**Original Article**

Ultrasonographic Study of the Effects of Essential Hypertension on the Luminal Diameter and Doppler Velocimetric Indices of the Abdominal Aorta in Adults

## John Oluwafemi Adetokunbo1, Bolanle Olubunmi Ibitoye2,

**Abstract**

**Objective:** To investigate the effects of essential hypertension on the luminal diameter (caliber) and Doppler velocimetric indices of the abdominal aorta (AA) in adult patients with systemic hypertension. **Materials and Methods:** This was a prospective descriptive comparative study of 254 participants (127 with essential hypertension and 127 age/sex-matched controls). Their anthropometric parameters, fasting blood pressure, lipid profile, fasting blood sugar, and triplex sonography of the suprarenal and infrarenal abdominal aorta (Peak systolic velocity, PSV; End- diastolic velocity, EDV; Resistive Index, RI; and luminal diameter) were evaluated. **Results:** The mean age of the male subjects was 64.02 ± 10.02 years, while the mean age of the male controls was

63.14 ± 10.52 years (*P* > 0.05). The mean age of female subjects was 61.23 ± 10.09 years, while the mean age of the female controls was 61.76 ± 10.26 years (*P* > 0.05). The age group 60 – 69 years had the highest number of subjects and controls. The mean duration of hypertension in the subjects was

12.5 ± 5.2 years. The suprarenal and infrarenal abdominal aortic diameters (AAD) were higher in males than age-matched female counterparts. AAD increased with age mostly in hypertensive male subjects. PSV (in males) and RI (in both sexes) were elevated in hypertensive subjects compared to controls, while EDV (in both sexes) was significantly lower in subjects than controls. Multivariate linear regression showed that age and diastolic blood pressure were significant independent predictors for both suprarenal and infrarenal AADs. **Conclusion:** Systemic hypertension causes structural and hemodynamic changes in the abdominal aorta which are detectable on triplex sonography.

**Keywords:** *Abdominal aorta, doppler ultrasonography, end diastolic velocity, hypertension, peak systolic velocity, resistive index*

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# Introduction

Systemic Hypertension is persistently elevated blood pressure equal to or greater than 140/90 mmHg. It is a chronic illness with significant morbidity, affecting millions of people worldwide.[1,2] Up to 90% of adult hypertension is essential hypertension, i.e., without a discernible cause.[3] With

In many countries, the prevalence dramatically increases in patients older than 60 years. Furthermore, people of African descent have a higher disease burden than other racial groups. The prevalence of systemic hypertension is also higher in low- and-middle-income countries (LMICs) than in high-income nations.[7]

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increasing systolic and diastolic pressures,

Elevated systemic arterial blood pressure

the risk of mortality or morbidity increases gradually.[4] The disease has a multiorgan effect, with far-reaching consequences on the eye, brain, heart, kidneys and vascular system, endocrine organs, etc.[3,5]

Hypertension affects 31.1% (1.39 billion individuals) of adults worldwide (which is anticipated to reach 1.5 billion by 2025), with men and women experiencing similar rates of hypertension.[1,6]

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is a major cause of premature vascular disease, leading to cerebrovascular accidents, ischemic heart disease, and peripheral vascular disease.[3] Structural alterations in the abdominal aorta (calcified atherosclerotic plaque or lumen dilatation) are associated with conventional cardiovascular disease risk factors.[8] There is a 66% increased risk of developing abdominal aortic aneurysm in patients with systemic hypertension compared to non-

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hypertensive adults.[6] There is a close relationship between hypertension and the vascular morphology in the body, as blood pressure is a product of cardiac output and total peripheral resistance.

A detailed anatomical assessment of the blood vessels and their branches can be achieved using vascular ultrasonography. Therefore, the effects and possible complications of essential hypertension on the abdominal aorta can be studied using ultrasonography. The abdominal aorta dilates early in systemic hypertension, characterized by a progressive enlargement of the lumen, because of the weakening of the aortic walls.[9]

This study analyzes the effects of essential hypertension on the luminal diameter (caliber) and Doppler velocimetric indices of the abdominal aorta in adult patients with systemic hypertension.

# Materials and Methods

This was a prospective descriptive comparative study carried out at the radiology department of Obafemi Awolowo University Teaching Hospitals Complex, Ile-Ife, Osun state, Nigeria. The Ethics and Research Committee of the institution approved the study (Approval number: ERC/2016/07/05), and was conducted in accordance with the Declaration of Helsinki 2013.

One hundred and twenty-seven clinically stable adults (aged 40 - 80 years) diagnosed with essential hypertension were recruited consecutively from the cardiology clinic of the hospital, while healthy volunteers (with a normal systemic blood pressure ≤ 120/80 mmHg) served as age-/sex-matched controls (*n* = 127). The controls were volunteer colleagues, hospital staff members, and patient relatives. All the participants gave written informed consent.

The exclusion criteria were co-existing essential hypertension and diabetes mellitus, secondary hypertension, hyperlipidemia, endocrinopathies, and known vasculitides.

### Clinical assessment

The participants’ age, gender, and duration of hypertension were recorded. The weight (in kilograms, to the nearest

0.5 kg) and height (in meters, to the nearest 0.1 m) were measured using a weighing scale with an attached stadiometer (model ZT-160; China). The Body Mass Index (BMI in kg/m2) was calculated by dividing the weight by the square of the height. The participants were categorized into normal BMI, overweight, obese, and morbidly obese at BMI of 18.5 - 24.9 kg/m2, 25.0 - 29.9 kg/m2, 30 - 39.9 kg/ m2; and ≥ 40 kg/m2, respectively.[10]

The blood pressure (BP) was measured in the sitting position after the participant had rested for at least 15 minutes. Three readings were taken and the average of the last two recorded.

Hypertension was taken as BP ≥ 140/90 mmHg or blood pressure elevation requiring treatment with antihypertensive medications.[11]

### Laboratory evaluation

Fasting blood sugar and blood lipids were done, at the point of contact, in the Radiology department. An Accu- check glucometer was used to assess fasting blood sugar. Fasting blood sugar of 4.1 - 5.9 mmol/L was considered as normal.[12] Venous blood (5 ml) was taken from each subject and control for the determination of fasting serum lipid profile (All components of the lipid profile were stated in mmol/L). Serum total cholesterol (TC), High- density lipoprotein (HDL), and Triglycerides (TG) were determined by direct assay. Low-density lipoprotein (LDL) was calculated using the empirical relationship of the Friedewald formula:[13,14]

LDL = TC – HDL - TG/5 (mg/dL) LDL = TC – HDL - TG/2.2 (mmol/L)

### Sonographic assessment

All the participants were scanned by the first author, who was a 5th-year radiology resident doctor, under the supervision of a consultant radiologist with 18 years’ experience. Ultrasonic evaluation was performed in real- time using a Mindray® ultrasound scanner model DC-7 (Shenzhen Mindray Bio-medical Electronics, Nanshan, Shenzhen, China) with Doppler facilities and a convex transducer (frequency = 3.5 - 5.0 MHz).

Each participant was placed in a supine position with arms by the side to relax the abdominal wall. The luminal diameter of the abdominal aorta was measured at two levels [Figure 1]. The proximal (suprarenal) diameter was taken at a level posterior to the confluence of the splenic vein and portal vein, with the left renal vein crossing anterior to the abdominal aorta.[15] The distal (infrarenal) diameter was measured just 1 cm above the aortic bifurcation.[15-21]

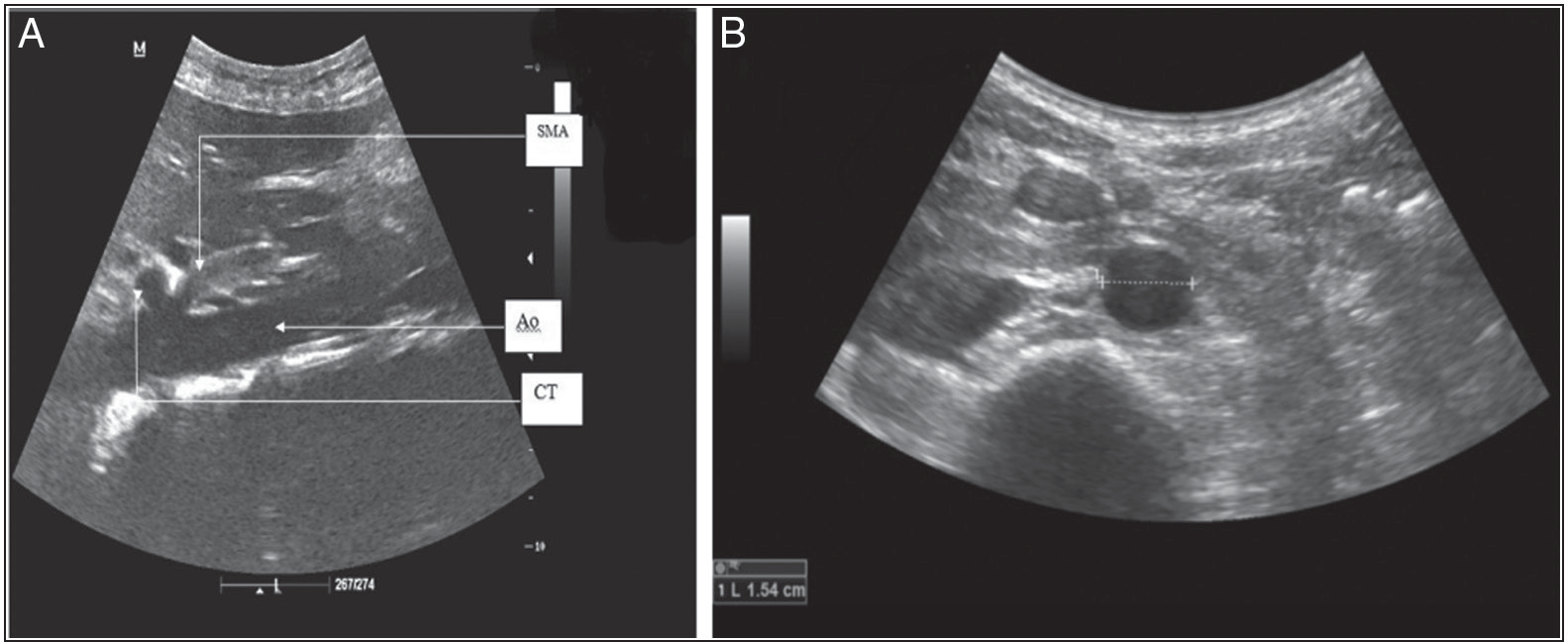
Triplex Doppler ultrasound was used to measure the peak systolic velocity (PSV), End diastolic velocity (EDV) and Resistive Index (RI) of the abdominal aorta at the two locations mentioned above, using the automatic tracing function on the ultrasound machine [Figure 2]. Velocity measurements were taken with a Doppler sample gate that was two-thirds of the vessel lumen, and an insonation angle

<600, to avoid aliasing errors.

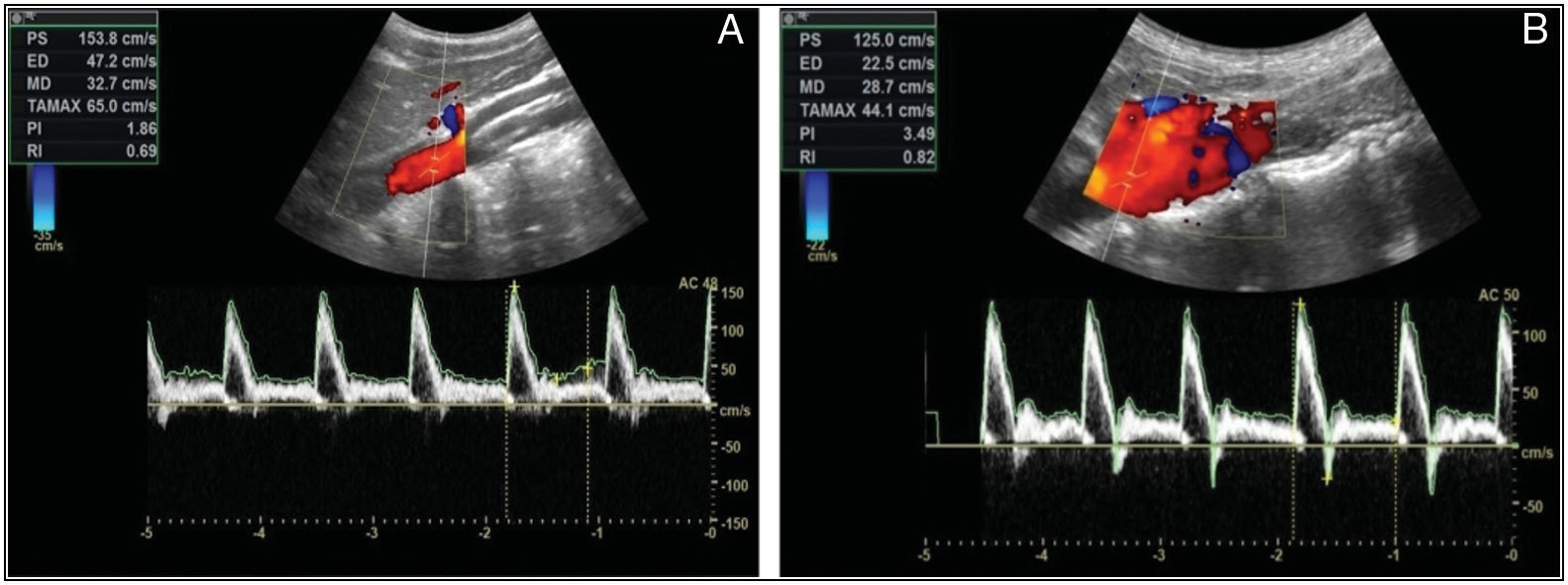
### Data analysis

Statistical analysis was done using the IBM SPSS Statistics for Windows, version 22 (IBM Corp., Armonk, N.Y., USA). Test of Normality was performed with the Kolmogorov Smirnov’s test. Continuous variables were presented as mean ± SD (standard deviation) while categorical variables were presented as percentages and frequencies. Statistical

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**Figure 1: B-Mode longitudinal view (A) and transverse view (B) of the suprarenal abdominal aorta showing the proximal abdominal aortic branches (SMA = superior mesenteric artery; Ao = Abdominal aorta; CT = Celiac trunk) and measurement of the aortic luminal diameter, respectively**



**Figure 2: Longitudinal triplex Doppler ultrasound image showing the spectral tracing and velocimetric indices of the suprarenal (A) and infrarenal (B) abdominal aorta**

significance was set at *P* ≤ 0.05. Regression analysis was used to evaluate the predictors of abdominal aortic diameter.

# Results

### General characteristics of the study population

There were 127 hypertensive subjects and 127 controls. The mean age of the male subjects was 64.02 ± 10.02 years, while the mean age of the male controls was 63.14 ± 10.52 years (*P* = 0.122). The mean age of female subjects was

61.23 ± 10.09 years, while the mean age of the female controls was 61.76 ± 10.26 years (*P* = 0.122). The age group 60 – 69 years had the highest number of subjects and controls, while the age group 40 - 49 years had the least [Table 1]. The other sociodemographic and clinic- laboratory parameters of the subjects and controls are compared in [Tables 1 & 2]. The mean duration of systemic hypertension in the subjects was 12.5 ± 5.2 years. The duration of hypertension was <10 years in 40 subjects (31.5%), 10–19 years in 60 subjects (47.2%), 20–29 years in 17 subjects (13.4%), and ≥ 30 years in 10 subjects (7.9%)

### Age and abdominal aortic diameter (AAD)

The abdominal aortic diameter (suprarenal and infrarenal) increased with age mostly in hypertensive male subjects

(*P*<0.0001). Greater luminal diameter increase was noticed among the cases than controls in both males and females, as evidenced by the significant increase of luminal diameter in age groups 60–69 years and ≥70 years [Table 3].

### Peak systolic velocity (PSV), end diastolic velocity (EDV) and resistive index (RI)

There were significantly higher PSV, higher RI, and lower EDV in the hypertensive subjects than the controls in both the suprarenal and infrarenal abdominal aorta [Tables 4 & 5] except for the suprarenal abdominal aorta of hypertensive female subjects where there was no significant difference in PSV between subjects and controls (*P* = 0.480). Although, the PSV in the suprarenal abdominal aorta was higher in female hypertensives than the controls, the difference was not statistically significant.

### Correlation and regression analyses evaluating predictors of abdominal aortic diameter (AAD) and intima medial thickness (IMT)

Bivariate correlational analysis revealed that age, height, weight, and diastolic blood pressure (DBP) correlated significantly with both suprarenal AAD and infrarenal AAD. Triglyceride level and sex correlated significantly with only the suprarenal AAD, while the duration of

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### Table 1: Participants’ demographic characteristics

|  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- |
| **Variables** |  | **Cases** | **Control** | **T** | **χ2** | **df** | **P value** |
|  |  | **n = 127** | **n = 127** |  |  |  |  |
| Age (yrs) | Total | 62.31 ± 10.39 | 62.32 ± 10.52 | -0.06 |  |  | 0.995 |
| (Mean ± SD) | Male | 64.02 ± 10.73 | 63.14 ± 10.94 | 2.411 |  |  | 0.122 |
|  | Female | 61.23 ± 10.09 | 61.76 ± 10.26 |  |  |  |  |
|  | Range | (40 - 80) | (40 - 80) |  |  |  |  |
| Age group | 40 – 49 | 18 (14.2) | 14 (11.0) |  | 0.945 | 3 | 0.815 |
| *n (%)* | 50 – 59 | 30 (23.6) | 35 (27.6) |  |  |  |  |
|  | 60 – 69 | 44 (34.6) | 42 (33.1) |  |  |  |  |
|  | ≥ 70 | 35 (27.6) | 36 (28.3) |  |  |  |  |
| Gender | Male | 49 (38.6) | 51 (40.2) |  | 0.066 | 1 | 0.898 |
| *n (%)* | Female | 78 (61.4) | 76 (59.8) |  |  |  |  |
| Education, | Primary | 24 (18.9) | 55 (32.3) |  | 17.72 | 2 | **<0.0001** |
| *n (%)* | Secondary | 62 (48.8) | 42 (23.6) |  |  |  |  |
|  | Tertiary | 41 (32.3) | 30 (28.0) |  |  |  |  |
| Smoking | Yes | 2 (1.2) | 0 (0.0) |  | 2.02 | 1 | 0.156 |
| *n (%)* | No | 125 (98.8) | 127 (100.0) |  |  |  |  |
| Alcohol | Yes | 5 (3.9) | 0 (0.0) |  | 5.10 | 1 | **0.024** |
| *n (%)* | No | 122 (96.1) | 127 (100.0) |  |  |  |  |

**Table 2: Clinical and laboratory parameters of the study population**

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| **Variables** | **Cases** | **Controls** | **t** | **χ2** | **Df** | **P value** |
|  | **n = 127** | **n =127** |  |  |  |  |
| Height (m) | 1.65 ± 0.07 | 1.65 ± 0.07 | 0.317 | - | 252 | 0.752 |
| Weight (Kg) | 68.16 ± 14.81 | 62.67 ± 9.03 | 3.70 | - | 252 | **<0.0001** |
| BMI (Kg/m2) | 25.01 ± 5.04 | 23.03 ± 2.88 | 3.85 | - | 252 | **<0.0001** |
| Underweight | 9 (7.1) | 6 (4.7) | - | 24.88 | 3 | **<0.0001** |
| Normal Weight | 60 (47.2) | 96 (75.6) |  |  |  |  |
| Overweight | 37 (29.1) | 21 (16.5) |  |  |  |  |
| Obese | 21 (16.5) | 4 (3.1) |  |  |  |  |
| SBP (mmHg) | 153.42 ± 22.05 | 114.72 ± 10.14 | 17.97 |  | 252 | **<0.0001** |
| DBP (mmHg) | 91.54 ± 12.35 | 70.47 ± 7.85 | 16.23 |  | 252 | **<0.0001\*** |
| TC (mol/L) | 5.22 ± 0.86 | 4.10 ± 0.53 | 12.51 |  | 252 | **<0.0001\*** |
| HDL (mmol/L) | 1.47 ± 0.58 | 1.69 ± 0.39 | -3.52 |  | 252 | **0.001** |
| TG (mmol/L) | 1.43 ± 0.72 | 0.87 ± 0.22 | 8.42 |  | 252 | **<0.0001** |
| LDL (mmol/L) | 3.09 ± 0.95 | 2.04 ± 0.55 | 10.77 |  | 252 | **<0.0001** |
| FBS (mmol/L) | 4.59 ± 0.39 | 3.93 ± 0.15 | 17.96 |  | 252 | **<0.0001** |

*\*BMI – Body Mass Index; DBP – Diastolic Blood Pressure; FBS – Fasting Blood Sugar; HDL – High Density Lipoprotein; LDL- Low Density Lipoprotein; SBP-Systolic Blood Pressure; TC-Total Cholesterol; TG-Triglycerides*

hypertension and systolic blood pressure (SBP) correlated significantly with only the infrarenal AAD.

A multivariate linear regression of these significant predictors revealed that age (*P* < 0.0001), diastolic blood pressure (DBP) (*P* = 0.04), and serum triglyceride level (*P* = 0.024) were the only statistically significant independent predictors of suprarenal AAD, while age (*P* < 0.0001) and DBP (*P* = 0.04) were the only independent predictors of infrarenal AAD [Table 6].

# Discussion

In this study, there was a direct linear relationship between the abdominal aortic luminal diameters (suprarenal and infrarenal) and age in both study groups (cases and controls). This pattern was corroborated by Laughlin *et al.*[22] and

Joh *et al*.[23] Joh *et al.* reported the average diameter of the abdominal aorta as 17.55 mm and 18.1 mm for people in their 50s and 60s, respectively.[23] Similarly, Lanne and co-workers documented a 30% increase in the diameter of the abdominal aorta from age 25 to 71 years,[24] while Pedersen *et al*.[9] observed an annual increase of 0.08 mm and 0.05 mm in the luminal diameters of the proximal and distal abdominal aorta, respectively.

A greater increase in the abdominal aortic luminal diameter with aging was noticed in the case group (hypertensive subjects) compared to the controls in this study. This suggests an independent contribution from hypertension to the age-related increase in aortic circumference. Similarly, the association of hypertension with eventual aneurysmal dilatation of the abdominal aorta has been reported

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### Table 3: Aortic diameter by age

**Age group (yrs) Suprarenal Diameter**

**(Mean ± SD)**

**Infrarenal Diameter (Mean ± SD)**

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
|  | | **Cases** | **Controls** |  | **Cases** | **Controls** |
| **(mm)** | **(mm)** |  | **(mm)** | **(mm)** |
| 40 – 49 | Male | 14.80 ± 2.75 | 13.13 ± 0.87 |  | 12.38 ± 2.07 | 10.35 ± 0.57 |
|  | Female | 15.59 ± 2.48 | 12.36 ± 0.59 |  | 13.71 ± 2.75 | 9.70 ± 1.22 |
|  | Total | 15.41 ± 2.48 | 12.58 ± 0.74 |  | 13.42 ± 2.62 | 9.89 ± 1.10 |
| 50 – 59 | Male | 16.24 ± 2.56 | 14.60 ± 0.95 |  | 12.58 ± 2.16 | 12.16 ± 1.40 |
|  | Female | 14.97 ± 1.40 | 13.04 ± 1.21 |  | 13.09 ± 1.39 | 10.23 ± 0.93 |
|  | Total | 15.48 ± 2.01 | 13.80 ± 1.33 |  | 12.89 ± 1.72 | 11.17 ± 1.52 |
| 60 – 69 | Male | 17.51 ± 1.84 | 15.13 ± 0.91 |  | 14.31 ± 1.14 | 12.65 ± 1.35 |
|  | Female | 16.24 ± 1.57 | 12.92 ± 0.49 |  | 14.02 ± 1.43 | 10.31 ± 0.96 |
|  | Total | 16.68 ± 1.76 | 13.55 ± 1.19 |  | 14.12 ± 1.33 | 10.98 ± 1.52 |
| ≥70 | Male | 17.58 ± 2.14 | 14.82 ± 1.40 |  | 15.04 ± 2.52 | 12.48 ± 1.63 |
|  | Female | 16.44 ± 1.54 | 13.10 ± 0.64 |  | 14.51 ± 1.12 | 10.50 ± 1.08 |
|  | Total | 17.02 ± 1.94 | 13.96 ± 1.38 |  | 14.79 ± 1.96 | 11.49 ± 1.69 |
| F |  | 8.698 |  |  | 10.542 |  |
| *P* value |  | **<0.0001** |  |  | **0.001** |  |

### Table 4: Doppler parameters of the suprarenal abdominal aorta

|  |  |  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- | --- | --- |
| **Sex** | **Suprarenal**  **Doppler** | **Groups** | **N** | **Mean** | **SD** | **Mean difference** | **T** | **df** | **P-value** |
| Male | PSV (cm/sec) | **Cases** | 49 | 63.0 | 20.2 | 7.8 | 2.2 | 98 | **0.030** |
|  |  | **Control** | 51 | 55.2 | 14.9 |  |  |  |  |
|  | EDV (cm/sec) | **Cases** | 49 | 8.8 | 3.1 | -8.7 | -9.3 | 98 | **<0.0001** |
|  |  | **Control** | 51 | 17.5 | 5.8 |  |  |  |  |
|  | RI | **Cases** | 49 | 0.9 | 0.0 | 0.2 | 17.3 | 98 | **<0.0001** |
|  |  | **Control** | 51 | 0.7 | 0.1 |  |  |  |  |
| Female | PSV (cm/sec) | **Cases** | 78 | 59.0 | 19.4 | 2.1 | 0.7 | 152 | 0.480 |
|  |  | **Control** | 76 | 56.9 | 17.6 |  |  |  |  |
|  | EDV (cm/sec) | **Cases** | 78 | 8.1 | 3.7 | -9.1 | -11.1 | 152 | **<0.0001** |
|  |  | **Control** | 76 | 17.2 | 6.2 |  |  |  |  |
|  | RI | Cases | 78 | 0.9 | 0.1 | 0.2 | 19.0 | 152 | **<0.0001** |
|  |  | Control | 76 | 0.7 | 0.1 |  |  |  |  |

*PSV- peak systolic velocity; EDV- end diastolic velocity; RI- resistive index*

previously in multiple studies.[25-28] In addition, a systematic review and meta-analysis of cohort studies found a 66% increased risk of abdominal aortic aneurysm in systemic hypertension.[6]

In the study by Joh *et al*.[23] in South Korea, infrarenal abdominal aortic diameter was 19 mm in males and 17.9 mm in females. A similar study in the USA (using the same landmark) by Ouriel *et al*.[29] documented diameters of 23 mm and 19 mm in males and females, respectively. Sariosmanoglu *et al*.[30] in Turkey had also reported that the mean aortic diameters was 16 mm in males and 15 mm in females. Men have a larger abdominal aortic diameter than women according to Esposito *et al.*,[31] and both aging and overweight have a significant effect on this measurement. These aforementioned researchers and other investigators[32,33] reported larger values of abdominal aortic diameter in males compared to females. A similar finding was noted in this study with male mean aortic luminal

diameter being larger than the females’ in both controls and cases. The larger diameter of the abdominal aorta in the males might be attributable to the anabolic effect of testosterone.[22]

There are relatively few reports evaluating racial differences in the aortic diameter. In a study of the differences in abdominal aortic luminal diameters between racial groups, Laughlin *et al.*[22] noted that the abdominal aortic diameters of people of Chinese, African and Hispanic descent were smaller than the abdominal aortic diameter of Caucasians. This was true even after adjusting for differences in body size and other factors. They also observed that the abdominal aortic diameter was greater in the male than the female age- matched groups. This could explain the smaller diameter in the controls of this study compared to the values in the normal subjects in other regions of the world, as stated above.

The normal Doppler waveforms in the aorta varies with location.[34] The suprarenal aorta has a narrow and well

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### Table 5: Doppler parameters of the infrarenal abdominal aorta

|  |  |  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- | --- | --- |
| **Sex** | **Infrarenal Doppler** | **Groups** | **N** | **Mean** | **SD** | **Mean difference** | **T** | **df** | **P-value** |
| Male | PSV (cm/sec) | **Cases** | 49 | 83.8 | 21.0 | 11.3 | 2.9 | 98 | **0.010** |
|  |  | **Control** | 51 | 72.5 | 18.3 |  |  |  |  |
|  | EDV (cm/sec) | **Cases** | 49 | 10.5 | 2.8 | -11.1 | -12.2 | 98 | **<0.0001** |
|  |  | **Control** | 51 | 21.6 | 5.7 |  |  |  |  |
|  | RI | **Cases** | 49 | 0.9 | 0.0 | 0.2 | 19.3 | 98 | **<0.0001** |
|  |  | **Control** | 51 | 0.7 | 0.1 |  |  |  |  |
| Female | PSV (cm/sec) | **Cases** | 78 | 79.1 | 20.8 | 11.2 | 3.6 | 152 | **<0.0001** |
|  |  | **Control** | 76 | 67.9 | 17.8 |  |  |  |  |
|  | EDV (cm/sec) | **Cases** | 78 | 9.6 | 3.7 | -10.1 | -11.6 | 152 | **<0.0001** |
|  |  | **Control** | 76 | 19.6 | 6.7 |  |  |  |  |
|  | RI | **Cases** | 78 | 0.9 | 0.0 | 0.2 | 24.9 | 152 | **<0.0001** |
|  |  | Control | 76 | 0.7 | 0.0 |  |  |  |  |

*PSV- peak systolic velocity; EDV- end diastolic velocity; RI- resistive index*

### Table 6: Regression analyses of the association between suprarenal and infrarenal abdominal aortic luminal diameters and

**other parameters**

**Independent Variables B Std. Error t P value R2**

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| Model I\* | (Constant) | 0.270 | 4.941 | 0.055 | 0.957 | 0.286 |
|  | Age (years) | 0.061 | 0.016 | 3.810 | **<0.0001** |  |
|  | Height | 4.873 | 2.870 | 0.168 | 0.092 |  |
|  | Sex | -0.403 | 0.389 | -1.035 | 0.303 |  |
|  | Weight | 0.023 | 0.012 | 1.859 | 0.065 |  |
|  | Diastolic BP | 0.028 | 0.014 | 2.010 | **0.041** |  |
|  | Triglyceride | 0.510 | 0.223 | 2.284 | **0.024** |  |
| Model II\*\* | (Constant) | -4.142 | 4.750 | -0.872 | 0.385 | 0.269 |
|  | Age (years) | 0.066 | 0.016 | 4.011 | **<0.0001** |  |
|  | Height | 4.716 | 2.751 | 1.174 | 0.089 |  |
|  | Sex | 0.562 | 0.373 | 1.507 | 0.134 |  |
|  | Weight | 0.020 | 0.012 | 1.707 | 0.090 |  |
|  | Duration of HTN | 0.020 | 0.023 | 0.880 | 0.381 |  |
|  | Systolic BP | 0.004 | 0.009 | 0.393 | 0.069 |  |
|  | Diastolic BP | 0.035 | 0.017 | 2.077 | **0.040** |  |

*\*Outcome variable is suprarenal aortic luminal diameter; \*\*Outcome variable is infrarenal aortic luminal diameter HTN- Hypertension; BP- Blood Pressure*

defined systolic complex with forward flow during diastole. Below the renal arteries (distal/infrarenal aorta), the diastolic flow is much reduced. The mean PSV, EDV and RI in cases were higher than controls in this study. These lower values in controls is probably due to the effect of hypertension on the abdominal aorta which causes rigidity and structural aortic wall thickening.[22] Laughlin *et al*.[22] stated that systolic wave form and pulse pressure increase with hypertension.

In the index study, hypertension appears contributory to the increase in luminal diameter of the abdominal aorta – in agreement with study of Agu *et al.*[35] However, it is at variance with that of Steinberg *et al.*[36] who found that no part of the abdominal aorta, in a group of patients with hypertension, was any wider than that of the normotensive group at similar levels of measurement. This disparity may be due to their use of arteriography for measurement.

The limitations of this study are as follows: firstly, obese patients were difficult to assess due to attenuation of ultrasonic waves resulting from increased tissue depth. This was minimized by turning patient on right lateral position and ultrasound measurement of the abdominal aorta taken from the left using the left lobe of the liver as a window. Secondly, bowel gas limited penetration of ultrasonic waves. Patient had to be turned to right lateral side sometimes to displace copious intraabdominal gas. The patients were also scanned in a fasted state to reduce gaseous obscuration of visualization of the abdominal aorta. Thirdly, the involvement of subjects on medications might have introduced drug induced changes of the abdominal aorta. Patients were encouraged to come fasted without taking medications which can be taken after completion of examination. Fourthly, since all subjects were recruited from only one tertiary health facility, selection bias was inevitable.

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In conclusion, abdominal aortic luminal diameter in patients with systemic hypertension increased with age (mostly in male subjects). The aortic luminal diameter (suprarenal and infrarenal portions) was higher in males than in their age-matched female counterparts. PSV (in males) and RI (in both sexes) were elevated in hypertensive subjects compared to age/sex-matched controls, while EDV (in both sexes) was significantly lower in subjects than controls.

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### Conflicts of interest

There are no conflicts of interest.

### Author contributions

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This manuscript has been read and approved by all the authors, the requirements for authorship as stated in the JWAS author instructions have been met, and each author believes that the manuscript represents honest work.

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