Original Article

# Prevalence of Aortic Root Dilation and Correlates of Aortic Root Dimensions in Treatment Naïve Hypertensive Patients in North-Eastern Nigeria

**\*Ibrahim Abubakar Galtimari[1], Buba Faruk[1,2], Oyati Imhoagene-Albert[3,4],
Mohammed Abdullahi Talle[1,2], Charles Oladele Anjorin[1]**

[1]Cardiology Unit, Department of Medicine, University of Maiduguri Teaching Hospital, Maiduguri, Nigeria.
[2]Department of Medicine, Faculty of Clinical Sciences, College of Medical Sciences, University of Maiduguri, Nigeria. [3]Department of Medicine, Faculty of Clinical Sciences, College of Medical Sciences, Ahmadu Bello University, Zaria, Nigeria. [4]Department of Medicine, Ahmadu Bello University Teaching Hospital, Shika, Nigeria.

## Abstract

**Background:** Systemic arterial hypertension is an independent predictor of cardiovascular diseases including aortic root dilation. Aortic root dilation is a hypertension-mediated organ damage entity regardless of age, gender, and body size. This study aims at determining the prevalence and associations of aortic root dilatation among treatment naïve hypertensive patients.

**Methodology:** The study was a cross-sectional study conducted at the University of Maiduguri Teaching Hospital from June 2019 to June 2021. Study participants were treatment naïve hypertensive patients recruited consecutively from the outpatient clinic of the hospital and normotensive sex- and age-matched controls. End diastolic aortic root diameter was measured using 2D transthoracic echocardiography at the annulus, sinuses of Valsalva, and Sinotubular junction by leading-edge to a leading-edge convention as recommended by the American Society of Echocardiography in parasternal long-axis view.

**Results:** Three hundred treatment naïve hypertensive patients (39.0% females) and 300 health normotensive age-and sex-matched controls (38.3% females) were enrolled. The mean ages of the patients and controls were 46.4±12.5 years and 46.4 ± 12.4 years respectively. The mean aortic root diameter (in mm) was significantly higher in the treatment naïve hypertensive arm of the study at the levels [AoA (24.7 ± 3.9mm versus 22.5 ± 2.0mm, $p = 0.002$), SoV (33.1 ± 3.4mm versus 31.4 ± 3.4mm, $p = 0.023$), and STJ (27.8 ± 3.5mm versus 25.9 ± 2.2mm, $p = 0.002$)]. Males had larger absolute aortic root diameters than females, however, after indexing aortic root diameters for BSA, there was no significant difference. The prevalence of aortic root dilatation amongst the treatment naïve hypertensive patients was 1.3% at all considered levels.

**Conclusion:** The aortic root dimensions in the treatment naïve hypertensive patients were larger than in normotensive adults. The prevalence of aortic root dilation at all levels amongst treatment naïve hypertensive patients is 1.3%.

**Keywords:** 2D Echocardiography; Treatment Naïve Hypertensive; Aortic Root Dimension; Aortic Annulus; Sinuses of Valsalva; Sinotubular Junction; Left Ventricular Mass.

**Corresponding Author:** *Ibrahim Abubakar Galtimari, [1]Cardiology Unit, Department of Medicine, University of Maiduguri Teaching Hospital, Maiduguri, Nigeria. dr.galtimari@gmail.com.

**Quick Response Code:**

## Introduction

The World Health Organisation (WHO) estimated that about 46% of adults aged 25 years and older Africans are affected by hypertension, compared to 35% to 40% in the other continents. Many hypertensive people in Africa do not know their status and are rarely treated or poorly controlled, making them at the highest risk of end-organ damage notably, stroke, heart and kidney diseases[1].

The aortic root is comprised of the annulus of the aorta (AoA), the sinuses of Valsalva (SoV) and the Sinotubular junction (STJ)[2]. Aortic root dimensions are directly related to sex, age, weight and height (Body Mass Index and Body Surface Area) in the general population[3].Hypertension is regarded as a cause of aortic root dilation. However, this assertion generates controversies and debates amongresearchers[4–6]

The prevalence of Aortic root dilation (ARD) is high (16.7%) among western population hypertensive patients, suggesting ARD is common among hypertensive patients[7]. Aortic root dilatation is strongly associated with the presence and severity of aortic regurgitation and increased risk for aortic dissection.[4]Aortic root diameters at the annulus and sinuses of Valsalva tend to be higher in hypertensive individuals[5,8–10].

The aortic root dilation and systemic arterial hypertension are usually asymptomatic and found incidentally on routine imaging studies such as chest radiograph, echocardiography, chest computed tomography, magnetic resonance imaging and routine clinic evaluation respectively. Transthoracic echocardiography has good specificity and sensitivity in the evaluation of the aortic roots[11].

ARD may be the earliest vascular abnormality to occur in hypertension. Aortic root dilation poses catastrophic complications such as aortic dissection, aortic rupture, and congestive heart failure from aortic insufficiency. Therefore, this study aims at determining the prevalence and correlates of aortic root dimensions among treatment naïve hypertensive patients in north-eastern Nigeria.

## Methodology

It is a hospital-based, cross-sectional, observational study. The study population was made of 300 consecutive treatment naïve hypertensive adult patients aged 18 years and above that presented to the Cardiology Clinic and emergency room of the University of Maiduguri Teaching Hospital (UMTH) from June 2019 to June 2021. 300 Healthy age-and sex-matched non-hypertensive adults comprising inpatients' relatives, medical students, porters, security personnel and other hospital staff were enrolled to serve as a control group.

Subjects' inclusion criteria for the hypertensive patients includes adult of age 18 years and above, with average Office Blood Pressure (OBP) ≥ 140/90mmHg and for the non-hypertensive control include adult aged 18 years and above, average Office Blood Pressure (OBP) of <140/90mmHg. The exclusion criteria include hypertensive patients on antihypertensive drugs, valvular heart diseases and clinical characteristics suggesting a genetic predisposition to an aortic disease such as Marfan syndrome, and chest or sternum deformity, eGFR of less than 60ml\min\1.72m2, suspected aortic dissection and Diabetic Mellitus as well as poor echocardiographic image qualities.

### Biodata, anthropometric and clinical details

A well-structured proforma was used to collect data from eligible patients after obtaining written, informed consent and ethical approval from the Health Research and Ethics committee of the UMTH. Those who refused to give informed consent were not recruited and they did not suffer in any way in the process of their treatment. Information obtained using the proforma included sociodemographic parameters and anthropometric parameters. The Height and weight were measured using Health Care Scale Adult with Height Measurement Standard (Model: RGZ-160) and the BMI and the BSA were derived from the measurements[12,13].

The radial pulse was palpated at the wrist against the radial bone while the subject was in relaxed condition and the rate was counted over 60 seconds to determine the number of beats/minutes. Office BP measurements were performed using Accoson®sphygmomanometer and stethoscope. The BP was measured while the patients were in a sitting position, the arm at the level of the heart using an appropriate-size cuff; cuff length and width of 80% and 40% of arm circumference, respectively, deflating the cuff at ≤ 2 mm Hg/sec. The systolic and diastolic blood pressure were determined using the first and fifth Korokoff sounds. Three different BPs were taken about five minutes apart and the average was used. Pulse pressure, (PP) and mean arteria lBP, (MABP) were calculated from the DBP and SBP as follows:

PP = SBP— DBP, MABP= *PP/3 + DBP*. Blood glucose was measured from the capillary blood at the tip of the fingers using Glucometer (one touch ultramini ACCU-CHEK®Aviva). Blood urea nitrogen, and creatinine were analysed in the central chemical pathology laboratory using auto-analyser model cobas 311 analyser (F. Hoffman-La Roche Ltd).

## Echocardiography

Echocardiographic examinations were performed using KT-LM 200HDPE (SeimensAcusonX300, Seimen medical solution, USA) ultrasound systems equipped with the appropriate two-dimensional transthoracic probe. Participants were evaluated in the left lateral decubitus position and images were acquired from standard parasternal, suprasternal, and apical windows using second-harmonic two-dimensional imaging. Care was taken to acquire images displaying the largest aortic lumen, and the acquisition was done during breath-hold to minimize translational movements.

The diameters of the aortic root were measured using the leading edge–to–leading-edge convention at end-diastole as recommended by the American Society of Echocardiography in parasternal long-axis view[14].The following sites were considered (1)the annulus of the aortic valve (AoA); (2) the sinuses of Valsalva (SoV); (3) the Sinotubular junction (STJ). The aortic root diameters were indexed to BSA using the formula Aortic root diameter/BSA. The Roman's nomogram of BSA-indexed aortic root diameter was used to determine the upper limit of normal aortic root diameters (Aortic annulus (AoA) ≥ 14mm/m$^2$, Sinuses of Valsalva (SoV) ≥ 21mm/m$^2$ and Sinotubular junction (STJ) ≥ 19mm/m$^2$)[2].

Using M mode, the left ventricular dimensions, interventricular septal thickness (IVST), and left ventricular posterior wall thickness (LVPWT) were measured in the parasternal long axis (PSLAX) window. These were used to determine left ventricular mass (LVM) using the Devereux regression formula[15].TheLVMI of greater than 115 g/m2 and greater than 95g/m2 were considered as left ventricularhypertrophy (LVH) for males and females respectively[14].

## Data Analysis

Statistical analysis was carried out using the Statistical Package for Social Science (SPSS) software version 23 (Chicago, Illinois, USA). The continuous variables which include age, anthropometric measurements, BP indices, pulse rate, serum creatinine, blood urea nitrogen, serum lipids and echocardiographic measurements were normally distributed and were expressed as mean ± SD. The student's t-test was used to compare the mean± SD of the treatment naïve hypertensive patients and normotensive controls to determine statistical significance and a *p*-value of ≤ 0.05 was considered significant. The categorical variables were expressed as absolute values and percentages. Chi-square ($\chi^2$) was used to determine the significant difference in the frequencies of categorical variables of the treatment naïve hypertensive patient and the non-hypertensive controls. A *p*-value of ≤ 0.05 was considered significant. The relationships of aortic root measurements with blood pressure, age and echocardiographic and anthropometric data were analysed using the Pearson correlation coefficient (*r*) and a *p*-value of ≤ 0.05 was considered as statistically significant the prevalence of ARD at all levels and the individual levels were expressed as fractions and percentages.

## Results

From June 2019 through June 2021, a total of six hundred and eighty-one (681) participants were assessed for enrolment into the study, three hundred and fifty-six (356) treatment naïve hypertensive patients and three hundred and twenty-five (325) non-hypertensive controls. Fifty-six (56) treatment naïve hypertensive patients and twenty-five (25) non-hypertensive participants were excluded from the study. Forty (40) treatment naïve hypertensive patients and twenty-three (23) non-hypertensive controls had poor quality echocardiographic images while ten (10) treatment naïve hypertensive patients had renal impairment with eGFR < 60ml/min/1.73m$^2$ and six (6) treatment naïve hypertensive patients and two (2) non-hypertensive controls were diabetic (figure 1).

Table 1 showed three hundred (300) subjects and three (300) controls were recruited into the study comprising 117(39%) and 115 (38.3%) females, 183 (61%) and 185 (61.7% ) males in the treatment naïve hypertensive patients and the non-hypertensive controls respectively. There were no significant differences in the mean ± SD ages between the treatment naïve hypertensive patients and the non-hypertensive (46.4±12.4 versus 46.4±12.5, $p$ = 0.95).The mean ± SD of height showed no significant difference between treatment-naïve hypertensive patients and non-hypertensive controls (1.67 ± 0.06 versus 1.68 ± 0.07, $p$ = 0.78).On the other hand, the treatment naïve hypertensive group had significantly higher BMI (26.3 ±5.1 versus 24.6±3.8, $p$ < 0.001) and BSA (1.83 ± 0.18 versus 1.78 ± 0.15, $p$ < 0.001) than the control. The mean ± SD of the serum creatinine demonstrated no significant difference between the treatment naïve hypertensive patients and the non-hypertensive controls (85.3±18.5 versus 83.1±16.2, $p$ = 0.142). There was no significant difference in blood urea nitrogen between the treatment naïve hypertensive group and the controls (6.0±2.1 versus5.9±1.5, $p$ = 0.559) and the same was observed for random blood glucose (RBG) (6.3±1.6versus 6.2±1.6, $p$ = 0.809).

The mean ± SD systolic blood pressure was significantly higher in the newly diagnosed hypertensive patients (155.7 ± 16.6 versus 118.2 ± 9.3, $p$ < 0.001), a similar pattern was observed in the diastolic blood pressure (98.3 ± 10.8 versus 77.8 ± 6.9, $p$ < 0.001), the pulse pressure (PP) (57.3 ± 11.1 versus 40.4 ± 7.1, $p$ < 0.001) and the mean arterial blood pressure (MABP)(117.5 ± 11.9 versus 91.3 ± 7.0, $p$ < 0.001). However, there was no significant difference between the newly diagnosed hypertensive group and the non-hypertensive group in the mean± SD pulse rate (PR) (85.0 ± 13.4 versus 82.8 ± 9.1, $p$ = 0.467) as described in Table I

**Echocardiographic characteristics**
Tables 2 the means of the left ventricular internal diameter in diastole (LVIDd) showed no statistical difference between the treatment naïve hypertensive group and non-hypertensive adults (47.8 ± 6.8 versus 46.7 ± 4.1, p = 0.37). A similar pattern was observed in the left ventricular internal diameter in systole (LVIDs) (31.8 ± 6.9 versus30.2 ± 4.2, p = 0.12), end-diastolic volume (EDV) (116.9 ± 49.4 versus 108 ± 26.8, p = 0.23),the end-systolic volume (ESV) (37.9 ± 22.7 versus 32.4 ± 10.3, p = 0.061), the stroke volume(SV) (78.1 ± 34.1 versus 76.2 ± 18.6 p = 0.62), the left ventricular ejection fraction (LVEF)(67.7 ± 7.7 versus 69.9 ± 4.7, p = 0.22) and fraction shortening (FS) (34.3 ± 7.9 versus 34.4 ±3.3, p = 0.86).The mean± SD LVM was significantly higher in the treatment naïve hypertensive group than non-hypertensive group (188.9 ± 64.4 versus 164.2 ± 36.8, p = 0.004), the LVMI showed a similar pattern (102.8 ± 31.7 versus 93.1 ± 23.1, p = 0.016). Left ventricular hypertrophy (LVH) was observed in 164 (54.7%) of the treatment naïve hypertensive patients while 32 (10.7%) of the non-hypertensive control with significant difference (p < 0.001).

**The comparison of the AR dimension between treatment naïve hypertensive patients and normotensive controls**
Table 3 showed that the mean aortic root diameter was significantly larger in the treatment naïve hypertensive arm of the study at the levels [AoA (24.7 ± 3.9 versus 22.5 ± 2.0, p = 0.002),SoV (33.1 ± 3.4 versus 31.4 ± 3.4, p = 0.023), and STJ (27.8 ± 3.5 versus 25.9 ± 2.2, p =0.002)]. The AoA/BSA and the SoV/BSA were significantly larger among the treatment naïve hypertensive patients [AoA/BSA (14.2 ± 2.6

versus 12.8 ± 1.5, p < 0.001) SoV/BSA (18.9 ±2.6 versus 17.9 ± 2.2, p < 0.001)]. However, the STJ/BSA did not reveal any statistical significance between the two groups (15.4 ± 2.8 versus 15.1 ± 2.3, p = 0.068).

**Prevalence of aortic root dilation**
We found a prevalence of (1.3%) of the aortic root dilation at all three levels in the population of the treatment naïve hypertensive group while 0(0%) of the non-hypertensive control arm though the difference was not statically significant. The prevalence of aortic annular (AoA) dilation was 13% in the newly diagnosed hypertensive patients and 2.7% in the non-hypertensive control which was statistically significant. The prevalence of the SoV dilation was 23(7.7%) in the hypertensive group against 8(2.7%) in the non-hypertensive control arm of the study which was statically significant. Furthermore, the prevalence of sinoatrial junction (STJ) dilation was 4(1.3%) in the newly diagnosed hypertensive group versus 0(0%) in the non-hypertensive controls with no significant difference.

**Aortic root dimensions by gender**
Tables 4 shows the treatment naïve hypertensive males had a significantly larger AR diameter than their females' counterparts at the level of AoA (27.1±3.9 versus 23.2±3.7, p < 0.001), SoV (35.4±2.8 versus 33.4±4.0, p < 0.001) and STJ (28.9±3.3 versus 27.0±3.5, p < 0.001). Similarly, the males had significantly larger AR among the non-hypertensive group at the level of AoA (23.4±1.8 versus 22.2±2.2, p < 0.001), SoV (32.5±2.5 versus 31.3±3.2 p < 0.001 and STJ (26.6±1.8 versus 24.9±3.0, p < 0.001).The BSA-indexed aortic root diameter at the level of the AoA, the SoV and the STJ of the males among the treatment naïve hypertensive patients were not significantly different from the females [ AoA (14.7±2.6 versus 14.3±2.6, p = 0.18), SoV (19.3±2.3 versus 19.0±3.1, p = 0.38) STJ (15.7±2.5 versus 15.4±2.8, p = 0.34)]. Similarly, BSA-indexed AoA, SoV and STJ diameters of males among the non-hypertensive controls were not significantly larger than their female counterparts [ AoA (13.0±1.5 versus 13.0±1.6 p = 0.89), SoV (18.1±2.1 versus 18.3±2.0 p = 0.29), STJ (14.8±1.6 versus 14.6±2.0, p = 0.51)].

**Aortic root dimensions by ages ≥ 50 years and <50years**
There were 118 and 119 treatment naïve hypertensive patients and normotensive individuals aged greater than or equal to 50 years, respectively. While 182 treatment naïve patients and 181 normotensive individuals were less than 50 years of age, respectively. Treatment naïve hypertensive patients who were 50 years and older had significantly larger AoA diameter than those who were less than 50 years of age (24.7±2.5 versus 22.8±2.9, p = 0.001). The SoV diameter was significantly larger in the treatment naïve hypertensive that was 50 years and older compared to the younger treatment naïve hypertensive patients (34.5±2.2 versus 32.9±3.4, p = 0.046) and the STJ diameter was significantly larger in newly diagnosed hypertensive patients aged greater than or equal to 50 years as compared to the younger hypertensive patients (28.2±2.3 versus 27.2±2.8, p = 0.050).
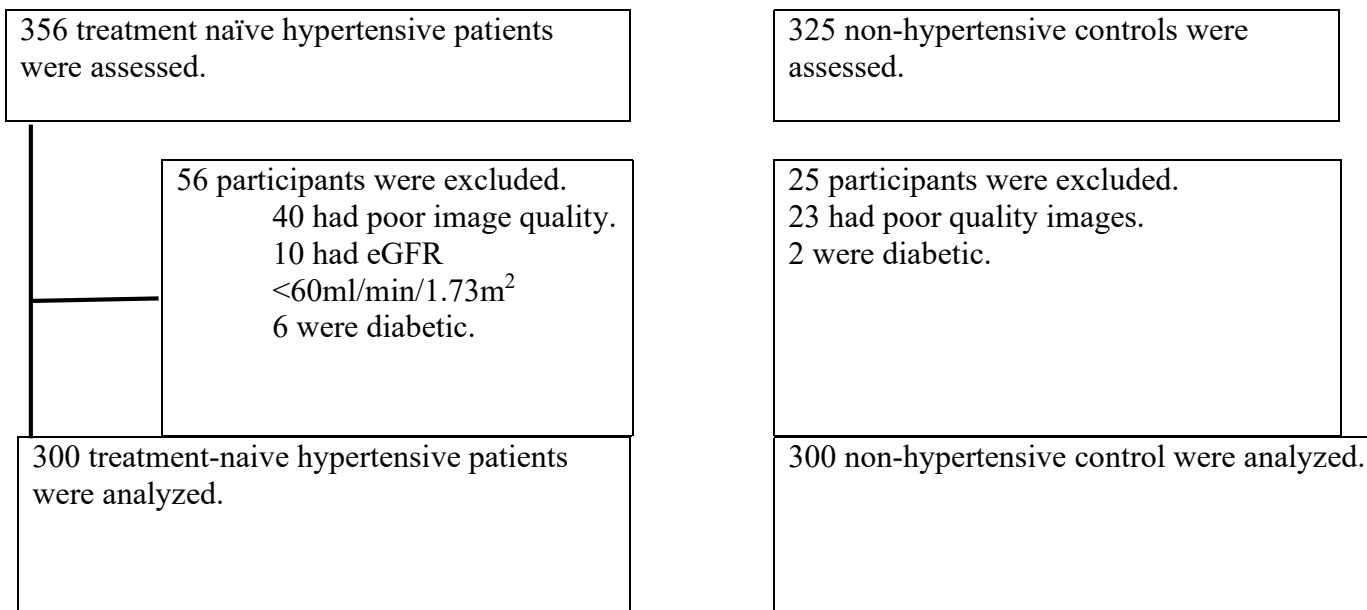
In the non-hypertensive control group, those aged 50 years and above had significantly larger AoA diameter (22.5±2.3 versus 23.5±1.9, p = 0.025), SoV diameter (32.3 ±2.6 versus 31.9 ±2.8, p = 0.046) and STJ diameter (27.4±0.4 versus 26.9±2.7, p = 0.034) compared to the normotensive younger individuals. Similarly, BSA-indexed SoV and STJ diameters of those ≥ 50 years among the non-hypertensive controls were significantly larger compared to the individuals who were less than 50 years of age: SoV (19.4 ±0.8 versus 18.0±1.0, p = 0.003) and STJ (17.2 ±0.5 versus 15.2±1.4, P = 0.002).

**Correlation of aortic root dimension at various levels with age, clinical characteristic, echocardiographic variables and serum lipid profile**
In table VI, all three levels of aortic roots measurements showed similar patterns, progressive enlargement with age [AoA (*r* = 0.413, 0.171, *p* < 0.001), SoV (*r* = 0.211, *p* = 0.004) and STJ (*r* = 0.269, *p* < 0.001)]. The study also demonstrated that the aortic sinuses of Valsalva (SoV) diameter showed a positive correlation with SBP (*r* = 0.193, *p* = 0.009), DBP (*r* = 0.188, *p* = 0.011), and MABP (*r* = 0.226, *p* = 0.002). Similarly, the AoA diameter positive correlation with DBP (*r* = 0.383, *p* < 0.001).

Of the body size parameters, height had a positive correlation between the aortic diameter at all levels [AoA ($r = 0.386$, $p < 0.001$), SoV ($r = 0.411$, $p < 0.001$) and STJ ($r = 0.317$, $p < 0.001$)] and the weight [AoA ($r = 0.215$, $p < 0.001$), SoV ($r = 0.352$, $p <0.001$) and STJ ($0.250$, $p < 0.00$). The body surface area (BSA) had a positive correlation with the aortic AoA (r = 0.239, p = 0.001), SoV diameter ($r = 0.453$, $p < 0.001$) and the STJ ($r = 0.317$, $p < 0.001$). while body mass index (BMI) showed no correlation with the aortic root at all levels [AoA ($r = 0.015$, $p = 0.844$), SoV ($r = 0.121$, $p = 0.102$) and STJ ($r = 0.062$, $p = 0.401$)].

Furthermore, left ventricular mass (LVM) showed a positive correlation with aortic root diameter at all levels [AoA ($r = 0.220$, $p = 0.003$, SoV ($r = 0.572$, $p < 0.001$) and STJ ($r = 0.451$, $p < 0.001$)]. Similarly, the left ventricular mass index (LVMI) demonstrated a positive correlation with the aortic root (AR) diameter at all levels [AoA ($r = 0.232$, $p = 0.002$, SoV ($r = 0.446$, $p < 0.001$) and STJ ($r = 0.381$, $p < 0.001$)]. The E/A ratio had a negative correlation with aortic root diameter at all levels [AoA ($r = -0.308$, $p < 0.001$, SoV ($r = -0.393$, $p < 0.001$) and STJ ($r = -0.429$, $p < 0.001$)] (figures 4.6, 4.7 and 4.8).

| 356 treatment naïve hypertensive patients were assessed. | 325 non-hypertensive controls were assessed. |
|---|---|
| 56 participants were excluded. 40 had poor image quality. 10 had eGFR <60ml/min/1.73m$^2$ 6 were diabetic. | 25 participants were excluded. 23 had poor quality images. 2 were diabetic. |
| 300 treatment-naive hypertensive patients were analyzed. | 300 non-hypertensive control were analyzed. |

**Figure 1**: Assessment and recruitment of treatment naïve hypertensive patients and non-hypertensive controls

**Table 1:** Demographic and clinical characteristics of treatment naïve hypertensive patients and the non-hypertensive controls

| Characteristics | Treatment naïve hypertensive patients (n = 300) | Non hypertensive Controls (n =300) | P |
|---|---|---|---|
| Female –no % | 117(39.0%) | 115(38.3%) | |
| Male –n% | (183) 61% | (185) 61.7% | |
| Age(yr) | 46.4±12.5 | 46.4 ± 12.4 | 0.95 |
| Weight (kg) | 74.9±20.3 | 66.33 ± 10.5 | 0.037 |
| Height (m) | 1.68±0.07 | 1.67 ± 0.06 | 0.78 |
| BMI (kg/m$^2$) | 26.3±5.1 | 24.6±3.8 | < 0.001 |
| BSA (m$^2$) | 1.83±0.18 | 1.78±0.15 | < 0.001 |
| SBP (mmHg) | 155.7±16.6 | 118.2±9.3 | < 0.001 |
| DBP(mmHg) | 98.3±10.8 | 77.8±6.9 | < 0.001 |
| PP(mmHg) | 57.3±11.1 | 40.4±7.1 | < 0.001 |
| MABP (mmHg) | 117.5±11.9 | 91.3±7.0 | < 0.001 |
| Pulse Rate (b/m) | 85.0±13.4 | 82.8± 9.1 | 0.467 |
| Urea (mmol/L) | 6.0±2.1 | 5.9±1.5 | 0.559 |
| Creatinine (ɥmol/L) | 85.3±18.5 | 83.1±16.2 | 0.142 |
| Total cholesterol (mmol/l) | 6.1±1.5 | 4.92±0.99 | < 0.001 |
| LDL cholesterol (mmol/l) | 3.79±1.3 | 2.92±0.83 | < 0.001 |
| HDL cholesterol (mmol/l) | 1.23±0.39 | 1.11±0.37 | < 0.001 |
| Triglyceride (mmol/l) | 1.79±0.97 | 1.33±0.64 | < 0.001 |
| RBG(mmol/L) | 6.3±1.6 | 6.2±1.6 | 0.809 |
| Thickened Arterial Wall | 22(7.3%) | 10 (3.3%) | 0.029 |
| Locomotor Brachialis | 14(4.7%) | 4(1.3%) | 0.017 |

*BMI; Body mass index, BSA Body surface area, SBP; Systolic Blood pressure, DBP; diastolic blood pressure, PP; Pulse pressure, MABP; mean arterial blood pressure, PR; pulse rate.*

**Table 2:** Echocardiography LV and LA characteristics of treatment naïve hypertensive patients and the non-hypertensive controls

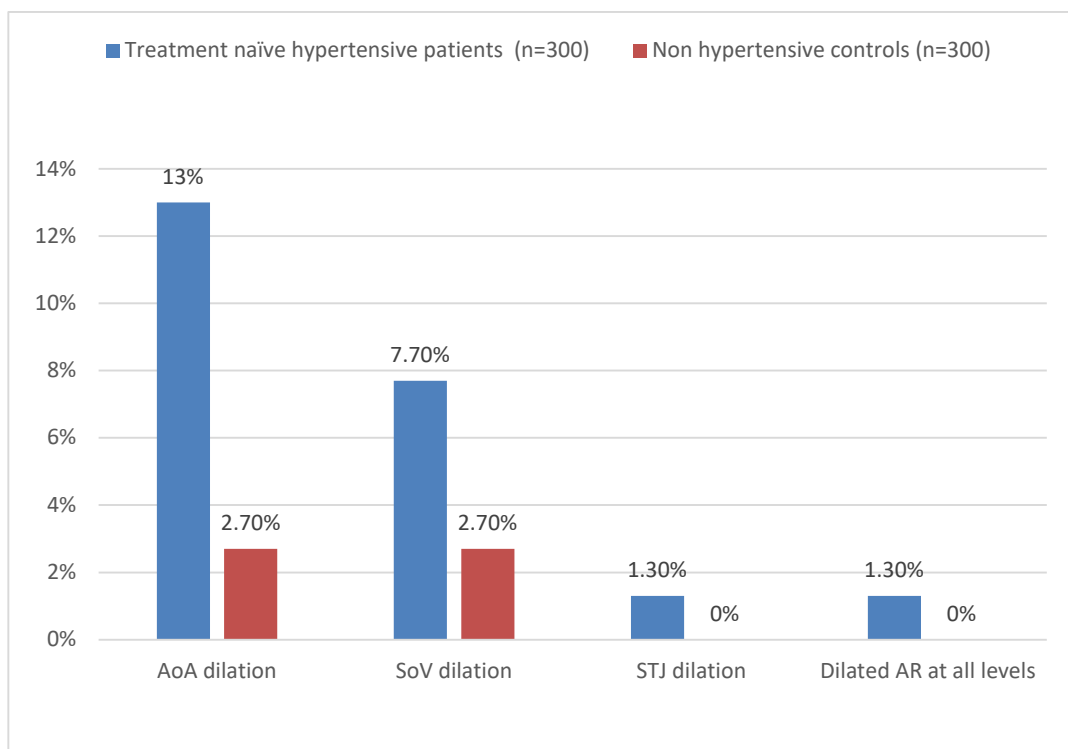|  | Treatment naive hypertensive patients<br>n = 300 | Non hypertensive controls<br>n = 300 | *P*-value |
|---|---|---|---|
| LVIDd(mm) | 47.8±6.8 | 46.7±4.1 | 0.37 |
| LVIDs (mm) | 31.8±6.9 | 30.2±4.2 | 0.12 |
| EDV(ml) | 116.9±49.4 | 108±26.8 | 0.23 |
| ESV(ml) | 37.9±22.7 | 32.4±10.3 | 0.061 |
| SV(ml) | 78.1±34.1 | 76.2±18.6 | 0.62 |
| LVEF (%) | 67.7±7.7 | 69.9±4.7 | 0.22 |
| FS (%) | 34.3±7.9 | 34.4±3.3 | 0.86 |
| IVSTd (mm) | 10.4±2.1 | 9.5±1.5 | 0.025 |
| PWTd (mm) | 10.9±2.5 | 9.5±1.2 | 0.001 |
| IVSTs (mm) | 15.1±3.1 | 14.1±1.7 | 0.045 |
| PWTs (mm) | 15.2±3.1 | 14.3±1.6 | 0.097 |
| LVM (gm) | 188.9±64.4 | 164.2±36.8 | 0.004 |
| LVMI (g/m$^2$) | 102.8±31.7 | 93.1±23.1 | 0.016 |
| LVH (g) | 164 (54.7%) | 32 (10.7%) | <0.001 |

**Table 3:** Aortic root dimensions at various levels among the treatment naïve hypertensive patients and the non-hypertensive controls

| Variables | Treatment naïve hypertensive group n = 300 Mean ±SD | Non-hypertensive controls n= 300 Mean ±SD | *P* |
|---|---|---|---|
| AoA diameter (mm) | 24.7±3.9 | 22.5±2.0 | 0.002 |
| SoV diameter (mm) | 33.1±3.4 | 31.4±3.4 | 0.023 |
| STJ diameter (mm) | 27.8±3.5 | 25.9±2.2 | 0.002 |
| AoA/ BSA (mm/m²) | 14.2±2.6 | 12.8±1.5 | < 0.001 |
| SoV/ BSA (mm/m²) | 18.9±2.6 | 17.9±2.2 | < 0.001 |
| STJ/BSA (mm/m²) | 15.4±2.8 | 15.1±2.3 | 0.068 |

*LVIDd, left ventricular internal diameter end diastole; LVIDs, left ventricular internal diameter in systole; EDV, end-diastolic volume; ESV, end-systolic volume; SV, stroke volume; LVEF left ventricular ejection fraction; FS, fraction shortening; IVSTd; interventricular septum thickness at end-diastole; PWTd; posterior wall thickness at end-diastole, IVSTs; interventricular septum thickness at end-systole; LVM, left ventricular mass; LVMI, left ventricular mass index; LAD, left ventricular diameter; LAA, left the atrial area; LAV, left atrial volume; LAVI, left atrial volume index; LVH, left ventricular hypertrophy.*

*AoA, Aortic annulus; SoV, sinuses of Valsalva; STJ, Sinotubular junction; BSA, body surface area*



**Figure 2:** Clustered bar charts demonstrating the prevalence of aortic root dilation at different levels

**Table 4:** Aortic dimensions for males and females among treatment naïve hypertensive patients and non-hypertensive controls

| Treatment naïve hypertensive patients | | | |
|---|---|---|---|
| **Variables** | Males (mean ± SD) n = 185 | Females (mean ± SD) n = 115 | *P* |
| **AoA diameter (mm)** | 27.1±3.9 | 22.2±3.7 | < 0.001 |
| **SoV dimeter (mm)** | 35.4±2.8 | 33.4±4.0 | < 0.001 |
| **STJ diameter (mm)** | 28.9±3.3 | 27.0±3.5 | <0.001 |
| **AoA/ BSA (mm/m$^2$)** | 14.7±2.6 | 14.3±2.6 | 0.18 |
| **SoV/ BSA (mm/m$^2$)** | 19.3±2.3 | 19.0±3.1 | 0.38 |
| **STJ/BSA (mm/m2)** | 15.7±2.5 | 15.4±2.8 | 0.34 |
| Non-hypertensive controls | | | |
| **Variables** | Males (mean ± SD) n = 183 | Females (mean ± SD) n = 117 | *P* |
| **AoA diameter (mm)** | 23.4±1.8 | 22.2±2.2 | < 0.001 |
| **SoV dimeter (mm)** | 32.5±2.5 | 31.3±3.2 | < 0.001 |
| **STJ diameter (mm)** | 26.6±1.8 | 24.9±3.0 | <0.001 |
| **AoA/ BSA (mm/m$^2$)** | 13.0±1.5 | 13.0±1.6 | 0.89 |
| **SoV/ BSA (mm/m$^2$)** | 18.1±2.1 | 18.3±2.0 | 0.29 |
| **STJ/BSA (mm/m$^2$)** | 14.8±1.6 | 14.6±2.0 | 0.51 |

*AoA, Aortic annulus; SoV, sinuses of Valsalva; STJ, Sinotubular junction;AoA/BSA, Aortic annulus indexed BSA; SoV/BSA, sinuses of Valsalvaindexed BSA; STJ/BSA, Sinotubular junctionindexed BSA; BSA, body surface area*

**Table 5:** Aortic root measurement and indexed body surface area (BSA) aortic root diameters in the treatment naïve hypertensive patients separate and non-hypertensive controls for ages ≥50years and <50years

| Treatment naïve hypertensive patients | | | |
|---|---|---|---|
| **Variables** | Age ≥ 50 years (mean ±SD)n = 118 | Age < 50 years (mean ±SD)n = 182 | *P* |
| **AoA diameter (mm)** | 24.7±2.5 | 22.8±2.9 | 0.001 |
| **SoV diameter (mm)** | 34.5±2.2 | 32.9±3.4 | 0.046 |
| **STJ diameter (mm)** | 28.2±2.3 | 27.2±2.8 | 0.050 |
| **AoA/ BSA (mm/m$^2$)** | 13.7±1.4 | 12.4±2.4 | 0.002 |
| **SoV/ BSA (mm/m$^2$)** | 18.8±1.7 | 17.7±2.5 | 0.430 |
| **STJ/BSA (mm/m$^2$)** | 15.6±1.6 | 14.7±2.4 | 0.035 |
| **Non-hypertensive controls** | | | |
| **Variables** | Age ≥ 50 years (mean ±SD) n =119 | Age < 50 years (mean ±SD) n = 181 | *P* |
| **AoA diameter (mm)** | 22.5±2.3 | 23.5±1.9 | 0.025 |
| **SoV diameter (mm)** | 32.3±2.6 | 31.9±2.8 | 0.046 |
| **STJ diameter (mm)** | 27.4±0.4 | 26.9±2.7 | 0.034 |
| **AoA/ BSA (mm/m$^2$)** | 13.5±1.1 | 13.3±0.9 | 0.663 |
| **SoV/ BSA (mm/m$^2$)** | 19.4 ±0.8 | 18.0±1.0 | 0.003 |
| **STJ/BSA (mm/m$^2$)** | 17.2 ±0.5 | 15.2±1.4 | 0.002 |

*AoA, Aortic annulus; SoV, sinuses of Valsalva; STJ, Sinotubular junction; AoA/BSA, Aortic annulus indexed BSA; SoV/BSA, sinuses of Valsalva indexed BSA; STJ/BSA, Sinotubular junction indexed BSA; BSA, body surface area*

**Table 6:** Correlation (Pearson R coefficients) between the aortic root dimensions and the age, the anthropometric measurements of the BP indices, LV indices and E/A among the treatment naïve hypertensive patients

| Variables | AoA | | SoV | | STJ | |
|---|---|---|---|---|---|---|
| | *r* | *p* | *r* | *P* | *r* | *p* |
| **Age ( years)** | 0.413 | <0.001 | 0.211 | 0.004 | 0.269 | **<0.001** |
| **Average SBP (mmHg)** | 0.096 | 0.192 | 0.193 | 0.009 | -0.049 | **0.509** |
| **Average DBP(mmHg)** | 0.383 | <0.001 | 0.188 | 0.011 | 0.043 | **0.563** |
| **PP (mmHg)** | 0.057 | 0.444 | 0.106 | 0.151 | -0.033 | **0.656** |
| **MABP (mmHg)** | 0.105 | 0.157 | 0.226 | 0.002 | -0.056 | **0.447** |
| **Height (m)** | 0.386 | <0.001 | 0.411 | <0.001 | 0.317 | **<0.001** |
| **Weight(kg** | 0.215 | <0.001 | 0.352 | <0.001 | 0.250 | **<0.001** |
| **BMI (Kg/m²)** | 0.015 | 0.844 | 0.121 | 0.102 | 0.062 | **0.401** |
| **BSA (m²)** | 0.239 | 0.001 | 0.453 | <0.001 | 0.329 | **<0.001** |
| **LVIDD (mm)** | 0.230 | 0.002 | 0.443 | <0.001 | 0.238 | **0.001** |
| **LVIDS (mm)** | 0.257 | <0.001 | 0.373 | <0.001 | 0.199 | **0.007** |
| **EDV(ml)** | 0.226 | 0.002 | 0.436 | <0.001 | 0.201 | **0.006** |
| **ESV(ml)** | 0.208 | 0.005 | 0.305 | <0.001 | 0.147 | **0.046** |
| **SV(ml)** | 0.171 | 0.020 | 0.370 | <0.001 | 0.181 | **0.014** |
| **IVSTd** | 0.159 | 0.031 | 0.411 | <0.001 | 0.457 | **<0.001** |
| **PWTd** | 0.010 | 0.894 | 0.322 | <0.001 | 0.315 | **<0.001** |
| **LVEF** | -0.100 | 0.178 | -0.025 | 0.731 | 0.005 | **0.946** |
| **LVM** | 0.220 | 0.003 | 0.572 | <0.001 | 0.451 | **<0.001** |
| **LVMI** | 0.232 | 0.002 | 0.466 | <0.001 | 0.381 | **<0.001** |
| **E/A** | -0.308 | <0.001 | -0.393 | <0.001 | -0.429 | **<0001** |

*SBP; Systolic blood pressure; DBP, diastolic blood pressure; PP, pulse pressure; MABP, mean arterial blood pressure; BMI body mass index; BSA; body surface area, LVIDD, left ventricular internal diameter in diastole; LVIDS, left ventricular internal diameter in systole; EDV, end diastolic volume; ESV, end systolic volume; SV, stroke volume; IVSTdinterventricular septum thickness at end diastole; PWTd; posterior wall thickness at end diastole, LVEF, left ventricular ejection fraction; LVM, left ventricular mass; LVMI, left ventricular mass index;; E/A, mitral inflow E velocity/mitral inflow A velocit*

## Discussion

The dimensions of the aortic root were larger among treatment hypertensive subjects than that of normotensive controls. Akintunde *et al* in a multicenter study in southwest Nigeria using M- mode to determine aortic root dimensions at the sinuses of Valsalva reported 31.68 ±3.58 mm in longstanding hypertensive patients[10].Katchunga *et al* in a hospital-based cross-sectional study reported an SoV dimension of 29.8 ± 4.2 among the Congolese population with longstanding hypertension and diabetes mellitus[16]. The difference might be due to methodological variations in determining the dimensions. Both Akintunde *et al* and Katchunga *et al* used M-mode to determine SoV dimensions.

Singh *et al* in an Indian population reported a larger mean aortic root diameter at AoA, SoV and STJ of hypertensive patients and normotensive individuals[17]. A similar finding was observed by Vizzardi *et al*, in the Italian population[6].The larger aortic root dimensions in the two studies above may be explained by the fact that the enroelees were longstanding hypertensive patients. In addition, racial, genetic and environmental factors could play a role in the marked difference in aortic root dimensions.

Furthermore, even after accounting for BSA, there were significant differences in aortic root (AR) diameters between the treatment naïve hypertensive and non-hypertensive control groups. This is in agreement with a previous report by Kim *et al* in a hospital-based study of 161 hypertensives and 207 normotensive American populations[8].

A study by Akintunde *et al* in southwest Nigeria among hypertensive patients using M-mode measurement at the SoV reported a prevalence of 8.4%for ARD[10]. The prevalence of ARD in the study by Akintunde *et al* was relatively higher than in our study, which is likely due to methodological differences and the participants in their study were patients with long-standing hypertension[10].Covella *et al*. in a systematic review found that the average prevalence of aortic root dilatation(ARD) at the SoV using M-mode modality in the pooled hypertensive population was 9.1%(95% CI 6.1–12.1) and ranged from 3.7% to 16.7% across the various studies[18].Cuspidi *et al* in a hospital-based descriptive, cross-sectional study in an Italian population of never-treated hypertensive patients (n = 519) reported a prevalence of 3.7% for SoV dilatation determined byM-mode modality.[19]In another study by Cuspidi *et al* a multicentre study including hypertensive patients from 14 centres found a prevalence of 10% for SoV dilatation[20].In the Evaluation of Target Organ Damage in Hypertension (ETODH) Registry, the prevalence of SoV dilatation was 6% though M-mode modality was used for the measurement amongst hypertensive subjects[21].While, Vizzardi *et al*. in a large prospective case-control study in a Caucasian population of hypertensive subjects and non-hypertensive controls using 2D measurement at end of diastole by a leading-edge to a leading-edge convention, reported the prevalence of aortic root dilatation at all levels to be 2.3% in the non-hypertensive controls and 19% among the hypertensive group at all levels[6].

The prevalence of aortic root dilatation at all levels and SoV in this study was lower than in the previous studies. This is probably because there are racial differences as the study population were African, black and partly due to methodological difference. Troy *et al*. in a large cross-sectional multi-racial study found that the black race has smaller absolute aortic root dimensions compared to the other races even after adjusting for body size[22]. This would further corroborate the fact that the black race has smaller aortic root dimensions than other races. In this study, it was found that most notably the dilation was at the proximal part of the aorta namely, the AoA and the SoV in the treatment naïve hypertensive patients. Singh *et al*. reported similar results in Indian hypertensive patients[17].

In this study, It was found that subjects aged greater than 50 years had larger aortic root diameters at all levels in both arms of the study, this is in agreement with Singh *et al* who reported a significant rise in AR dimensions at AoA and STJ after the fifth decade of life[17]. Similarly, Vizzardi *et al* reported a stiff rise in the aortic root dimensions at all levels at the ages of 50 years and above[6].This might be explained by the fact that cardiovascular disease risk factors begin to manifest after the fifth decade of life,[23]ARD is one of hypertension -mediated TOD.

Age had a positive correlation with aortic root dimensions at all levels in both genders. This is in agreement with Akintunde *et al*[10]. In addition, a report by Vandroux *et al* in a large cohort study conducted amongst the normotensive Beninese population showed that age had a positive correlation with SoV and STJ but not with AoA[24].A similar finding was also observed by Vriz *et al*. in a large cohort of 1,043 healthy adults with an age range of 16 to 92 years[25].

The absolute aortic root diameters were larger in males than in females' counterparts in both arms of the study. However, after indexing for BSA, the diameters were similar for both sexes. These findings are in agreement with the previous studies[6,24,25].

Furthermore, weight, height, and BSA had a positive correlation with aortic rootdiameter at all levels. This report is different from Singh *et al*. who noted an inverse relationship between BSA and aortic root size at all levels but strongly related to weight[17].

In the treatment naïve hypertensive population, the DBP was positively correlated with the aortic root dimension at the AoA and the SoV but not at the STJ. On applying multivariate analysis. Cuspidi *et al*. in the treated hypertensive population reported a positive correlation of the DPB[21].The longitudinal data from the Framingham Heart Study found individuals who had developed aortic root dilation to have higher baseline DBP. Kim *et al*[8]reported similar findings in an American population in a hospital-based cross-sectional study. The reason is yet to be established; however, these findings might lead to a hypothesis that elevated diastolic BP may play a key role in the pathogenesis of the aortic annular and the sinuses of Valsalva dilation due to dynamic changes during diastole.

In this study, it was demonstrated that a significant positive correlation exists between the LV and the LVMI with AR dimension at all the considered three levels. This is similar to the report by Akintunde *et al*[10] from southwest Nigeria where they showed a positive correlation between aortic root (SoV) dimensions and LVMI. In addition, similar findings were observed by the other studies[6,21].The hypertensive patients with ARD and left ventricular hypertrophy are observed to have a higher propensity for adjusted cardiovascular events[26].

The exact mechanism relating to the LVH and the ARD is still not well established. The E/A transmitral flow velocity ratio, a Doppler-derived index of LV diastolic dysfunction, increases the risk of cardiovascular morbidity in untreated and uncomplicated patients with essential hypertension[27].In this study, it was demonstrated that there was an inverse relationship between E/A and the aortic root dimensions in treatment naïve hypertensive patients. This is in agreement with Masugata *et al*[28]in a hospital-based cross-sectional descriptive study in the hypertensive Japanese population where they showed a negative correlation between E/A and aortic root dimension. In addition, the E/A-derived left ventricular diastolic dysfunction (LVDD) and left ventricular hypertrophy (LVH) may be associated with aortic root dilatations. The study's limitation was an inability to screen for syphilis, and autoimmune systemic diseases.

## Conclusion

The 2D transthoracic echocardiography determined aortic root dimension was higher amongst treatment naïve hypertensive patients compared to the non-hypertensive adults at all levels. Aortic root dilatation may be considered amarker of hypertension-mediated TOD.

## Reference

1. World Health Organization. A global brief on Hypertension - World Health Day 2013. *World Heal Organ*. 2013; **1**–40.
2. Roman MJ, Devereux RB, Kramer-Fox R, O'Loughlin J. Two-dimensional echocardiographic aortic root dimensions in normal children and adults. *Am J Cardiol*. 1989; **64**:507–12.
3. Biaggi P, Matthews F, Braun J, Rousson V, Kaufmann PA, Jenni R. Gender, Age, and Body Surface Area are the Major Determinants of Ascending Aorta Dimensions in Subjects With Apparently Normal Echocardiograms. *J Am Soc Echocardiogr*. 2009; **22**:720–5.
4. Roman MJ, Devereux RB, Niles NW, Hochreiter C, Kligfield P, Sato N, et al. Aortic root dilatation as a cause of isolated, severe aortic regurgitation. Prevalence, clinical and echocardiographic patterns, and relation to left ventricular hypertrophy and function. *Ann Intern Med*. 1987; **106**:800–7.
5. Palmieri V, Bella JN, Arnett DK, Roman MJ, Oberman A, Kitzman DW, et al. Aortic root dilatation at sinuses of valsalva and aortic regurgitation in hypertensive and normotensive subjects: The Hypertension Genetic Epidemiology Network Study . *Hypertension*. 2001; **37**:1229–35.
6. Vizzardi E, Maffessanti F, Lorusso R, Sciatti E, Bonadei I, Gelsomino S, et al. Ascending Aortic

Dimensions in Hypertensive Subjects: Reference Values for Two-Dimensional Echocardiography. *J Am Soc Echocardiogr*. 2016; **29**:827–37.

7. Covella M, Milan A, Totaro S, Cuspidi C, Re A, Rabbia F, et al. Echocardiographic aortic root dilatation in hypertensive patients: a systematic review and meta-analysis. *J Hypertens.* 2014; **32**:1928–35.

8. Kim M, Roman MJ, Cavallini MC, Schwartz JE, Pickering TG, Devereux RB. Effect of hypertension on aortic root size and prevalence of aortic regurgitation. *Hypertension*. 1996; **28**:47–52.

9. Jakrapanichakul D, Chirakarnjanakorn S. Comparison of aortic diameter in normal subjects and patients with systemic hypertension. *J Med Assoc Thai*. 2011; **94** Suppl 1:S51-6.

10. Akintunde A A. Aortic Root Dilatation in African Hypertensives Subjects: Frequency of Occurrence and Associations: A Multicentre Echocardiographic Survey. *Prensa Med*. 2013; **99**.

11. Leone D, Tosello F, Faletti R, Schivazappa G, Bruno G, Avenatti E, et al. Accuracy of transthoracic echocardiography in the assessment of proximal aortic diameter in hypertensive patients. *J Hypertens.* 2017; **35**:1626–34.

12. Lee JY, Lee J, Choi J, Kim H. Determination of Body Surface Area and Formulas to Estimate Body Surface Area Using the Alginate Method Determination of Body Surface Area and Formulas to Estimate Body Surface Area Using the Alginate Method. 2015; **27**:71–82.

13. Bell M. State of Alaska Measuring Height / Weight and Calculating BMI Guidelines for Schools. *Depertment Heal Soc Serv*. 2011; **1**–26.

14. Lang RM, Badano LP, Mor-Avi V, Afilalo J, Armstrong A, Ernande L, et al. Recommendations for Cardiac Chamber Quantification by Echocardiography in Adults: An Update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. *Eur Hear J – Cardiovasc Imaging*. 2015 Mar; **16**:233–71.

15. Devereux RB, Reichek N. Echocardiographic determination of left ventricular mass in man. Anatomic validation of the method. *Circulation*. 1977; **55**:613–8.

16. P. Katchunga a, b Ÿ, B DK, A RM, Mubalama E. Prevalence of initial aortic dilation and its relationship to remodeling left ventricular function in Congolese patients from South Kivu : study transverse. *Ann Cardiol Angiol*. 2018; **67**:250–5.

17. Singh V, Wander G, Mohan B, Aslam N, Tandon R, Chhabra S, et al. Hypertension and Size of Aortic Root – Cause-and-Effect Relationship. *J INDIAN Coll Cardiol*. 2021; **11**:13.

18. Covella M, Milan A, Totaro S, Cuspidi C, Re A, Rabbia F, et al. Echocardiographic aortic root dilatation in hypertensive patients: A systematic reviewand meta-analysis. *J Hypertens.* 2014; **32**:1928–35.

19. Cuspidi C, Meani S, Valerio C, Esposito A, Sala C, Maisaidi M, et al. Ambulatory blood pressure, target organ damage and aortic root size in never-treated essential hypertensive patients. *J Hum Hypertens*. 2007; **21**:531–8.

20. Cuspidi C, Negri F, Salvetti M, Lonati L, Capra A, Milan A, et al. Aortic root dilatation in hypertensive patients : A multicenter survey in echocardiographic practice survey in echocardiographic practice. 2011; **7051**:267–273.

21. Cuspidi C, Meani S, Fusi V, Valerio C, Sala C, Zanchetti A. Prevalence and correlates of aortic root dilatation in patients with essential hypertension: relationship with cardiac and extracardiac target organ damage. *J Hypertens*. 2006; **24**:573–80.

22. Labounty TM, Kolias TJ, Bossone E, Bach DS. Differences in Echocardiographic Measures of Aortic Dimensions by Race. *Am J ofCardiology*. 2019; **123**:2015–21.

23. Rodgers JL, Jones J, Bolleddu SI, Vanthenapalli S, Rodgers LE, Shah K, et al. Cardiovascular risks associated with gender and aging. *J Cardiovasc Dev Dis*. 2019; **6**.

24. Vandroux D, Houehanou C, Saka D, Sonou A, Magne J, Houinato D, et al. Normal values of aortic root diameters in sub saharan africans: the TAHES study. *Eur Heart J*. 2021; **42**:56–7.

25. Vriz O, Driussi C, Bettio M, Ferrara F, D'Andrea A, Bossone E. Aortic Root Dimensions and Stiffness in Healthy Subjects. *Am J Cardiol.* 2013; **112**:1224–9.

26. Lam CSP, Xanthakis V, Sullivan LM, Lieb W, Aragam J, Redfield MM, et al. Aortic Root Remodeling Over the Adult Life Course. *Circulation*. 2010; **122**:884–90.

27. Cuspidi C, Sala C, Casati A, Bombelli M, Grassi G, Mancia G. Clinical and prognostic value of hypertensive cardiac damage in the PAMELA Study. *Hypertens Res*. 2017; **40**:329–335.

28. Schillaci G, Pasqualini L, Verdecchia P, Vaudo G, Marchesi S, Porcellati C, et al. Prognostic significance of left ventricular diastolic dysfunction in essential hypertension. *J Am Coll Cardiol.* 2002; **39**:2005–11.

29. Masugata H, Senda S, Murao K, Okuyama H, Inukai M, Hosomi N, et al. Aortic root dilatation as a marker of subclinical left ventricular diastolic dysfunction in patients with cardiovascular risk factors. *J Int Med Res*. 2011; **39**:64–70.