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Study of the Left Ventricular Function in Pregnancy-Induced Hypertension

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Summary: Left ventricular (LV) morphological and functional characteristics in 9 women suffering from pregnancy-induced hypertension (PIH) were studied by means of echocardiograms. In order to distinguish which changes depended on the pressure values and which were the result of pregnancy, 10 nonpregnant control women with no heart disease and 10 normal pregnant women (NP) were studied and the results of each of the groups compared. To evaluate the structure, left ventricular systodiastolic diameters and wall thickness were measured. The only statistically significant difference was in the diastolic diameters between the PIH ($4{:}7 \pm 0.3$ cm) and the control group (4.4 ± 0.2 cm) $p < 0.01$. Left ventricular mass was significantly increased ($p < 0.01$) in the PIH patients (185 ± 53.1 g) compared to the NP patients (161 ± 29.6 g) and the control group (125 ± 17.4 g). No statistically significant differences were found in the radius thickness ratio in the three groups. The systolic function assessed by the shortening percentage was significantly lower ($p < 0.05$) in the control group ($32.8 \pm 4.4\%$) and in the NP patients ($37.8 \pm 5.2\%$) than in the PIH group ($39 \pm 6.5\%$). Afterload assessed by isovolumic period stress was significantly greater ($p < 0.01$) in the PIH patients (157 ± 10.6 dyne/cm^2) compared with the NP group (118.9 ± 7.01 dyne/cm^2). There were no significant differences between the first group and the control group (134.09 ± 8.7 dyne/cm^2). As evidence of the diastolic function, analysis was made, on the one hand, of diastolic isovolumic period length (DIP). Values in the control group were 50 ± 8.1 ms, in NP 54 ± 14.2 ms, and in PIH 50 ± 12.2 ms. There were no significant differences between the groups. On the other hand, peak velocity diameter diastolic changes were determined: that of the control group was 18 ± 3.8 cm/s; for the NP group, 18 ± 5.9 cm/s; and for the PIH group, 21 ± 5.3 cm/s. There were no statistically significant differences here either. The foregoing results lead us to the conclusion that PIH does not produce evident structural changes in the left ventricular cavity beyond those already caused by adaptation to pregnancy. The changes in systolic function may be secondary to the effects of an added adrenergic activity, suggested in turn as being responsible for this hypertension. Diastolic function is not altered despite the increase in the left ventricular mass in this group of patients.

Key words: pregnancy-induced hypertension, left ventricular function, systolic function, diastolic function, hypertension, pregnancy

Introduction

Arterial hypertension produced evident structural changes in the left ventricle,[1,2] usually acompanied by functional alterations.[3,4] In the great majority of cases these alterations precede the appearance of clinical manifestations.[5]

Although certain hemodynamic aspects relating to the pathophysiology of pregnancy-induced hypertension are known,[6,7] the structural and functional characteristics of the left ventricle have not yet been properly defined.[8,9]

The appearance of these changes prior to the detection of the clinical modifications and the importance of an individual approach for appropriate therapeutic management were the main reasons for our undertaking this research. Echocardiographic assessment was made of the morphological and functional characteristics of the left ventricle in a group of pregnant women suffering from pregnancy-induced hypertension.

Patients and Materials

A total of 29 nulliparous women between 16 and 35 years of age were studied. Group classification was: Ten

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nonpregnant women with no history of heart disease and with normal physical, electrocardiographic, radiological, and echocardiographic results, comprised the control group. Ten pregnant women with between 26 and 42 weeks of amenhorrhea, carrying a single fetus and with the same characteristics in the clinical, electrocardiographic, radiological, and echocardiographic tests as the previous group, constituted the normotensive pregnant group. The remaining 9 women, pregnant with characteristics similar to the previous group, but suffering from pregnancy-induced hypertension were the hypertensive pregnant group. They were judged to be suffering from pregnancy-induced hypertension when systolic pressure figures equal to or higher than 145 mmHg and diastolic figures equal to or higher than 95 mmHg were found in three successive checks after 26 weeks of amenorrhea, whether or not associated with edema and proteinuria. There was remission of this hypertension 6 weeks after the pregnancy ended.

Methods

In each case, a two-dimensional echocardiogram was performed along the long and short axes to obtain two- and four-chamber views. Left ventricular segmental motion was studied and different measurements recorded. M-mode recordings were obtained at a speed of 100 mm/s from the left ventricular cavity at level 1. With the ultrasound beam placed between the papillary muscles along the short axis or between the free end of the mitral valve and that of the papillary muscle along the long axis, the section most perpendicular to the endocardium of the posterior wall was selected for M-mode recording. The mitral echogram was recorded at level 2 to allow observation of the opening of this valve at the same recording velocity; at the same time, a phonocardiogram was obtained with the microphone placed in the aortic area in order to identify the closing of the aortic valve.

From the foregoing recordings, parameters were established indicative of the structural characteristics of the left ventricle and of both its systolic and diastolic functions.

For an analysis of the structural characteristics, measurements were taken of the systolic (Sd) and diastolic (Dd) diameters of the left ventricle, as well as of the thickness of its walls. These measurements were carried out in M-mode at level 1, following established recommendations.[10] Ventricular mass[11] and the relative thickness of the posterior wall[12] were calculated using diameter and thickness.

The systolic function was assessed by the shortening percentage

$$\%Ac = \frac{Dd - Sd}{Dd} \times 100$$

Myocardial tension was also calculated at the end of the isovolumic period,[3] as an indication of afterload.

The diastolic function was studied from the duration of the diastolic isovolumic period, measured from the closing of the aortic valve (phonocardiogram) to the opening of the mitral valve (mitral echogram) and from the peak velocity of diameter change in the left ventricle. In order to obtain this value, a cardiac cycle was divided into 40 ms on the level 1 recording, the cavity diameter being studied at each interval. Using these data, a diameter curve was drawn as a function of time, the first derivative (change of diameter/change of time) being calculated, taking the maximum positive value as the peak velocity of diameter increase. Figure 1 shows a curve from the echocardiogram analysis of one of the patients studied.

Blood pressure was obtained by indirect means with a sphyngomanometer in the prostrate position prior to echocardiographic study. The average of three consecutive measurements was taken. Mean blood pressure was calculated using systolic and diastolic pressure values.

For the statistical analysis of the variables Student's *t*-test was carried out, statistically significant differences were those with a p value < 0.05.

Results

The normotensive group had a higher average age (27) than the hypertensive group (24) and the control group (21).

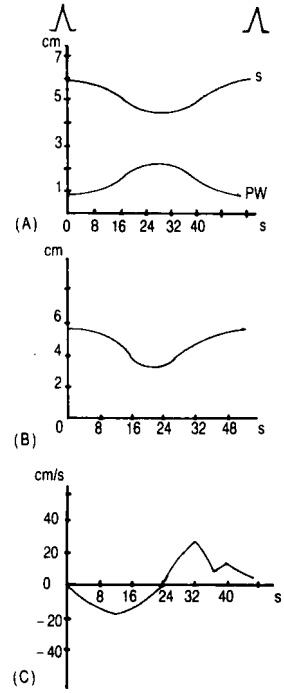

FIG. 1 Graph of the echocardiac analysis of one patient studied. (A) Diagram of the endocardium of the interventricular septum (s) and that of the posterior wall (PW) of the left ventricle, showing their variation in position throughout the cardiac cycle. (B) Variation of left ventricular cavity diameter as a function of time. (C) First derivative (dd/dt) of the curve drawn in B.

Heart Rate and Blood Pressure

Heart rate was slightly higher in the hypertensive group (119±21.3 beats/min) than in the normotensive one (118±14.9 beats/min) and the control group (108±10.2 beats/min). These differences were not statistically significant.

Systolic blood pressure was 123±3.8 mmHg in the control group, 116±2.7 mmHg in the normotensive pregnant group (NS), and 151.6±5.7 mmHg in the hypertensive pregnant group ($p<0.01$ for both groups). Diastolic pressures were 77±1.5 mmHg, 74±2.3 mmHg (NS), and 105.3±4.3 mmHg ($p<0.001$ for both groups). Mean pressure values also showed a significant increase in the hypertensive group (120±4 mmHg) in relation to the normotensive pregnant group (90.6±2.5 mmHg, $p<0.01$) and to the control group (92±1.8 mmHg, $p<0.01$).

Ventricular Structure

Left ventricular diastolic diameter was larger in the hypertensive pregnant group (4.7±0.3 cm) in comparison with the normotensive group (4.6±0.3 cm) (NS) and the control group (4.4±0.2 cm $p<0.01$).

Systolic diameters were similar in the three groups (control group 2.9±0.2 cm, normotensive pregnant group 2.9±0.2 cm, and hypertensive pregnant group 2.9±0.3 cm), with no statistically significant differences. Figure 2 shows a graph of the ventricular diameter study.

Left ventricular mass was significantly increased in the hypertensive pregnant group (185±53.1 g) in comparison with the normotensive pregnant group (161±29.6 g, $p<0.01$) and the control group (127±17.4 g, $p<0.01$) (Fig. 3).

The relative thickness of the posterior wall was similar in the three groups (control: 0.33±0.01, normotensive pregnant: 0.38±0.02, and hypertensive pregnant: 0.36±0.02).

Systolic Function

In no case were there any changes in the left ventricular segmental motion.

Afterload, assessed by isovolumic period stress was 134.09±8.7 dyne/cm² in the control group with lower values in the normotensive pregnant group (118.9±7.01 dyne/cm², NS). The hypertensive pregnant group showed the highest average value (157±10.6 dyne/cm²) (NS in comparison with the control group and $p<0.01$ compared with the normotensive pregnant group).

Shortening percentage was significantly lower in the control (32.8±4.4%) and normotensive pregnant (37.8±5.2%) groups than in the hypertensive pregnant one (39±6.5%) ($p<0.05$ for both groups).

Figure 4 analyzes the behavior of these parameters.

Diastolic Function

Diastolic isovolumic period was slightly longer in the normotensive pregnant group (54±14.2 ms) compared with the hypertensive pregnant group (50±12.2 ms) and the control group (50±8.1 ms). No statistically significant differences were found.

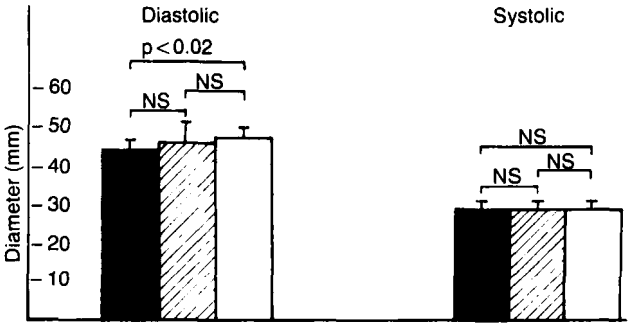

FIG. 2 Bar graph showing average left ventricular systolic and diastolic diameters and standard deviations in the groups studied. Dark bar: control group; striped bar: normotensive pregnant group; light bar: hypertensive pregnant group.

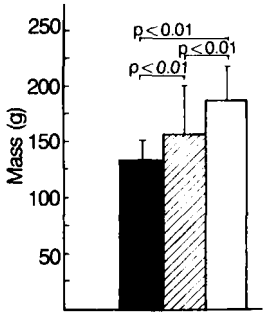

FIG. 3 Similar to Figure 2, but showing the left ventricular mass. Note increase in the pregnant group mass in comparison to the control group.

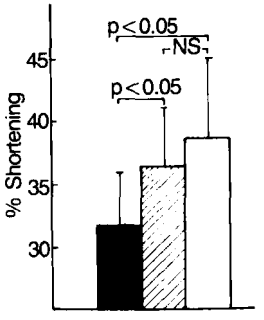

FIG. 4 The bar graph enables us to analyze the systolic function characteristics in the three groups, using the shortening percentage. The bars show average value and respective standard deviations. The groups are represented as in Figure 2. Values are greater in the pregnant group, regardless of whether or not they are hypertensive, compared to the control group.

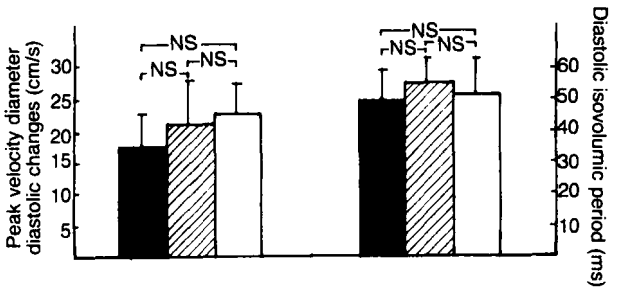

FIG. 5 Analysis of the groups' diastolic function. Two parameters that assess the diastolic function are represented in bars using the same criteria and significance as in the other figures. On the right, diastolic isovolumic period length, and on the left peak left ventricular diameter diastolic change velocity. As can be seen, there are no statistically significant differences in any of the parameters.

Change in left ventricular peak velocity of diameter change values were similar, no statistically significant differences were found between the groups: control group 18 ± 3.8 cm/s; normotensive pregnant group 20 ± 5.9 cm/s; and hypertensive pregnant group 21 ± 5.3 cm/s. Figure 5 shows the results discussed here.

Discussion

Volume overload, such as occurs during pregnancy, together with a pressure overload from the induced hypertension, are sufficient to provoke left ventricular structural and/or functional changes.[1,13,14]

Our results coincide with those of other authors,[15,16] with pregnant women giving evidence of a higher heart rate and an increased diastolic diameter in the left ventricle compared to the control group. However, we should point out that the lack of statistical significance between the differences when the ventricular diameter was compared in the case of the normotensive pregnant and the control groups. This lack of significance may be due in part to the reduction in ventricular filling time in these groups because of their higher average heart rates, and in part, to the possible influence of the supine position due to the change in venous return produced by the pregnant uterus in this position. In our cases, the recordings were made with the aim of achieving a good echocardiographic definition, which is sometimes obtained in positions somewhere between a lateral prostrate and a supine position.

Katz *et al.*[17] have indicated an increase in ventricular mass throughout pregnancy, with maximum values being reached shortly before term. This increase in mass coincides with our observations. If there was high blood pressure during the pregnancy, there was a greater increase in the mass. Although calculation of the ventricular mass is important in the study of patients with high blood pressure, it is also known that the volume overload produces an increase in the thickness of the ventricular wall as a compensatory mechanism. In the situation analyzed here it is therefore difficult to define the meaning of the hypertrophy only in relation to pressure overload. The type of hypertrophy that was observed in the pregnant women has characteristics of eccentric hypertrophy.[17] If the increase in mass is analyzed more precisely in order to decide whether it responds to an eccentric or concentric type of hypertrophy, for example via the relative thickness of the posterior wall, we observe that in our cases it is closer to the first type.

The findings in previous studies on the left ventricular systolic function in patients with induced hypertension are controversial. Lim and Walter[7] found a reduction in the systolic function in patients with induced hypertension in an assessment made by the measurement of systolic times. Using echocardiographic parameters, Larkin *et al.*[8] and Kuzniar *et al.*[18] found no change in the systolic function between pregnant women suffering from pregnancy-induced hypertension and those who were not. Rafferty and Berkowitz[19] and Benedetti *et al.*[20] showed a hyperdynamic response with an increase in ventricular function assessment indexes via invasive analysis.

These discrepancies may be a consequence of different characteristics in the populations studied, arising, for example, from the use of medication in some groups. Both the shortening percentage and the circumferential shortening velocity were higher in the hypertensive pregnant group. If we bear in mind the scant variation found in ventricular diameters between the hypertensive and normotensive pregnant groups, which would mean only a slight modification in the preload, and the significant increase in the afterload in the latter group, it is possible to accept that the greater value of these indexes apparently corresponds to a real inotropic increase in the hypertensive pregnant group.

Hanrath *et al.*[6] have shown a modification in the diastolic function indicated by a lengthening of the isovolumic period and a reduction in the ventricular filling velocity in patients with left ventricular hypertrophy. Inouge *et al.*[21] have also indicated changes in relaxation in slightly to moderately hypertensive women, but in close association with the development of ventricular hypertrophy. We have not, however, found any studies on the behavior of diastolic function in this type of hypertension. An analysis of our results presents no significant changes in the left ventricular diastolic behavior in the hypertensive pregnant group. The lack of concentric ventricular hypertrophy might explain this normal diastolic function. Not only did we not find any deterioration in this function, but from an analysis of the length of the isovolumic period, which was shorter in the hypertensive group when the rise in the pressure figures would have indicated the opposite behavior,[22] one could suggest the possibility of an improvement in relaxation not sufficiently appreciated due to the rise in pressure of this group.

The sympathetic hyperactivity described by some authors in patients with induced hypertension[7] may explain some of the findings discussed, such as the increase in the systolic function, the increase in heart rate, and the possible increase in relaxation velocity.

Conclusion

According to our observations, we conclude that pregnancy-induced hypertension does not produce clear structural changes in the ventricular cavity beyond those already produced by adaptation to pregnancy. The changes in ventricular function may be due to the effects of an increase in adrenergic activity, postulated as responsible for this hypertension, rather than to the presence of the hypertension.

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