ضغط الدم الحملي أو تسمم الحمل.

طرق العمل: تم تضمين ما مجموعة (١٢٠) إمرأة في هذه الدراسة. وتتراوح أعمارهم بين (١٧ و ٤٢) سنة ؛ تصنف على النحو التالي: (٦٠) من النساء الحوامل ذوات ضغط دم طبيعي و (٦٠) امرأة حامل مع إرتفاع ضغط الدم الحملي (GH) أو تسمم الحمل (PE) وتنقسم هذه المجموعة إلى: (٣٠) من النساء الحوامل المصابات بإرتفاع ضغط الدم اللواتي يتناولن علاج إرتفاع ضغط الدم (ميثيل دوبا) و (٣٠) بدون دواء خافض للضغط. و خضعت جميع النساء لفحص تخطيط صدى القلب من قبل طبيب مختص.

النتائج : مقارنة مع النساء الحوامل الأصحاء، إرتبطت النساء الحوامل غير المعالجات المصابات بـإرتفاع ضغط الدم الحملي أو تسمم الحمل بزيادة متوسط ضغط الدم الشرياني (9.083 ± 82.81 ملم زئبق مقابل 7.327 ± 113.66 ملم زئبق،0.001 > p)، زيادة إجمالي المقاومة المحيطية للأوعية الدموية (425.65 ± 1379.01 داين.ثا.سم-5 مقابل396.97 ± 1733.99 داين.ثا.سم-5،0.001> p)، زيادة معدل النتاج القلبي (1.10 ± 5.01 لتر/دقيقة مقابل 1.42 ± 5.55 لتر/دقيقة ، p = 0.04)، زيادة معدل الكسر القذفي أو الجزء المقذوف للبطين الأيسر (4.95± 64.9٪ مقابل 6.51 ± 67.4٪ ، p = 0.04) والكسر التقصري (3.492 ±34.76 مقابل 6.561±37.34٪،p=0.003)، زيادة كتلة البطين الأيسر (29.22 ± 136.41 جرامًا مقابل 41.04 ± 174.16 جرام،0.001> p) وإنخفاض نسبة الامتلاء الانبساطي المبكر/ الامتلاء الانبساطي المتأخر (0.166±1.39 مقابل 0.19 ± 1.30،p = 0.02).

المريضات الحوامل ذوات إرتفاع ضغط الدم اللاتي يتعالجن بميثيل دوبا; مقارنة بالمجموعة غير المعالجة أظهرن إنخفاض معنوي في متوسط ضغط الدم الشرياني (13.874 ± 99.91 ملم زئبق مقابل 7.325 ± 113.66 ملم زئبق،0.001> p)، انخفاض في إجمالي المقاومة المحيطية للأوعية الدموية (396.97 ± 1733.99 داين.ثا.سم- 5 مقابل 349.47 ± 1534.58 داين.ثا.سم- 5 ،0.04 = p)، إنخفاض معدل الكسر القذفي (p = 0.014 , % 6.561 ± 67.4 vs. 4.621% ± 63.7) ، انخفاض في كتلة البطين الأيسر (41.04 ± 174.16 جرام مقابل 33.778 ± 153.087 جرام،0.03 = p) وزيادة معنوية في نسبة الامتلاء الانبساطي المبكر/ الامتلاء الانبساطي المتأخر (0.262 ± 1.444 مقابل 0.190 ± 1.306، 0.02 = p).

الاستنتاج: اظهرت النساء الحوامل المصابات بـإرتفاع ضغط الدم الحملي أو تسمم الحمل تغييرات واضحة في حركية الدم والتي تستدعي إجراء فحص صدى قلب روتيني حتى عند غياب الأعراض القلبية.

INTRODUCTION

The hypertensive disorders of pregnancy (HDP) are the most common complication of pregnancy and are major cause of maternal and perinatal morbidity and mortality [1]. Hypertension (HT) in pregnancy complicates about 10 % of all pregnancies worldwide [2]. General classification of HDP (according to ACOG):

- Gestational or transient hypertension.
- Preeclampsia or eclampsia.
- Chronic hypertension.
- Chronic hypertension with superimposed preeclampsia [2].

Gestational hypertension (GH); is hypertension (systolic blood pressure ≥ 140 mmHg and / or diastolic blood pressure ≥ 90 mmHg, on two occasions, taken at least 4 hours apart) without proteinuria or other signs of organ dysfunction, first appears after 20 weeks' gestation and resolves by 12 weeks postpartum[3]. From a management point of view, the final diagnosis can only be done after the pregnancy has been finished so, the assumption must be made that any woman who develops hypertension must be considered to be at risk of maternal and fetal complications[4].

Preeclampsia (PE) is defined as new onset of hypertension (systolic blood pressure ≥ 140 mmHg and / or diastolic blood pressure ≥ 90 mmHg, on two occasions, taken at least 4 hours apart) in the 2nd half of gestation in a woman with a previously normal blood pressure and may remain as late as 4 – 6 weeks postpartum , often accompanies by proteinuria or associated with other signs and symptoms including headache, visual disturbance, epigastric pain and the rapidly developing edema[2,3] .

Transthoracic echocardiography is often considered as the reference standard for cardiovascular (CV) system diagnosis, monitoring and research uses. It is valid, precise and reproducible measurement device providing information not only about cardiac output but also

about systolic and diastolic function, and structural and functional information[5]. It is the most widely used imaging modality for assessment of CV function during pregnancy[6].

MATERIALS AND METHODS

This is a case – control study, performed between November 2017 till April 2018 in the Echocardiography Unit at Al-Salam Teaching Hospital and Ibn altheer Teaching Hospital in Mosul city.

Subjects

After institutional ethics approval and informed consent, a total of 120 women were included in this study; their ages range from 17 to 42 years; classified as follows:

- group I, (60) normotensive pregnant women.
- group II, (60) pregnant women with GH or PE. This group is subdivided into: (30) hypertensive pregnant women who take antihypertensive treatment (methyldopa) and (30) those without antihypertensive medications.

  The following criteria of exclusion were considered: Significant medical and surgical illness, structural heart disease and accidental echo finding of valvular heart disease and congenital heart disease, smoker, moderate and severe anemia, diabetes mellitus, essential hypertension, drugs that increase blood pressure as β_2 agonists or taking other antihypertensive medication (other than methyldopa), less than 20 weeks of gestation or uncertain dates, multiple pregnancy, fetal anomalies, uterine and placental abnormalities, antepartum hemorrhage and starting labour.

After medical history and clinical examination, standing height and body weight were measured for each subject at the time of the study, and those values were used to calculate body mass index (BMI) and body surface area (BSA), arterial pulse rate was measured by palpating radial artery, electrocardiograph (ECG) was done and blood pressure were obtained from the brachial artery using a calibrated mercury sphygmomanometer in the sitting position after the women had rested for at least 10 minutes with arm supported at the level of the heart, recording the diastolic value as Korotkoff V was used according to the American Heart Association. The mean arterial pressure (MAP) was calculated from: diastolic pressure + 1/3 pulse pressure while total peripheral resistance (TPR) was calculated from: MAP×80/CO dynes.sec/cm[5].

Echocardiography

With the subjects resting in the left lateral decubitus position, standard two- dimensional, M-mode and Doppler transthoracic echocardiographic examinations were performed by experience echocardiographer according to ASE/EAE guidelines[7,8], using Sono ACE X6, Medison/Chorea and Logiq S6, GE Healthcare/USA machines. M-mode study was done at the level of mitral valve leaflet tips with M-mode line perpendicular to the long axis of the heart in the parasternal long axis view to measure: Left ventricular internal dimension at diastole (LVIDd) and systole (LVIDs). Interventricular septal thickness at diastole (IVSd) and posterior left ventricular wall thickness during diastole (PWTd). From which the following parameters were obtained: End diastolic volume (EDV) and end systolic volume (ESV) were calculated by the machine using the Teichholz formula and the difference between them is stroke volume (SV), cardiac output (CO) was obtained by multiplying SV by HR per minute. Ejection Fraction (EF) = (EDV – ESV) / EDV, and Fractional Shortening (FS)= (LVIDd – LVIDs)/ (LVIDd) ×100 were also obtained. Left ventricular mass (LVM) was calculated using the following formula: LVM (gram) = 0.8 × [1.04 (LVIDd + PWTd + IVSd) ^{3} – LVIDd^3] + 0.6. LV mass index (g/m^2) was obtained by dividing LV mass on the subject's BSA. Transmitral flow pattern (peak E wave velocity, peak A wave velocity and E/A ratio) were measured; using pulsed wave Doppler; obtained from the apical four chamber view.

Statistical Analysis

All data were analyzed using the Statistical Package for Social Science (SPSS) software version 21 and were expressed in terms of mean ± standard deviation. Unpaired independent t-test was used to compare between normotensive pregnant and non-treated pregnant with GH and PE, and between treated and non-treated

pregnants with GH and PE. Chi (X^2) test was used to test the association between categorical variables. Pearson's test; to test the correlation between BMI, LVM and MAP; was used. In all tests, P – value < 0.05 was considered to be statistically significant.

Results:

Maternal distributions according to age, parity and BMI

Regarding maternal age, a significant association exits with PE or GH. (40%) of the hypertensive pregnant women were above (35 years) which represent the highest percent, (36.7%) of hypertensive pregnant women aged between (26-35 years), the lowest percent (23.3%) was among those aged between (17 – 25 years).

BMI has significant association to PE or GH. Only 6 (10%) of hypertensive prgnants had normal weight, while 20 (33.3%) were obese class I, (26.7%) were obese class II, (23.3%) were overweight and (6.7%) were obese class III. (**Table 1).**

A significant association between parity and women with PE or GH. A (53.3%) of hypertensive pregnant women were grand multiparous, (26.7%) of them were multiparous and (20%) of women were primiparous.

Comparison between normotensive pregnant women and untreated PE or GH

Compared with normotensive group, untreated PE or GH group was significantly older (28.03 ± 6.48 years vs. 31.67 ± 8.691 years), higher BMI (28.12 ± 6.41 kg/m^2 vs. 33 ± 5.67 kg/m^2), higher BSA (1.733±0.19 vs. 1.89 ± 0.191 m^2), (**Table 2**).

MAP (113.66 ± 7.327 mm Hg vs. 82.81 ± 9.083 mm Hg), TPR (1733.99 ± 396.97 dyne.s.cm^{-5} vs. 1379.01 ± 425.65 dyne.s.cm^{-5}), SV (63.75±15.95 ml vs. 55.18±12.285 ml) and CO (5.55±1.42 L/min vs. 5.01±1.100 L/min) were significantly increased in untreated group compared to normotensive pregnant group (**Table 3**).

There were no significant change in LV dimensions and volumes. IVSd (1.03±0.18 cm) and PWTd (1.14±0.135 cm) are markedly higher in untreated PE or GH than that of normotensive pregnant group (0.918±0.162 cm, 1±0.139 cm, respectively) and so, LVM and LVMI were higher in the untreated group compared to normotensive pregnant women (174.16±41.04 gm, 92.25±20.02 gm/m^2 vs. 136.41±29.22 gm, 78.79±14.569 gm/m^2, respectively). EF (67.4±6.561% vs. 64.9 ±4.955 %) and FS (37.34±4.21% vs. 34.76±3.492%) were also significantly higher in untreated PE or GH than that of normotensive pregnant group (**Table 4**).

A mitral wave velocity is significantly increase (67.5±10 cm/sec vs 59.57±11.22 cm/sec) with significant decrease in E/A ratio (1.3±0.19 vs 1.394 ±0.166) were noticed in the untreated group (**Table 5**).

Table 1. Age, BMI and parity distributions among pregnant women.

Parameter	Category	Normotensive pregnant	PE or GH
Age	17-25	(36.7%) 22	(23.3%) 14
	26-35	(53.3%) 32	(36.7%) 22
	≥36	(10%) 6	(40%) 24
Total		(100%) 60	(100%) 60
chi=15.364	p-value=0.002		
BMI	Normal weight 18.5 - 24.9	(40%) 24	(10%) 6
	Over weight 25-29.9	(30%) 18	(23.3%) 14
	Obese class I 30-34.9	(6.7%) 4	(33.3%) 20
	Obese class II 35-39.9	(20%) 12	(26.7%) 16
	Obese class III >40	(3.3%) 2	(6.7%) 4
Total		(100%) 60	(100%) 60
chi=13.581	p-value=0.003		
Parity	0	(23.3%)14	(20%) 12
	1 - 4	(46.7%) 28	(26.7%) 16
	≥5	(30%) 18	(53.3%) 32
Total		(100%) 60	(100%) 60
chi=16.203	p-value=0.001		

The Chi-square test is significant at the 0.05 level.

Table 2. Comparison of baseline characteristics between normotensive pregnant women and untreated PE or GH.

Parameter	Normotensive pregnant (n=60)	Untreated PE or GH (n=30)	P– value
Age (years)	28.03 ± 6.481	31.67±8.691	0.02
Parity	3± 2.255	3.8 ± 3.158	NS
Gestational age (GA) (weeks)	27 ± 5.386	28.8 ± 5.827	NS
BMI (kg/m^2)	28.12±6.413	33 ± 5.67	<0.001
BSA (m^2)	1.73±0.190	1.89±0.191	<0.001

Table 3. Comparison of hemodynamic characteristics between normotensive pregnant women and untreated PE or GH.

Parameter	Normotensive pregnant (n=60)	Untreated PE or GH (n=30)	P value
MAP (mm Hg)	82.81 ± 9.083	113.66 ± 7.327	<0.001
PR (bpm)	92.56 ± 8.495	88.53 ± 13.505	NS
TPR ($dyne.s.cm^5$)	1379.01±425.65	1733.99 ± 396.97	<0.001
SV (ml)	55.18 ±12.285	63.75 ± 15.95	<0.01
CO (L/min)	5.01 ± 1.100	5.55 ± 1.42	0.04

Table 4. Comparison of echocardiographic parameters between normotensive pregnant women and untreated PE or GH.

Parameter	Normotensive pregnant (n=60)	Untreated PE or GH (n=30)	P value
LVIDd (cm)	4.32 ± 0.396	4.47 ± 0.499	NS
EDV (l)	85.186±17.826	92.96 ± 24.39	NS
LVIDs (cm)	2.79 ± 0.302	2.8 ± 0.414	NS
ESV (ml)	29.75 ± 7.749	32.46±14.526	NS
EF (%)	64.9 ± 4.9	67.4 ± 6.561	0.04
FS (%)	34.76 ± 3.492	37.34 ± 4.21	0.003
IVSd (cm)	0.918 ± 0.162	1.03 ± 0.18	0.003
PWTd (cm)	1 ± 0.139	1.14 ± 0.135	<0.001
LVM (g)	136.41 ± 29.22	174.16±41.04	<0.001
LVMI(g/m^2)	78.79 ± 14.569	92.25 ± 20.02	<0.001

Table 5. Comparison of Mitral flow velocities between normotensive pregnant women and untreated PE or GH.

Parameter	Normotensive pregnant (n=60)	Untreated PE or GH (n=30)	P-value
E wave velocity (cm/sec)	83.97±18.228	88.5±15.761	NS
A wave velocity (cm/sec)	59.57±11.221	67.5 ± 10	0.001
E / A ratio	1.39±0.166	1.30± 0.19	0.02

Comparison between treated and untreated PE or GH

Within the hypertensive group, comparisons were made between the treated and untreated patients (**Table 6,7**). Regarding general characteristics, mean age, parity, GA and BMI were similar in both groups. In untreated hypertensive women, systolic BP (150.33±10.580 mm Hg), diastolic BP (95.33± 10.080 mm Hg) and MAP (113.66±7.325 mm Hg) were significantly higher than treated group were systolic BP was (129.166±16.870 mm Hg), diastolic BP (85.33±13.450 mm Hg) and MAP was (99.91±13.874 mm Hg), TPR is significantly decreased in the treated group (1733.99 ± 396.97 vs. 1534.58±349.47 $dyn.sec.cm^{-5}$). No significant changes in CO and SV between the groups.

LVM was higher in untreated group compared to treated one (174.16±41.04 vs. 153.087±33.778, respectively). A significant decrease in transmitral A velocity in treated group as compared to untreated one (59.533±9.964 vs 67.527±10, respectively) and in E/A ratio (1.444±0.262 vs. 1.306±0.190, respectively) with no significant change in transmitral E velocity.

Table 6. Comparison of baseline characteristics between treated and non-treated PE or GH.

Parameter	Non-treated patients (n=30)	Treated patients (n=30)	P- value
Age (years)	31.67±8.691	32.8 ± 8.002	NS
Parity	3.8 ± 3.158	4.8± 3.585	NS
GA (weeks)	28.8±5.827	30.87±6.516	NS
BMI (kg/m^2)	33 ± 5.67	31.847±5.969	NS
BSA (m^2)	1.89±0.191	1.84 ±0.138	NS

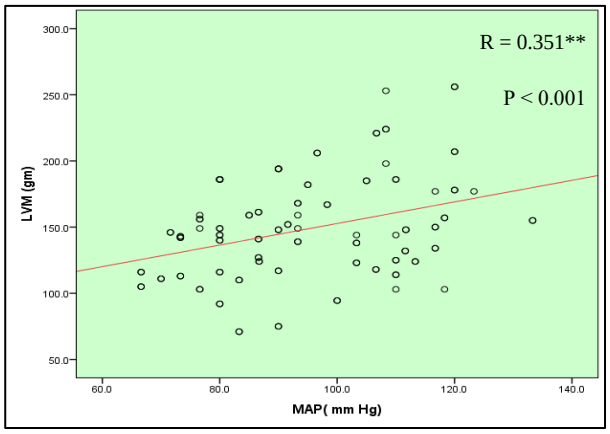

Figure 1. Correlation between LVM and MAP.

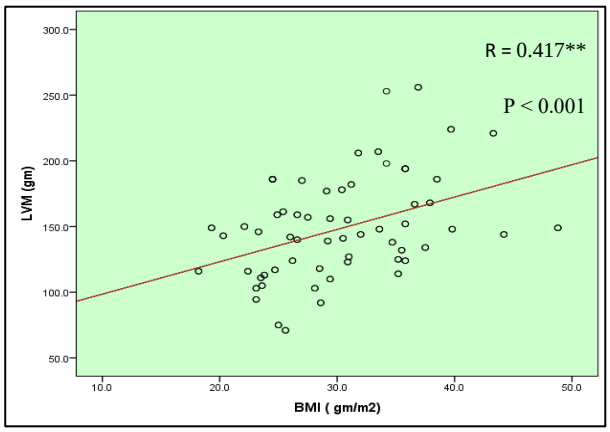

Figure 2. Correlation between LVM and BMI.

Table 7. Comparison of hemodynamic and echocardiographic parameters between treated and non-treated PE or GH.

Parameter	Non-treated patients (n=30)	Treated patients (n=30)	P–value
Systolic BP (mm Hg)	150.33± 10.580	129.166± 16.870	<0.001
Diastolic BP(mmHg)	95.33±10.080	85.33± 13.450	<0.001
Mean arterial pressure (mmHg)	113.66± 7.325	99.91± 13.874	<0.001
Pulse rate (beats per min)	88.53± 13.505	90.93 ± 6.658	NS
SV (ml)	63.75 ± 15.95	60.23± 13.372	NS
CO (L / min)	5.55 ± 1.42	5.40±1.141	NS
TPR (dyn.sec.cm^{-5})	1733.99± 396.97	1534.58±349.47	0.04
EF %	67.4 ± 6.561	63.7 ± 4.621	0.014
LVM (gm)	174.16± 41.04	153.087± 33.778	0.03
E wave velocity	88.51± 15.761	85.51± 14.196	NS
A wave velocity	67.527 ± 10	59.533± 9.964	0.003
E / A ratio	1.306 ± 0.190	1.444 ± 0.262	0.02

DISCUSSION

Hypertensive disorders of pregnancy, including GH and PE, remain the main cause of maternal morbidity and mortality in developing and developed countries. The hemodynamics of PE and GH are complex[9], and studies on left ventricular structures and function are not only scarce, but also controversial on many topics[10] There is a growing body of literature concerning the pathophysiology of preeclampsia which may be a heterogeneous disease with diverse hemodynamic subsets[9]. PE has been described variously as:

- a state of abnormally high CO and low systemic vascular resistance (SVR) .
- a state of abnormally low CO and high SVR.
- a state of abnormally high CO and high SVR[11].

This study showed that pregnant women with GH or PE were characterized by high CO and high TPR state and normal physiological changes in left ventricular structures and function were exaggerated in this group.

GH and PE have common risk factors[12]. By comparing age wise distribution between normal and hypertensive women, age has been found to have a significant effect on the incidence of HDP, with maternal age ≥ 35 years is important risk factor. About more than 1/3rd (40%) of the patient were above 35 years which also reported by Alrubaee, (2006)[13] and sheraz *et al*., (2006) who reported that PE is more common in patient younger than 21 years and older than 35 years, Duckitt *et al*., (2005) suggest that the increase placental villous reaction in women older than 30 years may contribute to PE development[14].

Not only primiparous are at high risk for developing PE but also the multiparous women, similar report was found by Alrubaee, (2006)[13].

High BMI is an important risk factor for PE and GH [13,15] that was confirmed by this study. The explanation may be an increased level of C-reactive protein, which is involved in the inflammatory process of HDP, is found in obese patients. In addition, some facts suggest that obesity augmented endothelial role and promoted systemic inflammatory reaction that linked to atherosclerosis which play a part in pregnancy induced hypertension[16].

Heart rate was not significantly changed in this study that is agree with others [17-19].

TPR was high in the hypertensive; as reported by other studies [10,18,19]; because systemic maternal arteries in women with PE show endothelial dysfunction that is characterized by endothelial injury, decreased dilatory responses, and imbalance in the bioavailability of endothelium-derived vasoactive substances [20].

There were no significant change in LV dimensions and volumes. These changes agree with other studies [10,17,21-25].

CO was significantly higher in untreated hypertensive group and this compatible with the proposed hyperdynamic model that is also reported by other studies [10,18,25].

The value of the finding a high CO in the group of untreated women lies not in the reality that because of the higher BMI in this group, but rather the enhancement in inotropy that was found in this group. In the usual situation, if someone is much larger than another person, then their CO is usually higher. The explanation for this, is that their heart is larger, their EDV are larger, consequently their SV are larger however, their FS and EF remain similar to the smaller individual. Dennis *et al* (2010) had demonstrated that the reason for the increased CO in women with untreated PE is an increase in inotropy not an increase in LV EDV [5,18].

LV EF is considered the most commonly reported measure of global LV function[26]. Significant increase observed in EF and FS between untreated hypertensive pregnant and normotensive pregnant groups as reported by Dennis *et al* (2010) [5,18] and Kim *et al* (2016) [17]; and this could be explained by increase cardiac inotropy and LVM, while others [21-25] suggested a non-significant changes. LV EF and FS are sensitive to altered loading condition, and this could explain this differences.

Mitral E (early) velocity was increased, this proposed that transmitral pressure gradient during early passive filling is greater and reflects alterations in passive cardiac muscles compliance in the hypertrophic ventricle, this non-significant difference in E wave velocity is found also in other studies[21,22]. A more(significant) increase in A (atrial) mitral velocity; and this is compatible with other results [21,22,25]; and decrease in E/A ratio as agree with others [21,22,25] was found in untreated hypertensive; give an opinion to the more role of atrial contraction in hypertrophied ventricular muscles of these pregnant.

IVSd, PWTd and thus the LVM and LVMI were significantly increased in hypertensive pregnant women that is agree with most studies [10,18,21-24]. **(Figure 1)**. The heart must adapt its wall thickness to this raise in pressure load in spite of the short lasting overload. The explanation of this is that, when the heart faces a hemodynamic load, it can work by following ways to compensate:

- Using Frank – Starling mechanism to increase cross bridges formation.
- Increase muscle mass to tolerate the extra load.
- Recruit neurohormonal mechanism as sympathetic or renin-angiotensin aldosterone system to augment contractility [27].

BMI is a significant factor for increased LVM, and there is a significant positive correlation between them as shown in **Figure 2**.

While treated vs. untreated PE or GH

As MAP depends on both CO and TPR, a reduction in BP could be achieved either by decreasing CO and/or TPR. Antihypertensive drugs benefit from these three mechanisms [28].

All of our treated patients were taking methyldopa only, a centrally acting antihypertensive drug that stimulate alpha adrenergic receptors in the brain stem inhibiting efferent sympathetic tone[28] , decreasing BP and TPR without significant changes on CO, PR and SV [29] and this is compatible with the results of this study.

Also treated women showed a reduction of LVM compared to untreated group and this effect of methyldopa confirmed clinically by other studies [30]. This is either due to hemodynamical effect by decreasing TPR or non-hemodynamically by a direct effect of methyldopa on decreasing cardiac muscle protein synthesis as shown by Pegram *et al.,* (2000) who mentioned that a short time period (three weeks in their study) treatment with methyldopa resulted in a reduction of LVM of spontaneously hypertensive rats [31].

A significant decrease in mitral A wave velocity and increase in E/A ratio reflects improvement in the LV compliance and thus diastolic performance in those treating with methyldopa[32].

CONCLUSION

- Transthoracic echocardiography considered a good non-invasive method for evaluation of hemodynamic during pregnancy.
- Monitoring of BP alone is not enough to identify risk of CV complications. Routine echo assessment for hypertensive women is needed to predict the risk for complications even before the appearance of symptoms.
- Women with PE or GH have increased CO, increased inotropy and increased TPR.
- Patients with PE or GH are associated with increased LVM. Heavy maternal weight may exaggerate this increase.
- Short lasting pressure overload in a previously young and healthy heart is able to make changes in LV.
- Women with PE or GH treated with methyldopa showed a significant decreased in MAP and TPR with improvement in the LV compliance and thus diastolic performance.

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