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PREGNANCY HYPERTENSION AND THE DIASTOLIC FUNCTION

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Abstract

Introduction: Heart's structural effects of pregnancy hypertension are not thoroughly investigated and yet are waiting to be studied here in Albania in the near future.

The aim of the study: To estimate and valuate the structural and functional effects in the heart due to pregnancy hypertension.

Methods and results: This study is prospective where the 106 individuals included are pregnant women in the third trimester of pregnancy, complicated by pregnancy hypertension. These patients are divided into three groups with: Chronic Hypertension, Pregnancy-induced Hypertension and Preeclampsia. The indexed measure of the left ventricle (LVMi) has been analyzed as well as its wall relative thickness (PWT). The form of geometric remodulation found more often within the three groups is eccentric hypertrophy and the diastolic dysfunction found within the three groups.

Conclusion: Pregnancy hypertension damages the diastolic functioning of the left ventricle and at the same time it influences its geometric remodeling. By estimating the geometry and diastolic functioning of the left ventricle, echocardiographicy can identify patients suffering from complications in the 3d trimester of pregnancy.

Key words: pregnancy hypertension, diastolic function, geometric remodeling

Introduction

Hypertension during pregnancy complicates about 2-3% of cases of pregnancy, which is estimated as one of the major causes of disease incidence, fetal and maternal mortality. Various clinical studies have shown that hypertension during pregnancy acts as a risk factor in the short and in the long run causing cardiovascular complications in women (1). Throughout the world preeclampsia and eclampsia are responsible for 14% of maternal deaths annually (50000-75000); whereas in the U.S.A they are accountable for 15% of premature births and 17.6% of maternal deaths. Only recently there has been an increase in the number of deaths up to 12% of all cases from 5% during 1987-1990; deaths attributed to cardiac problems, mainly to cardio-myopathy (2,3). Lately as a result of a mat analysis there has been found a link between the increased risk for cardiovascular diseases and the previous pregnancies complicated by preeclampsia. This means there exist 4 times more risks to develop hypertension and 2 times more risks to develop ischemic hear diseases, thrombo-embolism and cerebral insults (4). Other authors find a connection between preeclampsia and chronic hypertension among women later in their lives (RR, 2.00-8.00) as well as disease incidence and cardiovascular mortality (RR, 1.3-3.07) compared with those women who have had a normal intensive pregnancy. Moreover, women who are complicated by preeclampsia before the 36th week of their pregnancy and multiparous women with a history of pregnancy hypertension are more at risk than the rest (RR, 3.4-8.12) (5). The mechanisms related to preeclampsia are complex and with many factors. The common risky factors which connect preeclampsia with cardiovascular diseases are endothelial dysfunction, hypertension, obesity, hyperglycemia, the increased resistance of insulin, and dyslipidemia. Hypertension in preeclampsia happens because of vasospasms, atrial constriction and the reduced intravascular volume compared to normal pregnancies. Blood vessels in normal pregnant women reduce the response towards vaso-active peptides as Angiotensin II and Epinephrine. In contrast, women whose condition becomes complicated due to hypertension have developed an increased sensitivity to these hormones; a change which could be detected before hypertension and other signs of preeclampsia become visible. In patients with preeclampsia and chronic cypertension, a decrease of the arterial elasticity has been found as compared to

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normal intensive pregnant women (6). On the other hand, preeclampsia could cause damage of the diastolic functioning, hypertrophy of the left ventricle even after the contoll BP (7,8). Another form of hypertension during the discourse of pregnancy is the pregnancy-induced hypertension (PIH) which is contracted by 5-6% of pregnant women. Its physiopathology is still unknown but one third of patients with PIH complicated with preeclampsia (9). Women with PIH have developed a risk which is two times stronger to contract mellitus diabetes and a chronic hypertension in the years to come (10). Chronic hypertension is present in 22% of women of fertility age, but its prevalence depends on age, race and BMI. About 20-25% of women who suffer from chronic arterial hypertension develop preeclampsia thus endangering not only the mother but also the fetus itself. In cases with DBP about 110mmHg the risk of having the placenta detached, retardation of fetal growth and overlaying of preeclampsia increases. Maternal complications include eclamptic crises, cerebral insults, and pulmonary edema etc (9).

The aim of the study

The aim of this study is to estimate and valuate the structural and functional effects in the heart due to pregnancy hypertension which are not thoroughly investigated and yet are waiting to be studied here in Albania in the near future. However, the consequences of chronic hypertension have already been known. Yet not enough data have been accumulated on acute pressing overloads of which a typical case is pregnancy hypertension (11,12). During pregnancy cardiac images are limited in modalities that do not allow any sort of radiation. Echocardiography is appropriate and widely used to serve such an aim. The bibliography on the criteria to assess normal and non-normal echocardiographic parameters in normal intensive pregnant women suffering from hypertension is lacking (13). There exist a few studies which determine the normal values of the changes of the echocardiographic parameters (dimensions, volumes, speed and ingredients) which tend to increase during pregnancy (14,15).

Methods

This study is prospective where the 106 individuals included are pregnant women in the third trimester of pregnancy, complicated by pregnancy hypertension. These patients are divided into three groups with: 1) Chronic Hypertension; 2) Pregnancy-induced Hypertension and 3) Preeclampsia. There are estimated the cases with chronic hypertension when BP is larger than or equal to 140/90 mmHg (diagnosed before the 20th week of pregnancy); pregnancy-induced hypertension when BP is larger than or equal to 140/90mm Hg (transitory or chronic hypertension diagnosed in the last half of pregnancy) and with preeclampsia when BP is larger than or equal to 140/90 mm Hg and albuminuria is 24/h larger than 3 grams (diagnosed after the 20th week of pregnancy). Patients with the heart valves diseases and diabetes are excluded. ECG, measuring of BP, weight, length, weight gain, BMI, lab examinations such as those of blood analysis, albuminuria 24/h, uremia, azotemia, creatinemia, glycemia, lipidogram, have all been carried out, as well as transthoracic echocardiography to evaluated the systolic and diastolic functioning of left ventricle.

Table nr.1. Analyzed Variables

Variables	Average ± SD
Age	30.71± 8.21
Weeks of pregnancy	33.94±4.65
Weight	84.06±14.23
Weight before pregnancy	69.06±12,02
Length	162.18±6.05
BMI	31.97±5.88
Nr of pregnancy	2.16±1.19
Diastolic BP	161.98 ± 15.75
Sistolic BP	101.27±9.08
Heart rate	91.0± 17.9
Years/other pregnancy	4.5±3.08

The use of echocardiography in the evaluation of left ventricular diastolic functioning: All patients were subject to standard 2-dimensional (2D) transthoracic echocardiography. Measures were all done in "decubitus lateral" position using the following apparatus: Esaote Lab 50, Philips (HDI 5000 Sono CT) and a probe 3.5 MHz. M-mode image obtained from a (2D) image using anatomical M-mode imaging was averaged after the three circles. The diastolic functioning was estimated using the pulsing Doppler in the apical view. Mitral inflow is measured by inserting the cursor on top of the mitral leaflets. E wave (the peak early filling), A wave (the atrial filling at the end of the diastole, hence during the atria constriction), (E/A), ratio of (E) and (A) filling velocities, (DTE) the deceleration time of the early mitral inflow, (IVRT) the isovolumetric relaxation time tested through the continuous Doppler as a time from the aortic valve closure to the mitral valve opening, have all been measured. Despite the fact of being or not being under treatment, the patients at the exact time of examination, had their BP stabilized and were taken for an examination in order to exclude the influence of the increased after load in the parameters of the systolic and diastolic functioning of the left ventricle (12).

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The Geometric Model of the Left Ventricle

The quantity of LV hypertrophy is considered an important determining factor of its functioning. This is closely linked with the ventricular geometry, the mural thickness, the mass, intraventricular pressure and volume. The mass in grams of the LV is calculated according to the Devereux formula considering the diastolic measurements of left ventricular diameter, interventricular septal thickness (IVST) and posterior wall thickness (PWT):

LVM=1.04{(IVSTP+DTD+ PWTD) 3-DTD3}-13.6gr.

The Indexed mass of LV is calculated according to the formula: LVMI (g/m^2) = (=LVM/ Body surface area).

The relative wall thickness (RWT) is calculated at the end diastole as the ratio between double posterior wall thicknesses (PWT) to the LV diastolic cavity diameter. RWT= (2x PWTD/DTD). The LV geometries were classified into 4 groups based on LVMI and RWT as the following.

The Values of LVMI >_100g/m2 and RWT >_0.45 are considered normal (13). LV is considered normal in the cases when both, the indexed mass of LV (LVMI) and its relative mural thickness (RWT) are normal. When LVMI and RWT are increased LV is considered remodeled from the geometric point of view. The concentric remodeling of the left ventricle is taken into consideration when LVMI is normal and RWT is augmented. The concentric hypertrophy (CH) happens when both LVMI and RWT are grown. The eccentric hypertrophy (EH) is present when LVMI is augmented and RWT is normal.

Statistical Analyses

The continuous data are presented in an average value and SD. The discreet variables are given in absolute value and percentage. Links among variables are accomplished via the coefficients of Kendal's correlation. The differences among groups are analyzed through the student's test for constant values and square Hi for discreet variables. The analysis is carried out by means of the statistical packet SPSS 19.0. Values p<_5% are considered significant.

Results

The 106 patients of the age 30.71(±8.21) part of this study are all pregnant in 33.94 (±4.65) week of pregnancy complicated by Pregnancy Hypertension. It is estimated that 37 patients or 34.6% of all cases suffer from Chronic Hypertension, 81 patients or 75.7% of all cases from Pregnancy-induced Hypertension and 59 patients or 55.1 of all cases from Preeclampsia.

Table nr. 2 Women's Age and Weeks of Pregnancy

Groups	Women's Age	Weeks of Pregnancy
PIH (n=81)	30.2+6.0	33.3+5.3
Preeclampsia (n=59)	30.6+6.9	33.2+6.9
Chronic Hypertension (n=37)	32.1+5.2	33.1+6.4

Patients with Chronic Hypertension are older than those of the other 2 groups (32.1/30.10) but they are not characterized by any changes statistically important (p=0.465). The weight of the women with preeclampsia before pregnancy is heavier than that of the women in the other 2 groups (70.39/65.70) but without any changes statistically important (p=0.081). Women with preeclampsia are characterized by a weight gain heavier than the rest (16.37±5.340) as compared to those without Preeclampsia (p=0.022) even BMI is greater (32.88±5.59) but with reference to BMI the difference among groups is not significant (p=0.072). The result: Geometric Model of the Left Ventricle.

Table nr.3 Determining the Geometric Model of the Left Ventricle

Geometric Model	Preeklamsia n=59	PIH n=81	CH n=37
Normal	18.6%	12.3%	18.9%
Eccentric Hypertrophy	64.4%	71.6%	64.9%
Concentric Hypertrophy	16.9%	16.0%	16.2%

In 18.6% of cases with Preeclampsia, 12.3% of cases with Pregnancy-induced Hypertension, 18.9% of cases with Chronic Hypertension, the left ventricle is found normal. In 64.4% of cases with Preeclampsia, 71.6% of cases with Pregnancy-induced Hypertension, 64.9% of cases with Chronic Hypertension the Eccentric Hypertrophy of the left ventricle is present. In 16.9% of cases with preeclampsia, 16.0% of cases with Pregnancy-induced Hypertension and 16.2% of cases with Chronic Hypertension, Concentric Hypertrophy is present. It has not been found any Concentric Remodeling of the left ventricle.

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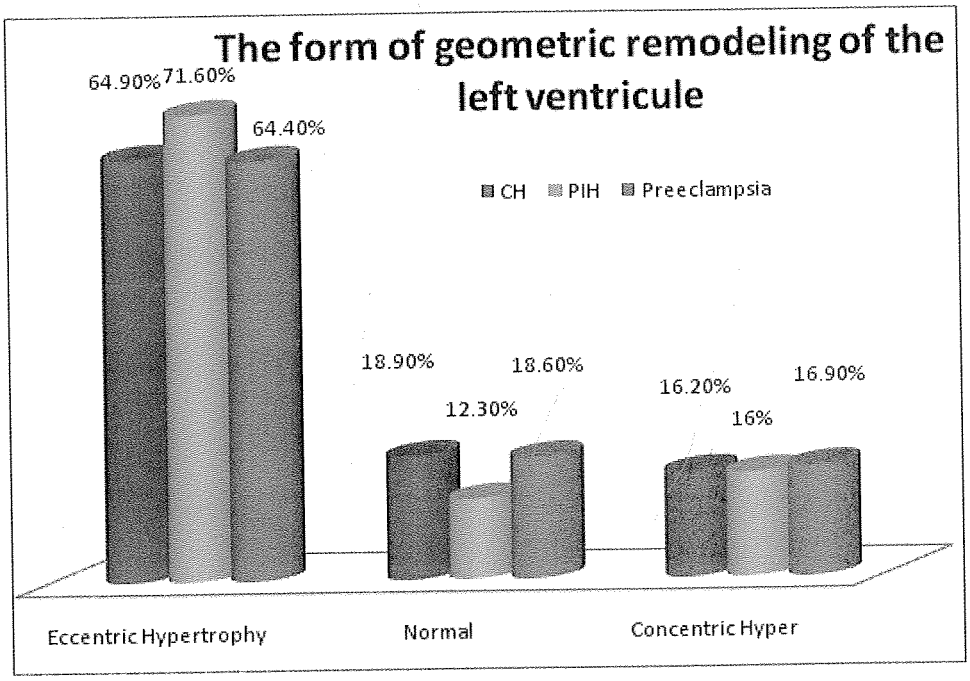

Graphic nr.1
Result: Valuation of the Diastolic Functioning of the LV

Table nr. 4. The data of Echocardiography

Echo-parameters	Mesatare	SD
DTD	49.82	3.8
DTS	32.17	3.28
FS%	36.02	4.21
EF%	65.21	5.53
SWT	11.51	14.01
PWT	9.5	3.48
MVM	176.25	44.41
LAD	37.18	3.63
LAs	20.83	19.72
A4.dia	4.27	1.01
E	0.58	0.03
A	0.67	0.05
E/A	1.03	0.06
DTE	183.67	40.82
IVRT	112.33	39.2
Vp	61.66	30.89

Being based on the echo-parameters, it is decided that the situation is normal when IVRT 50-100 (ms), DTE 150-200 (ms) and E/A 1-2, the impaired relaxation of the LV when we have IVRT >100 (ms) and DTE>200 (ms) and E/A <1, a pseudo normal filling pattern of the LV when IVRT 50-100(ms), DT 150-200 (ms) and E/A <1, a restrictive pattern of LV's filling when IVRT is <50 (ms), DT<150 (ms) and E/A 1-2 or E/A<<1.
Result: Echo Data via ANOVA

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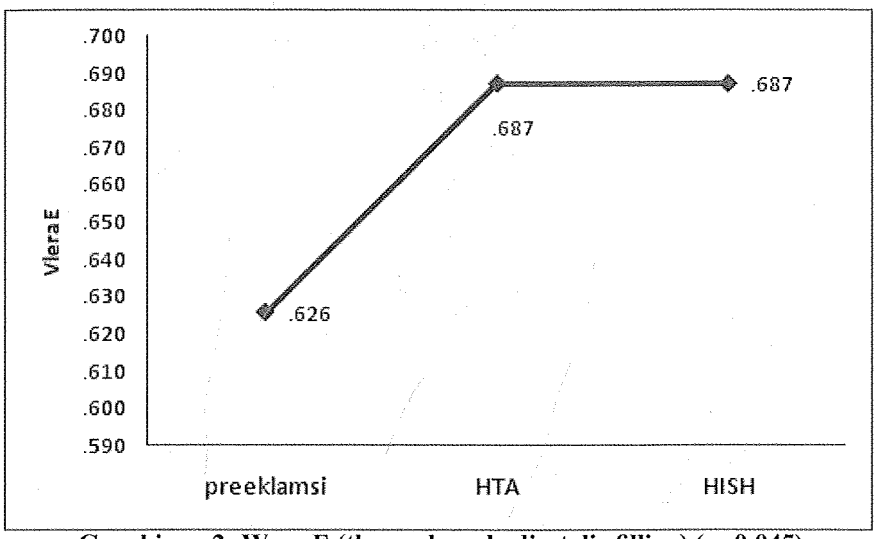
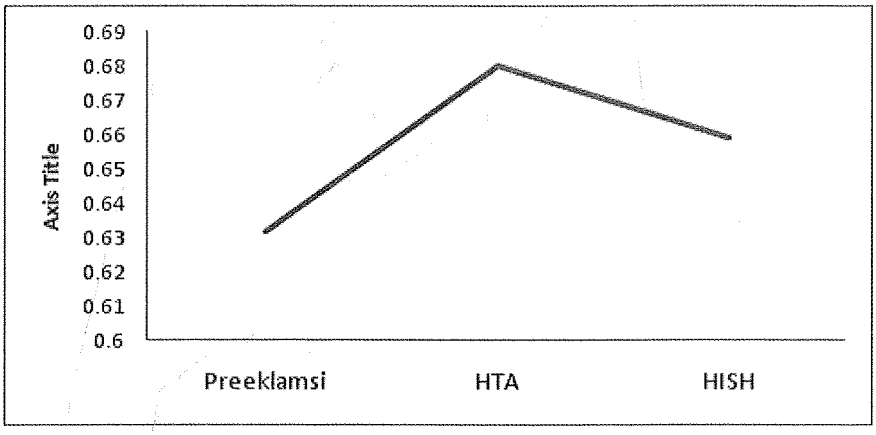
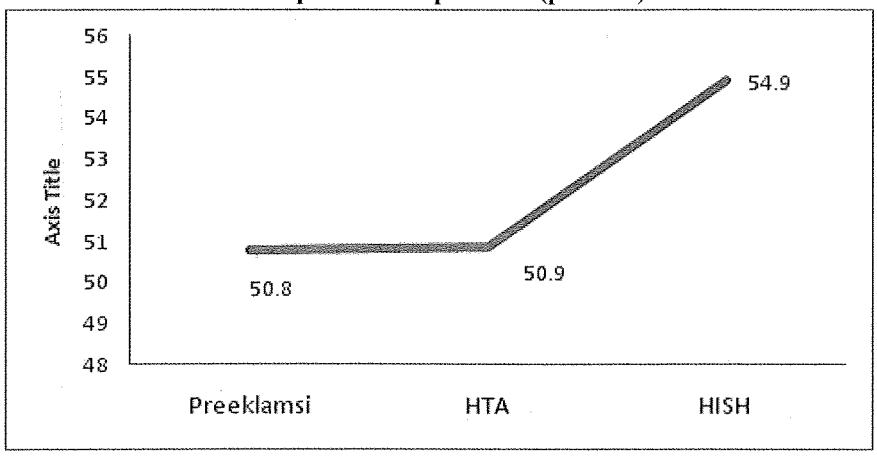


Graphic nr.2: Wave E (the peak early diastolic filling) (p=0.045)



Graphic nr.3 Wave A (the atrial filling at the end of the diastole) (p=0.013)

Graphic nr.4 Report E/A (p=0.031)



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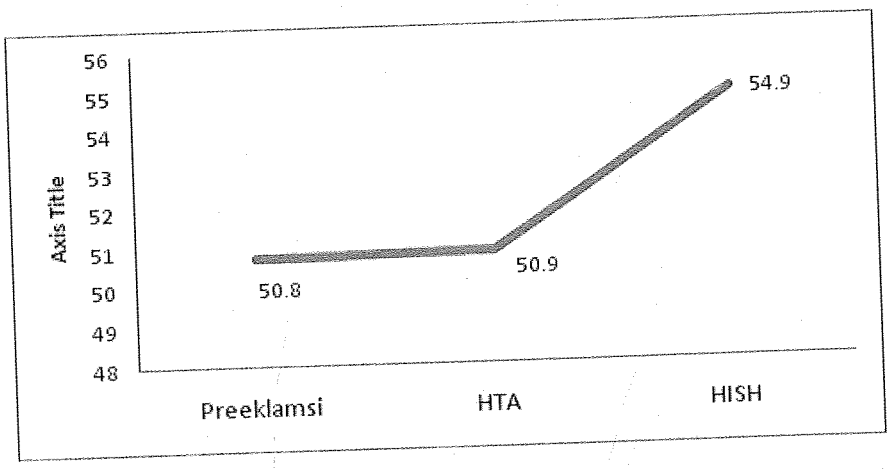
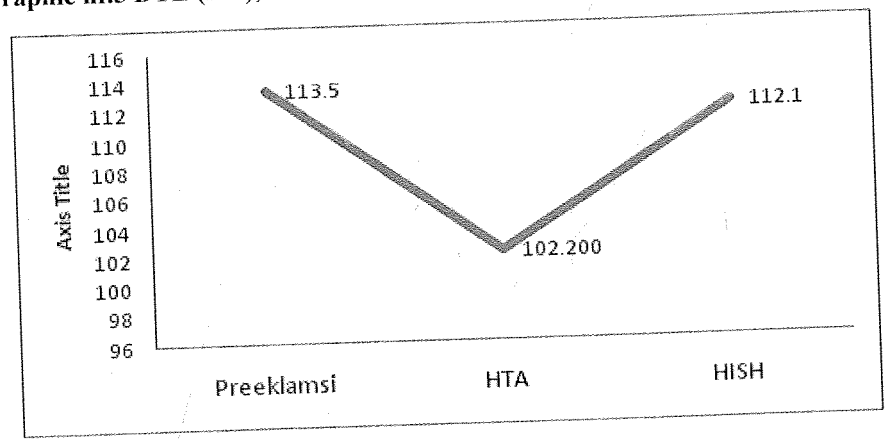


Graphic nr.5 DTE (m/s), the deceleration time of the early mitral inflow (p=0.023)



Graphic nr.6 IVRT (m/s), the isovolumetric relaxation time (p=0.005)

Table nr.5 Dyastolic function linked with the groups of Hypertension

	Normal (n=16)	Impaired relaxation (n=41)	Pseudo normalization (n=33)	Restrictive pattern (n=16)	Total
ChH	5	16	11	5	37
	14%	43%	30%	14%	100%
Preeclamsia	12	19	15	13	59
	20%	32%	25%	22%	100%
PIH	11	32	26	12	81
	14%	40%	32%	15%	100%

The normal pattern is found in the three groups but more in the group with Preeclampsia (20%). Impaired relaxation is present in the 3 groups but it is seen more often in the group with Chronic Hypertension (43%). The Pseudonormalization pattern is evident in the three groups but more in the group with Pregnancy-induced Hypertension (32%). The Restrictive pattern is found in the 3 groups but more often it is seen in the group with Preeclampsia (22%). Via the coefficients of the Kendal's correlation a reverse link is found between the damage of the diastolic functioning estimated by ECHO and geometric remodeling (r=-0.127, p=0.051), (the presence of the geometric remodeling talks about the damage of diastolic functioning). No link is found between the echo-graphic pattern with the weight, weight gain and BMI.

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Discussions

In normal pregnancies cardiac output and heart rate increase whereas peripheral resistances decrease. As a result of such differences women's heart could suffer structural alterations and remodel during pregnancy. Here, the growth of diastolic diameter and the structural changes of the left ventricle after the first half of pregnancy are included. On the contrary in pregnancy hypertension peripheral resistances increase whereas blood and plasmatic volumes remain normal or are likely to decrease. When the heart is set under such a blood related dynamic burden, it acts in three ways to compensate. These ways are as follows:

1. Using Frank Starling mechanism.
2. Increasing its muscular mass to cope with the increased burden, to diminish mural stress and precisely the type of overload the heart is subject to determines the type of hypertrophy.
3. Using neuro-hormonal mechanisms to increase contraction.

In general voluminous overload produces an increase of the intra-ventricular volume and pressing overload causes the growth of the ventricular mass. Hypertrophy, as a consequence of this pressing overload, has an important influence in the cardiovascular system and different geometric models might be created. These changes cause an increase of the after load of the LV which has to be normalized after childbirth (16). However, the data presented by various authors referring to the structural alterations of the LV are not the same. Sanchez, Thompson and their collaborators do not find changes in the mass of the left ventricle (17). Vazquez Blanco and his coauthors point out that the geometric pattern is modified during pregnancy hypertension and there is a growth of mass and relative mural thickness of LV (8). These authors find an increase of the mass and the relative mural thickness of LV, and they say that the most frequent geometric model is Eccentric Hypertrophy as well as Concentric Remodeling (18). Vlahovic-Stipac and others report that there is a growth of the mural thickness which brings about an increase of the mass of the LV but they do not find significant changes in volume (19). Other researchers have found a visible increase of the mass of the LV which is reflected by a visible increase of the septum and posterior wall's thickness but without any changes in the diastolic diameters of the left ventricle (20). Finally there are other researchers who have found the mass increased, the left ventricle's Eccentric Hypertrophy, damage of the diastolic functioning and longitude systolic dysfunctioning (21). Pregnancy Hypertension in contrast with the essential hypertension influences the cardiovascular system of women, who have been healthy for about 9 months, time which it seems has been inadequate to adapt to this new pressing overload. The latter being increased by hypertension builds up on the increased voluminous physiology of pregnancy. Now the fact that obesity serves as a risk factor for hypertension has become well-known and overweight women are prone to complications due to hypertension during pregnancy (22). The combination of increased pressure and volume in cases of Pregnancy Hypertension explains maybe the tendency to Eccentric Hypertrophy of the Left Ventricle as it is referred to by other researchers (23). The dilation of the cavity with a fall of the ratio between mural thickness and cavity proportions is compensatory at the very beginning. Alike damages could be found in cases of Cardiac Insufficiency with an increased voluminous overload. In spite of the fact that this has not been observed in our cases, the possibility cannot be excluded that this form of the geometric remodeling might cover up a mute form of LV's contractile insufficiency (24,25). In our study the form of geometric remodeling often found in the 3 groups of study is the Eccentric and Concentric Hypertrophy of the Left Ventricle, this possibly explained with the fact that an increase of the peripheral resistances and a decrease of preloading is caused by the contraction of the intravascular volume. Unfortunately patients who suffer from Preeclampsia and have developed the Eccentric Hypertrophy of the LV can be laden with peri-portal Cardio-myopathy, thus showing that the form of geometric remodeling might be a predisposing factor (26). Concentric Hypertrophy is likely to be, in the cases which are characterized by an increase of after load, a mechanism appropriate for adaption but the small percentage this form of remodeling occupies in our study can be explained with the fact that in cases with Inducted Pregnancy Hypertension and Preeclampsia this is a phenomenon which does not last for a long time, it lasts about 10 months. A considerable number of the hypertonic might have a normal geometric model as it is also found in some of our patients, a situation which could explain probably the lack of structural changes with the special contractile status of Myocardium or genetic causes. Damages of the diastolic functioning are found present even in normal pregnancies, in spite of the fact that there are some researchers who dot approve of their existence. Nevertheless, what different studies have in common is the idea that they are present even in the pregnancy hypertension (27,28). There are authors who claim that the repot E/A tends to fall but the speed of wave E does not change, whereas wave A and IVRT tend to increase (29). These data suggest that structural and functional changes of Hypertension during pregnancy are persistent even when hypertension values normalize. Despite the fact that a part of our patients have a normal geometric model, this transitive pressing overload is able to start injecting changes in the left ventricle's structure. The parameters of the LV, the left atrium and the aorta's root do not change, perhaps because of insufficient time to act.

Conclusions

In our study it is stated that the mass of the left ventricle is grown as a result of the growth of the septum's thickness and that of the posterior wall. No significant differences are found in the LV's parameters. The indexed mass of the Left Ventricle is found increased as well as its relative mural thickness. Another finding is the existence of the geometric remodeling of the LV with the presence of Eccentric Hypertrophy (EH 67%) and (CH 16%) as well as the presence of damages of diastolic functioning. Pregnancy hypertension damages the diastolic functioning of the left ventricle and at the same time it influences its geometric remodeling. In order to evaluate the persistence of such changes, all patients should be attended to see their developments after childbirth. However, by echocardiographicy can identify patients suffering from complications in the 3d trimester of pregnancy.

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