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Full Length Research Paper

Linear regression models for quantitative assessment of left ventricular function and structures using M-mode echocardiography

Okwor, C. A.[1], Ugwu, A. C.[2]* and Eze, J.[3]

[1]Radiology Department, Ebonyi State, University Teaching Hospital, Ebonyi State, Nigeria.
[2]Radiology Department, Federal Medical Centre, P.M.B.102, Abakaliki, Ebonyi State, Nigeria.
[3]Radiology Department, National Orthopedic Hospital Enugu State, Nigeria.

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Changes in left ventricular structures and function have been reported in cardiomyopathies. No prediction models have been established in this environment. This study established regression models for prediction of left ventricular structures in normal subjects. A sample of normal subjects was drawn from a large urban population. Echocardiographic end diastolic diameters, end systolic diameters, posterior wall thicknesses in both systole and diastole, septal wall thicknesses in both systole and diastole were used to calculate left ventricular mass, left ventricular mass index, relative wall thickness and fractional shortening. Heights, weights, ages, and blood pressures of subjects were obtained. Pearson's correlation coefficients were computed. Tests were two tailed with $P < 0.05$ indicating statistical significance. Three hundred and twenty two normal subjects of Ibo descent were enrolled in this study as volunteers between June, 2006 and April, 2007. Correlation coefficients between measured left ventricular structures and functions, and some anthropometric variables were computed. Linear regression models for the prediction of left ventricular structures were established. Prediction models for left ventricular structures have been established and could be useful in assessing morbidity in cardiomyopathies.

Key words: Echocardiography, left ventricle, cardiomyopathy.

INTRODUCTION

Quantitative echocardiography using M-mode provides information on atrial, ventricular and great vessels dimension and function. M-mode measurements continue to be used, although the inherent limits of spatial orientation may be problematic in some subjects. Excellent temporal resolution is the strenght of M-mode. If M-mode images using 2D guidance are obtained in a standard left parasternal window, the measurements are usually accurate.

Doppler-measured parameters such as Bernoulli's equation, pressure half time, and the continuity equation (based on hydrodynamic and hemodynamic principles) have become an integral part of the echocardiography examination (Waggoner, 2005). Some changes in ventricular functions and structures have been noted in cardiomyopathies. Values for systolic left ventricular function (M-mode fractional shortening) were reported to be above normal in subjects with white coat hypertension whereas diastolic filling and left atrial size were similar to those in nor-motension (Muscholl et al., 1998). Left ventricular mass is dependent on values of end diastolic diameter, septal wall thickness and postenor wall thickness (Keil et al., 1988). In left ventricular hypertrophy, values of left ventricular mass are above normal range of values. Relative wall thicknesses were increased in hypertensive subjects (Muscholl et al., 1998).

To the best of our knowledge, prediction models for left

*Corresponding author. E-mail: tonybullng@yahoo.ca. Tel.: + 2438076241297.

ventricular structure and function have not been established in any African population. Echocardiographic evidence of left ventricular hypertrophy is an independent predictor of overall mortality (Levy et al., 1990). This study aimed at establishing linear regression models that could be used in the prediction of clinical outcomes in individuals with cardiomyopathies by comparing predicted and measured values of ventricular structures.

MATERIALS AND METHODS

Subjects' eligibility

Subjects with a history of cardiomyopathy were excluded. Also excluded were subjects with obstructive airway disease, sickle cell disease, pregnancy, immune suppression, and intra-abdominal masses. Sportsmen involved in either isotonic or isometric sporting activities were also excluded due to anatomical changes, which could accompany those activities. Subjects had to be aware of the nature of the study and willingly provide informed consent before entering the study. Ethics board review and approval were obtained at Ebonyi State University Teaching Hospital.

Evaluation

Echocardiography studies were performed with SL I, siemens Medical system, USA Inc, ultrasound Group, Issaquah WA with a 3.5 MHZ sector transducer. Images were obtained in parasternal long and short axis views with the patient either in supine or left postenor oblique position (Reynolds, 1993). M-mode tracing was obtained in the short axis view. To reduce inter observer variability (Muscholl et al., 1998), all M-mode tracings were analyzed by a single imaging scientist (CA). The following cardiac parameters were measured using M-mode of 2D derived image of the heart: (1) left ventricular internal diameter (LVID) in end systole (End systolic diameter – EDD) and in end diastole (End diastolic diameter – EDD). (2) Interventricular septal wall thickness in systole (Septal wall thickness in systoles -SWTS) and in diastole (septal wall thickness in diastole – SWTD). (3) Posterior left ventricular wall thickness on systole (Posterior wall thickness in systole – PWTS and Posterior wall thickness in diastole – PWTD).

FS = [(EDD –ESD)/EDD] x 100 (Reynolds, 1993)

FS is the fractional shortening of the heart. Measurements of left ventricular mass were taken just below the tips of the mitral valve (Muscholl et al., 1998).

Relative wall thickness (RWT) was calculated at end diastole as:

RWT = 2 x (posterior wall thickness/end diastolic diameter)

(Muscholl et al., 1998).

Left ventricular mass was calculated according to the formula of Penn as:

Left ventricular mass (g) = 1.0 4 (end diastolic diameter + septal wall thickness + posterior wall thickness) – (end diastolic diameter) – 13.6 g (Keil et al., 1988).
Left ventricular Mass was indexed to height in g/m (Levy et al., 1987). This would possibly adjust variations in heat size due to differences in body size.

The body weights were measured on a bathroom scale: Model H 89 LT Blue, and height measured on a calibrated vertical wall. The body mass index (BMI) was calculated as weight/height (Muscholl et al., 1998). While the body surface area (BSA) was calculated using the formula derived by DU bois and DU Bois (1916) as:

$BSA = (Weight^{0.425} \times Height^{0.725}) \times 0.007184$

The blood pressure (BP) was measured with a sphygmomanometer and was standardized for cuff size and position. SBP and DBP represent systolic and diastolic blood pressures.

Data analysis

Data were not categorized according to gender, as a previous study (Cavallini et al., 1995) reported no significant relationship between left ventricular Mass Index (LVMI) and gender. It also reported that no significant correlations exist between postenor wall thickness (PWT) and demographic variables. SPSS 11.0 software was used for correlation analysis and developing regression equations. Pearson's correlation coefficients were generated. P values < 0.05 were considered statistically significant.

RESULTS AND DISCUSSION

Three hundred and twenty two normal subjects of Ibo ethnic group were enrolled in this study as volunteers between June, 2006 and April, 2007. Equal numbers of men and women enrolled into the study (161 each). Subject ages ranged from 18 to 73 years with a mean age ± standard deviation of 40.59 ± 16 41 and 39. 39 ± 11.35 in men and women, respectively. Two subjects (a man and of women) had blood pressure > 140/90 and were classified hypertensive (Cavallini et al., 1995). These two subjects were hence excluded from the study and the characteristics of the remaining three hundred and twenty subjects are shown in Table 1.

Table 1 shows Pearson correlation coefficients(r) and their P – values between left ventricular parameters and some anthropometric variables. The following linear regression equations were established:

PWTD (mm) = 0.0378 Age + 7.875
PWTS (mm) = 0.038 Age + 0.0183 Weight + 8.31
SWTS (mm) = 0.03 Age + 7.08
SWTS (mm) = 0.0277 Age + 0.015 Weight + 5.06
ESD (mm) = 0.12 Age + 0.36 BMI + 22.79
EDD (mm) = 0.134 Age + 0.38 BMI + 34.85
LVM (g) = 1.7 Age + 2.8 BMI + 26.84
LVMI (g/m) = 1.1 Age + 1.74 BMI + 16.27

Only significant predictors of cardiac parameters were considered in the development of linear regression models.

As a complement to imaging cardiac structure, quantification of cardiac size, function, and blood flow is an integral part of echocardiography, M-mode, the first echocardiography technique to provide quantitative data,

Table 1. Pearson's correlation coefficients (r) and P-values between lv parameters and some anthropometric variables.

LV parameter	Anthropometric variables						
	Age (years)	**Height (m)**	**Weight (kg)**	**SBP (mmkg)**	**DBP (mmHg)**	**BMI (Kg/m^2)**	**BSA (m^2)**
PWTD	r=0.465 p=0.00(s)	r=0.014 p=0.86(NS)	r=0.132 P=0.10(NS)	r=0.427 P=0.00(S)	r=0.389 P=0.00(S)	r=0.141 p=0.075(NS)	r=0.101 P=0.202(NS)
PWTS	r=0.470 p=0.0(s)	r=0.025 p=0.86(NS)	r=0.158 P=0.046(s)	r=0.490 P=0.00(s)	r=0.404 P=0.00(s)	r=0.170 p=0.031(s)	r=0.126 p=0.112(NS)
SWTS	r=0.423 p=0.00(s)	r=0.074 p=0.35(NS)	r=0.152 P=0.55(NS)	r=0.435 P=.0.0(s)	r=0.318 P=0.00(s)	r=0.119 P=0.133(NS)	r=0.138 P=0.112(NS)
SWTD	r=419 p=0.00(s)	r=0.10 p=0.21(NS)	r=0.16 P=0.045(s)	r=0.453 p=0.00(s)	r=0.374 p=0.00(s)	r=0.109 p=0.171(NS)	r=0.154 P=0.051(NS)
EDD	r=0.373 p=0.0(s)	r=0.204 p=0.10(s)	r=0.327 p=0.0(s)	r=0.368 p=0.00(s)	r=0.422 p=0.00(s)	r=0.249 p=0.002(s)	r=0.322 p=0.00(s)
ESD	r=0.358 p=0.0(s)	r=0.224 p=0.004(s)	r=0.336 p=0.00	r=0.295 p=0.00(s)	r=0.323 p=0.00(s)	r=0.25 p=0.001(s)	r=0.336 p=0.00(s)
LVMI	r=0.499 p=0.00(s)	r=0.018 p=0.82(NS)	r=0.185 p=0.019(s)	r=0.0457 p=0.00(s)	r=0.457 p=0.00(s)	r=0.202 p=0.01(s)	r=0.144 p=0.07(NS)
LVM	r=0.48 p=0.00(s)	r=0.161 p=0.04(NS)	r=0.267 p=0.001(s)	r=0.488 p=0.00(s)	r=0.481 p=0.00(s)	r=0.201 p=0.11(s)	r=0.258 p=0.001(s)
RWT	r=0.134 p=0.09(NS)	r=0.155 p=0.05(NS)	r=-0.155 p=0.052(NS)	r=0.102 p=0.197(NS)	r=0.02 p=0.81(NS)	r=-0.083 p=30(NS)	r=-0.176 p=0.026(s)
SF	r=-0.093 p=24(NS)	r=-0.109 p=0.17(NS)	r=-0.125 p=0.117(NS)	r=-0.017 p=0.83(NS)	r=-0.017 p=0.83(NS)	r=-0.82 p=0.30(NS)	r=-0.133 p=0.093(NS)

NS = Not significant; S = significant.
Correlation is significant at the 0.05 level (2 –tailed).

continues to be used for a variety of measurements. Echocardiography has been used as a gold standard in the electrocardiography diagnosis of right ventricular hypertrophy in children (Puchalaski et al., 2006).

The present study (Table 1) confirms previous findings that SBP is modestly related to LVMI (Savage et al., 1979) and that BMI was a significant predictor of LVMI (Sherwood et al., 2002). This study is also in agreement with a previous study (Henry et al., 1978) which reported that left ventricular mass varied linearly with the direct measurement of body surface area and that percent fractional shortening of the left ventricle is independent of BSA. In the healthy young adults of CARDIA (coronary artery risk development in young adults) cohort, LVM was highly correlated with body weight, sub scapula skin fold thickness, height, and systolic blood pressure across race and sex subgroups (Gardin et al., 1995). Furthermore, after adjustment for anthropometrics, BP, and other covariates, LVM remained higher in men than in women and in blacks than in whites. This report partly agrees with this present study, which shows that LVM did not significantly correlate with height but significantly correlated with weight and systolic blood pressure.

Julius et al. (1989) used hindquarter compression in dogs to elicit transient sympathetically mediated increases in BP for 6 h per day. After 9 weeks of this manipulation, there was no persistent elevation in BP, but a 28% increase in LVM was observed. For patients with hypertension, higher ambulatory BP variability has been found to be associated with increased LVM, independent of mean ambulatory BP levels (Parati et al., 1987). These observations are consistent with the findings of this study which showed significant positive correlations between BP parameters (in both diastole and systole), and LVM. Similar to a previous study (Sherwood et al., 2002), posterior wall thickness in this study correlated with BP. In this study, no significant correlation was recorded between relative wall thickness (RWT) and BP unlike it is with LVM and LVMI. Based on the fact that RWT is increased in hypertensive groups (Muscholl et al., 1998), it may be more potent than LVM and LVMI in the assessment of morbidity in hypertension. Fractional shortening was enhanced in subjects with white coat hypertension (Muscholl et al., 1998). No significant correlation identified in this study (normal subjects) between fractional shortening and BP could also be a pointer to high sensitivity of fractional shortening in the assessment of morbidity in hypertensive cardiopathy. The positive relationship between septal wall thicknesses and BP in this study agrees with a previous study (Muscholl et al.,

1998), which showed an increase in left ventricular septal wall thickness in white coat hypertension. The internal dimensions of the ventricle (EDD and ESD) correlated significantly with body surface area. This is similar to a previous study (Henry et al., 1978), which showed that internal dimensions of left ventricle varied in a linear relation to the cube root of the body surface area.

To the best of our knowledge, this is the first time prediction equations for the estimation of cardiac parameters are being established in this locality. Longitudinal studies are necessary to delineate the possible role of anthropometric variables in the genesis of LV hypertrophy. Replication of this study in a larger samples, age and gender categorized samples in other ethnic/racial groups within Africa and beyond is suggested. In this study, all the measurements were taken by one imaging scientist (CA). This was done to avoid possible interobserver errors as it has not been previously investigated that these measurements could be collected and reported equally as consistent between scientists. This study has established prediction formulas for cardiac structures upon which comparisons of measured values could be made in the assessment of cardiomyopathies.

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