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Echocardiography Evaluation of Cardiovascular Status in Pregnant Women Attending Antenatal Clinic in LAUTECH Teaching Hospital Ogbomoso, Nigeria

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ABSTRACT

Aim: This study is aimed to evaluate the echocardiographic changes in normal pregnant women.

Study Design:The study was a cross-sectional descriptive design

Method: The study wascarried out among Ninety-sixpregnant women in all trimesters of pregnancy and one hundred non-pregnant women between theages of 18 and 45 years.

Echocardiography was done with the LOGIQ Pro 6 machine. The M-mode, 2D and Doppler echo studies were performed. Data were analyzed using the Statistical Package for Social Sciences (SPSS) version 20.0.

Results: Pregnancy is associated with low diastolic blood, higher fractional shortening and ejection fraction. The Mitral E/A ratio is lower, and the left ventricular mass index, wall thickness and chamber dimensions are higher in pregnant women compared with controls. The cardiac axis is lower (leftward rotation), tachycardia, left bundle branch block and non-specific intraventricular block tend to occur more commonly during pregnancy.

Conclusion: Pregnancy is associated with significant dynamicchanges in cardiac functions, and echocardiography remained an invaluable tool in assessing cardiac functions and identifyingrisks associated with cardiovascular disease or that could exacerbate a preexisting one during pregnancy.

Keywords: Echocardiography, pregnancy, cardiovascular system, systolic function, diastolic function, echo in pregnancy.

I. INTRODUCTIONS

Pregnancy is associated with significant hemodynamic changes, hence, a potentially high-risk period for women with underlying cardiovascular disease[1]. Echocardiography remains the preferred modality for diagnosis and monitoring of pregnant women with cardiovascular disease as it is widely available[1]. It is simply the ultrasound evaluation of the heart. Having different imaging modalities, it can be used to non-invasively assess the function and structure of the heartfor determining both physiologic and pathologic changes, especially during pregnancy,Echocardiography poses no risk to both the fetus and the mother[2].

It is also a great tool for assessing the effect of hemodynamic changes on the heart[1]. Previous studies using echocardiographyin different populations have shown variable results. This variability may partly reflect differences in the study population characteristics like age and race, suggesting an entirely different pattern among the Blacks.However, the most consistent and prominent physiological hemodynamics change in pregnancy is the increase in cardiac output (CO) which is due to an increase in the blood volume that accompanies pregnancy[3].Cardiac output increases significantly in the early to mid-third trimester and is maintained until term. Peak Cardiac Output of 46-51% occurred from a mean of 15% increase in heart rate and about 24% increase in stroke volume[4].

In addition, during pregnancy left ventricular mass increases by 52%. There is an increase in left ventricular end-diastolic and end-systolic diameters (12% and 20%, respectively), left ventricular posterior wall diameter during diastole and systole (22% and 13%, respectively) and left intra-ventricular septum during diastole and systole (15% and 19%, respectively)[5].

The natural volume overload in pregnancy, leading to a reversible 'physiological' left ventricular hypertrophy, results in a short-term decrease in systolic function and a significant

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change in left ventricular diastolic function. Left ventricular diastolic function increases in the first two trimesters but declines in the third trimester with a decrease in acceleration, consistent with an increase in ventricular compliance[5].

Mitral valve A-wave maximum velocity increases during pregnancy by 19%, while mitral valve E-wave maximum velocity and the ratio of E-wave/A-wave velocities increase early in pregnancy by about 14% and 6% respectively, with a subsequent decline to 4% and 10%, respectively, below non-pregnant levels[6]Changes in heart rate, preload, and contractility as well as stage of pregnancy influence this alteration[7].While left ventricular systolic function is normal in all patients one week after childbirth, left ventricular hypertrophy and left ventricular diastolic dysfunction could persist for nearly two months[8].
There is a paucity of data on this subject, particularly in the study area, for which reason this study is aimed to evaluate the echocardiographic changes in normal pregnant Nigerian women.

II. METHODOLOGY

2.1 Study design

The study was a cross-sectional descriptive design carried out at theLadoke Akintola University of Technology Teaching Hospital (LTH) Ogbomoso, Oyo State Nigeria. LAUTECH Teaching hospital is located in Ogbomoso town, Oyo state. Ogbomoso lies on the plateau of Yorubaland (elevation 1,200 feet) in an area of savannah and farmland with an estimated population of about a 1.2Million people and is the second largest city in Oyo state after the capital city of Ibadan[9].

2.2 Sample size

The sample size was derived using the proportion of various abnormalities onechocardiography from a previous study[10] in Nigeria.Ninety-six consecutive pregnant women in all trimesters of pregnancy; 24 in the first trimester, 39 in the second trimester and 33 in the third trimester and one hundred nonpregnant women between the ages of 18 and 45 years were recruited for the study.

2.3 Study Procedure

Each subject had a complete clinical cardiac status assessment, after signing informed consent.

The resting pulse rate and bloodpressure were taken.Echocardiography was carried out on each subject with the LOGIQ Pro 6 machine. The M-mode, 2D and Doppler echo studies were performed. The measurements were taken with subjects lying in the left lateral position[11].

Images of the heart were obtained in multiple cross-sectional planes with the use of standard transducer positions – the parasternal long and short axis views, apical long axis, two and four-chamber views with the subject in the left lateral position. Measurements were by the recommendations of the American Society of Echocardiography using the leading edges method of measurement.[11]

The following left ventricular dimension and function were measured/ calculated:

1. Left ventricular posterior wall thickness in diastole (PWTd)
2. Inter-ventricular septum thickness in diastole (IVSd)
3. Left ventricular end-diastolic dimension (LVDd)
4. Left ventricular end-systolic dimension (LVDs)
5. Left ventricular end-diastolic volume (LVEdV) = $(LVDd)^3$ x 1.05
6. Left ventricular end-systolic volume (LVEsV) = $(LVDs)^3$ x 1.05
7. Stroke volume (SV) = LVEdV – LVEsV
8. Ejection fraction (EF)% = SV/LVEdV X 100
9. Fractional shortening (FS)% = LVDd – LVDs / LVDd X 100
10. Left Ventricular Mass (LVM)[12] = 1.04 (LVDd + PWTd + IVSd)^3 –$(LVDd)^3$ – 13.6g.
11. Left Ventricular Mass Index (LVMI) = LVM/Body Surface area in $m^{2.}$
12. Stroke Volume Index (SVI) = SV / Body Surface area in m^2.
13. Left atrium cavity dimension (LA) mm.
14. Aortic root dimension (AO) mm.
15. Relative Wall Thickness[13], (RWT) = 2PWTd/LVDd.
16. Peak early transmitral filling velocity (E) m/s.
17. Peak late transmitral filling velocity (A) m/s
18. The ratio of early and late transmitral filling velocity (E/A).
19. Deceleration time of the early transmitral filling velocity in ms.
20. Isovolumic relaxation time in ms (IVRT, the time interval between aortic valve closure and mitral valve opening).
21. Systemic vascular resistance (dyn x sec/cm^5) = 80 (mean arterial pressure - 3)/cardiac output

2.4 Data Analysis

Data were analyzed using the Statistical Package for Social Sciences (SPSS) version 20.0. Continuous data were presented as mean ±standard deviation and categorical variables were presented

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as percentages. Statistical significance was taken as $p < 0.05$, and the confidence level of 95%.

2.5 Ethical Clearance

Approval was obtained from the Ethical Review Committee of Ladoke Akintola University Teaching Hospital Ogbomoso.

III. RESULTS

The study included 196 subjects consisting of 96 pregnant women and 100 controls. The mean age of both pregnant women and the controls was similar (28.1 ± 5.1 vs. 26.4 ± 5.3 years, p=0.10). Thirty-six percent (n= 35) of the pregnant women were primigravidae, while others were multigravidas. (Table 1)

Table I: Socio-demographic and Anthropometric Parameters of subjects and Controls

Variable	Subjects Mean ± SD (n = 96)	Controls Mean ± SD (n = 100)	P value
Age (years)	28.1 ± 5.1	26.4 ± 5.3	0.10
Weight (kg)	64.9 ± 11.8	59.7 ± 12.0	0.010*
Height (m)	1.58 ± 0.06	1.61 ± 0.06	0.003*
BMI (g/m^2)	26.0 ± 4.1	23.2 ± 4.4	< 0.001*
BSA (m^2)	1.68 ± 0.17	1.63 ± 017	0.093
Average income (Naira)	14,940 ± 18,792	13,401 ± 8,248	0.566
Parity (N)			
0	35 (36.5%)	84 (84.0%)	
1	29 (20.2%)	6 (6%)	
2	14 (14.6%)	2 (2%)	
3	10 (10.4%)	5 (5%)	
4	7 (7.3%)	2(2%)	
5	1 (1.0%)	1 (1%)	

Key: **SD**= standard deviation, **BMI=** Body mass index, **SBP**= systolic blood pressure, **DBP**= diastolic blood pressure, BSA= Body surface area, kg= kilograms, m= metres * statistically significant.

The mean ejection fraction was within the normal limits in both pregnant women and control but significantly higher in the pregnant women (65.1 ± 7.3 Vs 61.4 ± 10.1%, p= 0.015). Similarly higher values in the mean fractional shortening (36.4 ± 5.1 vs. 31.5 ± 6.1%, p< 0.001) as well as mean velocity of fractional shortening (1.08 ± 0.25 vs. 0,78 ± 0.18m/s, p< 0.001) were recorded in pregnant women compared with controls but all were within normal limits as shown in Table II

Table II: Indices of systolic function of subjects and controls

Variables	Subjects (N= 96) MEAN ± SD	Controls (N= 100) MEAN ± SD	p-value
Fractional Shortening (%)	36.4 ± 5.1	31.5 ± 6.1	<0.001*
Ejection Fraction (%)	65.1 ± 7.3	61.4 ± 10.1	0.015*
Velocity of circumferential shortening (m/s)	1.08 ± 0.25	0.78 ± 0.18	<0.001*

Key: **SD**= standard deviation, * statistical significance between all pregnant women and all non-pregnant women. * Statistical significance between all pregnant women and all non-pregnant women.

All the indices demonstrated higher systolic function beginning in the first trimester with a progressive rise throughout pregnancy. The increase was remarkable between the second and third trimesters. (Table III)

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Table III: ANOVA table comparing the indices of systolic function among subjects in each trimester and Controls

Variables	**T1** (N=24) Mean ± SD	**T2** (N= 39) Mean ± SD	**T3** (N= 33) Mean ± SD	**Control** (C) (N=100) Mean ± SD	p-value	Post Hoc Test§
Fractional Shortening (%)	34.0 ± 3.6	36.0 ± 5.4	38.2 ± 5.1	31.5 ± 6.1	< 0.001*	T2 > C T3 > C T3 > T1
Ejection Fraction (%)	63.0 ± 4.8	63.9 ± 7.5	67.4 ± 8.0	61.4 ± 10.1	0.019*	T3 > C
Velocity of circumferential shortening (m/s)	1.02 ± 0.2	1.07 ± 0.31	1.13 ± 0.2	0.78 ± 0.18	< 0.001*	T1 > C T2 > C T3 > C

Key: **SD**= standard deviation, * statistical significance between all pregnant women and all non-pregnant women. T1= first trimester, T2= second trimester, T3= third trimester. * Statistical significance among pregnant women in each trimester and controls. § Post hoc test with Bonferroni's correction

There was a higher maximum mitral e-velocity in the first trimester which rises further in the second trimester before a decline in the third trimester. The mean e-velocity was higher in pregnant women compared with controls (0.99 ± 0.17 vs. 0.21m/s, p= 0.005) both however remained within normal limits as shown in Table IV.

Table IV: Parameters of Diastolic function in subjects and controls

Variables	Subjects (N= 96) Mean ± SD	Controls (N= 100) Mean ± SD	p-value
Mitral E-velocity (m/s)	0.99 ± 0.17	0.89 ± 0.21	0.005*
Mitral A-velocity (m/s)	0.61 ± 0.08	0.55 ± 0.08	<0.001*
Mitral E/A ratio	1.59 ± 0.35	1.61 ± 0.21	0.663
IVRT (ms)	81.4 ± 9.3	88.1 ± 8.4	0.004*
DT (ms)	187.1 ± 32.6	167.2 ± 50.2	0.088
Tricuspid E-velocity(m/s)	0.65 ± 0.13	0.59 ± 0.13	0.011*
Tricuspid A-velocity(m/s)	0.61 ± 0.19	0.41 ± 0.12	<0.001*
Tricuspid E/A ratio	1.13 ± 0.33	1.57 ± 0.58	<0.001*

Key: **SD**= standard deviation. *= statistical significance between all subjects and all controls. T1= first trimester, T2= second trimester, T3= third trimester. **E** = early filling, **A**= Atrial contraction. * Statistical significance among pregnant women in each trimester and controls.

There was a significant increase in the left ventricular diastolic diameter (43.7 ± 5.9 vs. 40.8 ± 3.7mm, p< 0.001), interventricular septal wall thickness in diastole (9.9 ± 1.2 vs. 9.4 ± 1.1mm, p= 0.011), left atrial diameter (36.4 ± 5.2 vs. 31.6 ± 5.2mm, p< 0.001) and left ventricular mass index (96.6 ± 13.8 vs 83.4 ± 13.8g/m^2, p<0.001*) (Table V).

Table V: Echocardiographic Structural parameters of subjects and controls

Variables	Subjects (N= 96) Mean ± SD	Controls (N= 100) Mean ± SD	p- values
LVDD (mm)	43.7 ± 5.9	40.8 ± 3.7	<0.001*
LVSD (mm)	28.1 ± 3.2	28.1 ± 3.0	0.908
IVSTd (mm)	9.9 ± 1.2	9.4 ± 1.1	0.011*
PWTd (mm)	10.7 ± 1.7	10.7 ± 1.7	0.854

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RVD (mm)	21.8 ± 3.8	20.2 ± 3.7	0.193
LAD (mm)	36.4 ± 5.2	31.6 ± 5.2	<0.001*
LVM (g)	127.5	127.5 ± 28.3	<0.001*
LVMi (g/m^2)	96.6 ± 13.8	83.4 ± 13.8	<0.001*

Key: **SD**= standard deviation, * statistical significance between all pregnant women and all non-pregnant women. LVDD = left ventricular diastolic diameter. LVSD= left ventricular systolic diameter. IVSTd= interventricular septal wall thickness in diastole. PWTd= posterior wall thickness in diastole. RVD= right ventricular diameter. LAD= left atrial diameter. LVMi= left ventricular mass index.

The correlation analysis between the gestational age and echocardiographic indices revealed a statistically significant positive correlation with left ventricular mass index, LVMI (r= 0.457, P< 0.001). There was also a positive correlation with cardiac output, CO (r=0.260, p< 0.001), fractional shortening (r= 0.445, p< 0.001), ejection fraction (r= 0.311, p<0.001), velocity of circumferential fibre shortening (r= 0.514, p< 0.001) as well as the mitral e-velocity (r= 0.178, p< 0.001). There was however a negative correlation with mean arterial pressure (r= -0.185, p< 0.05) and systemic vascular resistance (r= -0.314, p< 0.05). However, there was no significant correlation with mitral a-velocity (r= 0.136, p= 0.114) and mitral E/A ratio (r= 0.133, p= 0.189) as shown in Table VI.The correlation analysis between the parity and echocardiographic indices revealed a statistically significant positive correlation with systemic vascular resistance (r= 0.194, p= 0.037) and left ventricular mass index, LVMI (r= 0.306, p< 001). There was however no correlation with other indices.

Table VI: Correlation between demographic and echocardiography parameters of subjects

	Gestational Age	Parity
Variables	r (p-value)	r (p-value)
MAP (mmHg)	-0.186 (0.031)*	-0.011 (0.892)
SVR (dyn x sec/cm^5)	-0.314 (0.001)*	0.194 (0.037)*
CO (L/min)	0.260 (0.006)*	-0.080 (0.398)
FS (%)	0.445 (<0.001)*	0.093 (0.268)
EF (%)	0.311 (<0.001)*	0.038 (0.649)
Vcf (m/s)	0.514 (<0.001)*	0.052 (0.566)
E –velocity (m/s)	0.178 (0.037)*	0.297 (<0.001)*
A –velocity (m/s)	0.136 (0.114)	0.067 (0.424)
E/A ratio	-0.113 (0.189)	0.075 (0.373)
LVMi (g/m^2)	0.457 (<0.001)*	0.306 (<0.001)*

P-value = 2 tailed significance; r= Pearson correlation coefficient; MAP= mean arterial pressure; SVR= systemic vascular resistance; CO= cardiac output; FS= fractional shortening; EF= ejection fraction; Vcf= velocity of circumferential fibre shortening; LVMi= left ventricular mass index

IV. DISCUSSION

There are fewer women in the first trimester because pregnant women do not usually come for antenatal care early enough in this study area. The mean age was similar between the pregnant women and controls. The age at first pregnancy was higher in the controls possibly because more had tertiary education which was likely to have delayed the age at marriage and child delivery.

This study demonstrated an increase in left ventricular systolic function assessed by ejection fraction and fractional shortening in pregnancy, representing a progressive rise from the first to the third trimester. This finding is similar to the report of Clapp and Capeless[14] who also found an increase in the ejection fraction up to the third trimester. However, unlike Clapp and Capeless whose work found a non-significant increase in the third trimester over the second, this study found a significant increase in the ejection fraction.This increase in left ventricular systolic function measured by the Teicholz formula could be a result of the Starling effect on left ventricular myofibers due to an increase in preload from the increase in the blood volume associated with pregnancy. Other hormonal changes could also be responsible for

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possible changes in left ventricular function during pregnancy.

However, the geometric assumptions made in the Teicholz formula of the left ventricles being an ellipsoid make it unreliable.[15] Furthermore, the left ventricular ellipsoid assumption will also not be valid in cases of ventricular dilatation[16] such as in pregnancy when there is an enormous increase in blood volume. Another shortcoming of the traditional indices is that the cursor-derived systolic and diastolic dimensions of the LV from the parasternal view are not from the same part of the LV because of the movement of the heart along the long axis during the cardiac cycle.

In pregnancy, calculation of the ejection fraction by two-dimensional echocardiography might also be limited because for the estimation of the ventricular systolic and diastolic dimensions good image quality is required for adequate tracing of the endocardial borders. This might be compromised, especially towards term, because the soft tissue edema and the change in position of the heart due to the pressure from the gravid uterus make adequate visualization of endocardial borders difficult.[15] Other indices of LV systolic function are needed in pregnancy in addition to the traditional Teicholz method.Some authors[5]are now considering the use of left ventricular long-axis displacement as an index of systolic function which is independent of the changes in LV geometry

The diastolic function in this study was assessed by the transmitral flow pattern which showed an overall increase in both e-wave and a-wave velocities in pregnant women compared with non-pregnant controls. The pattern of rising is such that there is an initial rise in both e- and a-wave peak velocities with a subsequent decline in both to varying degrees relative to each other. In the first trimester, there is an increase in both e- and a-wave velocities with a higher increase in the e-wave velocity relative to the a-velocity resulting in a significant increase in the first-trimestermitral E/A ratio. The second trimester demonstrated a similar pattern in both e- and a-wave velocities but with a higher a-wave velocity increase resulting in a lower mitral E/A ratio relative to the first trimester. The third trimester shows a similar increase in e- and a-wave velocities over the non-pregnant control but with a higher increase in a wave velocity relative to the e-wave velocity resulting in a much lower mitral E/A ratio than the non-pregnant control. Therefore, there is a progressive decline in the mitral E/A ratio throughout pregnancy.

The pattern of rising in the e-velocity relative to the a-velocity in the first trimester appears to be uniform in almost all the available studies[5,17,] However, there is a discrepancy over the pattern of change subsequently, with some studies showing no change in the e-velocity while others show a progressive increase in the e-velocity until term as in this study.

This study also showed a further rise in the e-velocityduring the second trimester followed by a fall in the third trimester similar to the finding of Bamfo et al.[18] in a longitudinal study of 63 women who had serial echocardiography during pregnancy. This also confirms the longitudinal observations by Valensiseet al.[19] as well as a cross-sectional study byKametaset al.[5] However, Mesa et al.[20]also reported a progressive increase in the e-velocity while Mabie et al.[21]demonstrated no change in the e-velocity after the initial rise in the first trimester. The initial rise in the maximum e-wave velocities in the first and second trimesters may be accounted for by the increase in the venous return in the left atrium (preload) while their subsequent decrease in the third trimester to a fall in the preload in the last trimester.

A relative higher a-velocity may be explained by the rise in afterload in the third trimester and the increase in left ventricular compliance. The increase in the afterload is a result of a rise in mean arterial pressure which is observed in the third trimester of pregnancy as shown in this study. The increase in left ventricular compliance may be consequent upon increasing LVMi which is also demonstrated in this study.

There is a total of 20% and 16% increase in the LV mass and LV mass index respectively in pregnancy. Previous studies' reports on the increase in left ventricular mass are variable being 26% in a longitudinal study of 35 healthy women by Desai et al.[22] and 34% increase in LVMi by Schannwellet al.[23]

This study demonstrated a progressive increase in the left ventricular mass and LV mass index during pregnancy from the first to the third trimester. This is particularly marked in the second trimester with a further increase in the third trimester similar to the findings of Mesa et al.[20]as well as that of Desai et al[22].This observation is generally consistent with the expected physiologic hypertrophy due to the increase in blood volume associated with pregnancy.Robson et al.[24] suggested that this increase is similar to that seen in long-distance runners. Kaz et al.[25] however demonstrated a progressive eccentric left ventricular enlargement related to a decrease in the

ratio of the posterior wall thickness and left ventricular end-diastolic radius.

V. CONCLUSION

Pregnancy may be associated with low diastolic blood pressure (below 60mmHg). Systolic function, using conventional indices such as fractional shortening and ejection fraction is higher in pregnant women compared with controls. The Mitral E/A ratio, an index of diastolic function is lower in pregnant women than in non-pregnant controls and this may indicate an impact on diastolic function in apparently healthy pregnant Nigerians. The left ventricular mass, left ventricular mass index, wall thickness and chamber dimensions are higher in pregnant women compared with controls. The cardiac axis is lower (leftward rotation) in pregnant women compared with non-pregnant controls. Tachycardia left bundle branch block and non-specific intraventricular block tend to occur more commonly in pregnant women than in non-pregnant controls.

Competing Interests

Authors have declared that no competing interests exist.

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