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COMPARISON OF CAROTID ARTERY LUMINAL DIAMETER BY B-MODE ULTRASOUND IN HYPERTENSIVE AND NORMOTENSIVE ADULTS

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ABSTRACT

Background: An increased carotid artery luminal diameter (CALD) is an indication of arterial remodelling and occurs in response to the development of cerebrovascular disease (CVD). However, there is a paucity of data comparing common carotid luminal diameter of hypertensive with normotensive adults in the study locality.

Objective: This study is therefore aimed at evaluating the CCALD in hypertensive and normotensive adults.

Methodology: This study involved 188 hypertensive and 144 normotensive subjects. The age range of the 332 subjects comprising 151 males 181 females was 18 to 80 years with mean ages of 52.49±15.31 years and 44.45±16.03 years for the hypertensive and normotensive subjects, respectively. The common carotid artery luminal diameter (CCALD) at the diastolic phase of the cardiac cycles was scanned and measured using a venue 50 ultrasound scanners with electronic callipers and a high frequency linear array transducer.

Results: The overall mean CCALD was 6.20±1.01 mm and 5.88±0.77 mm for hypertensive and normotensive subjects, respectively. The mean CCALD value was significantly higher in hypertensive compared to the normotensive subjects (0.000). The mean CCALD correlated positively with systolic and diastolic blood pressure in hypertensive females, while no significant correlations were observed among normotensives. Males in both cohort groups had significantly higher CCALD when compared to their female counterparts. The overall mean CCALD in hypertensive subjects was 6.31±1.07 mm and 6.09±0.94 mm for the right and left sides respectively, while normotensives had 6.05±0.79 mm and 5.71±0.70 mm for the right and left side, respectively.

Conclusion: In this study, the mean CCALD value in hypertensives is significantly higher compared to the normotensive subjects. Blood pressure levels have significant effects on CCALD in hypertensive female subjects.

Introduction

Hypertension (HTN) or high blood pressure is the leading preventable risk factor for cardiovascular diseases (CVD) and all-cause mortality globally.[1] Hypertension (HTN) is also known as the "silent killer" which is often asymptomatic, but can damage several important organs such as the blood vessels, heart, brain, and kidneys.[2] The hemodynamic hallmark of HTN is increased peripheral resistance, causing the heart to work harder than it should and this can occur through several pathways. Regardless of how the process begins, it appears to either follow or initiate vascular remodelling and hypertrophy in both large and small vessels.[3] Hypertension (HTN) may cause damage vessels due to increased shear stress and results in sub-endothelial exposure to numerous cell types that can stimulate inflammation, platelet adherence, the release of growth factors, and the replication of smooth muscle cells in the process of repair.[3] Atherosclerotic plaque forms in the intima-media of large blood vessels such as the carotid arteries. Such plaques can acutely rupture or gradually organize into occlusive lesions. In smaller vessels, hypertrophy, hyperplasia, and fibrosis may lead to widespread vascular occlusion.[3]

Arterial remodelling can be defined as the change in structural arterial properties through time in response to atherogenic or adverse hemodynamic alterations in the arterial environment,[4] which is manifested by carotid diameter enlargement (positive remodelling). The vascular remodelling is considered a compensation for initiation of atherosclerosis to preserve lumen diameter and restore normal blood flow. The condition or processes that may lead to the carotid arterial diameter enlargement including atherosclerosis,[5] blood pressure–related media thickening,[6,7] and arterial wall stiffening.[8] In addition, arterial diameter is known to be influenced by arterial wall thickness as well as age, race, sex, and anthropometric, metabolic, and hemodynamic parameters.[5,9] Carotid diameter enlargement may lead to an increase in circumferential wall stress.[10] An enlarged CALD has, therefore, been hypothesized as one of the important predictor of stroke.[11] Recent studies have shown that CCALD was associated with a higher risk of any cardiovascular event and mortality, despite adjusting for other carotid parameters such as arterial stiffness and pulse wave velocity.[12]

Most of the information on the development and progression of atherosclerosis is obtained using conventional or computed tomographic angiography, which are invasive, not readily available, accessible and affordable, especially in a resource constrained developing nation like ours. However, the development of ionizing radiation free, cheap, available and non-invasive imaging techniques such as the B-mode carotid artery ultrasound provides a mechanism for studying the evolution of atherosclerosis including plaque characterization. Despite these developments, there is a paucity of literatures on B-mode ultrasound evaluation CCALD in adults with HTN in the study locality. Therefore, this study is aimed at comparing the CCALD in hypertensive and normotensive subjects.

Methodology

This is a prospective, case-control study conducted at the ultrasound unit of the Maimusari clinic between October 2021 and January 2022. A total of 332 individuals comprising 188 hypertensive and 144 normotensive adult volunteers were recruited in to this study. Informed consent to participate in the study was obtained from all the participants. The participant's age, disease conditions, disease history and lifestyle were documented.

The participants' height in metres (m) was obtained using a stadiometer with the patient standing erect and backing the scale. A weighing scale was used to measure weight (kg) and calculate body mass index (BMI), body adiposity index (BAI) and body surface area were calculated. Fasting blood sugar (FBS) level and blood pressure measurements were also obtained and documented.

Non-probability (purposive) sampling method was adopted and participants were selected among the patients attending general outpatient clinic. Ethical approval was obtained from the Ethical committee of the state ministry of health.

Inclusion criteria

Individuals of either gender diagnosed with primary HTN, between the ages of 18 and 80 years and consented to participate voluntarily were included in the study. The normotensive subjects are apparently healthy individuals either gender non-pregnant at that moment and or without any history disease condition that might affect the morphology and function of the carotid artery. They were recruited from the general population and their age and gender distribution is similar to the hypertensive subjects.

Exclusion criteria

Hypertensive individuals who are either less than 18 or greater than 80 years of age and adults with a history of stroke or diabetes mellitus (DM) were excluded. Pregnant women were also excluded because of physiological changes and associated dilatation of the carotid artery during pregnancy. Normotensive individuals with a history of DM, HTN, or any clinical features suggestive of vasculitis were excluded.

Sonographic examinations

At the commencement of the examination, the researcher asked each participant to remove jewelleries and any other ornaments around the neck. The carotid artery was examined with the participant lying in supine position, right to the Sonographer/researcher on the ultrasound couch. The neck was hyper-extended (30^{o}) and placed on a small thick cylindrical foam pad to ensure adequate exposure of the neck depending on the subject's body physique. The head was then turned away from the examined side at about 45^{o} from the midline to the opposite side. An ultrasound gel was applied to the antero-lateral aspect of the neck along the anterior border of the sternocleidomastoid muscle from the root of the neck to the base of the skull. This is to ensure proper transducer-skin contact and to reduce friction between the two surfaces. At the beginning of the examination, the carotid arteries were evaluated in B-Mode with appropriate optimizing factors.

The study protocol involved scanning the far wall and lumen of the right and left carotid artery which span from the superior aspect of the clavicle to the angle of the mandible while the internal jugular vein was used as a window as described in the previous study (Takiuchi *et al*., 2014). Sonograms of the carotid arteries were obtained for measurements using two antero-lateral scanning views (transversal and longitudinal) for each of the carotid artery. A transversal scanning view of the carotid vessel from the root of the neck to the carotid bulb and to the base of the angle of the mandible (C3 vertebra) was performed to localize any plaque. A transversal scanning view of the vessel with the transducer placed anterior border of the sternocleidomastoid muscle (1 cm below the carotid bulb) to measure the CCALD. A single measurement was recorded at each location for CCALD, which was taken as the distance between the leading edges of the lumen intima interface and the media-adventitia interface (second bright line) of the far wall. All the measurements were taken at the diastolic phase of the cardiac cycle.

All the sonographic examinations were performed using the 7-12 MHz, multi-frequency linear array transducer (contact area; 8mm x 28mm) of high resolution, touch screen, Venue 50 ultrasound machine (GE Medical System:2014, made in China) equipped with an electronic callipers. High-frequency transducer (vascular custom preset) was used because it gives a better resolution for superficial structures such as the carotid artery.

Statistical analysis

The quantitative variables are expressed as mean ± standard deviation, minimum and maximum values, while qualitative variables are presented as frequencies and percentages. The mean CCALD between hypertensive and normotensive subjects; male and female subjects were compared using an independent sample t-test. While the mean values for the right and left sides were compared using a paired sample t-test. The association between the CCALD and continuous variables, such the age, BMI, BSA, BAI, BP and FBS levels, were calculated using univariate (Pearson's correlation coefficient). The data was analyzed using Statistical Package for Social Sciences (IBM SPSS]) Version 22.0. All the statistical tests were approved by assuming a null hypothesis of no difference, a $p \leq 0.05$ was considered statistically significant.

Results

There were 332 subjects comprising 188 hypertensive and 144 normotensive subjects with an age range of 18-80 years. The mean ages of subjects were 52.49±15.31 years and 44.45±16.03 years for hypertensive

and normotensive, respectively. There were 70(37.23%) males and 118(62.77%) female hypertensive subjects, while normotensive had 81 (56.25%) males and 63 (43.75%) females. There was a predominance of females over male hypertensives, while there was a male predominance over female normotensives. The predominant age group for the hypertensive and normotensive was age group ≥68 and 38-47 years respectively (Table 1 and 2).

The overall mean CCALD for the hypertensive and normotensive subjects was 6.20±1.01 mm and 5.88±0.77 mm respectively. There was a statistically significant difference between the two subject groups (p=0.000) as presented in table 3. The overall mean CCALD for male and female hypertensive subjects was 6.50±1.12 mm and 6.02±0.89 mm respectively; the difference was statistically significant (p=0.000). Similarly, the mean CCALD for males and females in normotensives was 5.98±0.83 mm and 5.75±0.67 mm respectively; there was also a statistically significant difference (p=0.011). The mean CCALD values in males were higher compared to females in both groups as presented in tables 3 and 4. In hypertensive subjects, the mean CCALD for the right and left sides was 6.31±1.07 mm and 6.09±0.94 mm respectively, while in healthy controls, the mean CCIMT for right and left sides was 6.05±0.79 mm and 5.71±0.70 mm respectively; there was a statistically significant difference observed between the right and left sides in both the hypertensive and normotensive subjects, as presented in table 5.

In hypertensive subjects, the mean CCALD for the age group 18-27 and >68 years was 5.97±0.34 mm and 6.45±1.06 mm respectively. While normotensive subjects had 5.93±0.73mm and 5.87±1.06 mm. The CCALD values changes inconsistently up to the 4th decade of life but consistently increase with age from the 5th decade upward hypertensive subjects. While the mean CCALD in normotensive subjects change but not consistently with age (Table 6). The mean CCALD values in hypertensive subjects are compared with the corresponding normotensive subjects in all the age groups. There was a very poor or weak correlation between the age and CCALD among the hypertensive subjects (r= 0.094 and 0.121 for males and females respectively) and very weak negative correlation among the normotensive subjects (r=-0.117 and -0.020 for males and females respectively) as presented in table 7. The correlations between CCALD and other measured variables were not statistically significant except for SBP in male (r=0.253, p=0.035) and FBS in female hypertensive subjects (r=0.230, p=0.012) (Table 7).

In this study, carotid plaques were seen in the common carotid artery (CCA) wall of 7 (3.72%) hypertensive subjects, while only 1 (0.53%) case was seen in the CCA of normotensive subjects. These plaques were more common in males (5) than in females (2) hypertensives and are more evident in the right CCA (4) than in the left (3).

Table 1: Age and gender distribution for hypertensive subjects

Age (years)	Male (n=70)		Female (n=118)		Total (n=188)	
	Frequency (%)	Mean ± SD	Frequency (%)	Mean ±SD	Frequency (%)	Mean ±SD
18-27	3(1.6)	22.67±3.79	8(4.26)	21.25±2.55	11(5.85)	21.63±2.80
28-37	5(2.66)	32.00±1.58	13(6.91)	32.62±2.53	18(9.57)	32.44±2.28
38-47	7(3.72)	42.43±2.37	31(16.49)	41.45±2.25	38(20.21)	41.63±2.27
48-57	7(3.72)	52.29±1.89	30(15.96)	50.77±3.91	37(19.68)	51.05±3.64
58-67	22(11.7)	62.41±2.97	18(9.57)	61.33±2.63	40(21.28)	61.93±2.84
≥68	26(13.83)	73.75±3.96	18(9.57)	71.81±4.27	44(23.41)	72.93±4.13
Total	70(37.23)	59.7±15.38	118(62.77)	48.22±13.63	188(100)	52.49±15.31

Table 2: Age and gender distribution for normotensive subjects

Age (years)	Male (n=81)		Female (n=63)		Total (n=144)	
	Frequency (%)	Mean ± SD	Frequency (%)	Mean ±SD	Frequency (%)	Mean ±SD
18-27	13(9.03)	24.54±2.5	9(6.25)	21.88±2.62	22(15.28)	21.68±2.50
28-37	10(6.94)	31.9±3.31	18(12.5)	33.72±2.42	28(19.44)	33.07±2.85
38-47	20(13.89)	41.05±2.21	18(12.5)	41.44±2.71	38(26.39)	41.24±2.43
48-57	8(5.56)	52.75±2.66	12(8.33)	51.58±3.11	20(13.89)	52.05±2.92
58-67	18(12.5)	62.28±2.21	4(2.78)	62.32±36	22(15.28)	62.23±2.4
≥68	12(8.33)	72.92±3.48	2(1.39)	80.00±00	14(9.72)	73.93±4.1
Total	81(56.25)	47.38±25	63(43.75)	40.90±13.20	144(100)	44.45±16.03

Table 3: Comparison of mean CCALD between hypertensive and normotensive subjects

Measurements	Gender	Hypertensives	Normotensives	p-value
		Mean±SD	Mean±SD	
Mean CCALD (mm)	Male	6.50±1.12	5.98±0.83	0.000*
	Female	6.02±0.89	5.75±0.67	0.003*
Overall mean CCALD (mm)		6.20±1.01	5.88±0.77	0.000*

A p-value of < 0.05 is considered significant

Table 4: Comparison of mean CCIMT between male and female hypertensive and normotensive subjects

Subjects	Males	Females	p-value
	Mean±SD (mm)	Mean±SD (mm)	
Hypertensives	6.50±1.12	5.98±0.83	0.000*
Normotensives	6.02±0.89	5.75±0.67	0.011*

A p-value of < 0.05 is considered significant

Table 5: Comparison of mean CCALD between right and left sides in hypertensive and normotensive subjects

Subjects	Right	Left	p-value
	Mean±SD (mm)	Mean±SD (mm)	
Hypertensives	6.31±1.07	6.09±0.94	0.003
Normotensives	6.05±0.79	5.71±0.70	0.000

Table 6: Mean CCALD (mm) value with age group in hypertensive and normotensive subjects

Age group (Years)	Hypertensives	Normotensives
	Mean±SD	Mean±SD
18-27	5.97±0.34	5.93±0.73
28-37	6.01±0.74	5.94±0.55
38-47	5.92±1.18	5.77±0.65
48-57	6.14±0.97	5.75±0.72
58-67	6.41±0.96	6.04±1.02
≥68	6.45±1.06	5.87±1.06

Table 7: Correlations between CCALD and some variables in hypertensive and normotensive subjects

Variables	Gender	Hypertensives		Normotensives	
		r	*p*	*r*	*P*
Age (Years)	**Male**	0.094	0.438	-0.117	0.299
	Female	0.121	0.192	-0.020	0.875
Height (m)	**Male**	0.118	0.332	0.032	0.777
	Female	-0.069	0.460	0.133	0.300
Weight (kg)	**Male**	0.229	0.056	0.076	0.502
	Female	0.009	0.921	0.020	0.877
BMI (kg/m^2)	**Male**	0.119	0.325	0.167	0.137
	Female	-0.026	0.780	0.026	0.839
BSA (m^2)	**Male**	0.089	0.466	0.115	0.307
	Female	-0.113	0.224	0.019	0.882
BAI (%)	**Male**	0.214	0.076	0.025	0.825
	Female	-0.002	0.984	-0.030	0.815
SBP (mmHg)	**Male**	0.253	0.035	0.062	0.585
	Female	0.022	0.8166	0.125	0.328
DBP (mmHg)	**Male**	0.032	0.790	0.045	0.691
	Female	-0.118	0.204	-0.083	0.516
FBS(mmol/L)	**Male**	-0.017	0.891	0.085	0.451
	Female	0.230	0.012	0.146	0.253

Hint: BMI= body mass index, BSA= body surface area, BAI=body adiposity index, SBP=systolic blood pressure, DBP=diastolic blood pressure, FBS=body surface area.

Discussion

Hypertension (HTN) is causes both structural and functional changes in the arterial wall which serve as an early hallmark of the hypertensive disease process and may be a reliable guide for prognosis.[13] An enlarged CCALD is closely associated with the morphological changes generally proceeding in tandem, which produces a complex relationship between the two parameters and atherosclerosis.[14] Arterial wall thickening may be in the intima-media or muscular layer. As the carotid artery is elastic, the muscular layer is relatively small. Hence, the thickening of the carotid arterial wall is essentially due to intima-media thickening.[15]

This study tends to find out whether HTN had a significant influence on the CCALD. This study involved a total number of 188 hypertensive and 144 normotensive subjects.

In this study, the mean age of the hypertensive and normotensive subjects was 52.49±15.31, years and 44.45±16.03 years, respectively. The middle-aged population preponderance noted in this study was because majority of people with HTN in sub-Saharan Africa are in that age range.[16] This shows that the controls are of lower age group compared to the study group. In addition, most of the consenting subjects in the study fall within that age group. There was also a female preponderance among hypertensive subjects.

These findings are in agreement with the earlier studies.[17,18] Male preponderance was also noted among the normotensive subjects in this study, which is in agreement with the earlier studies.[19,20]

The current study found that the overall CCALD value in hypertensive subjects was significantly higher compared to those of normotensive subjects. This finding is in agreement with virtually all the previous studies reviewed.[21-25] None of the reviewed literatures has documented contrary findings. These findings confirmed the fact that high blood pressure is a major determinant of CCALD irrespective of race, region, or gender. However, the mean values documented by Ayoola, et al[23] in Northwestern Nigeria for the two cohort groups (10.00mm and 7.00 mm) are far higher than other, studies including the index study. The reasons could be variations in sampling method, stage of the HTN, treatment status, measurement method, interobserver variability diet, life style and genetic factors among other factors. Higher mean CCALD values among hypertensive subjects documented was as a result of pulsatile pressure which is known to be most important factor generating an increased CCALD, [26] and enlargement of large arteries may be attributed to the fracture of the load-bearing elastin fibres of the vessel wall in response to the fatiguing effect of tensile stress.[7,27]

According to this study, the mean CCALD values on the right side were significantly compared to the left in both the hypertensive and normotensive subjects. This finding is in line with the findings of the previous researchers.[28,29] main reasons for such variation between the two sides is still not clear. However, the possible explanation for such variation could be that the left common carotid artery (LCCA) is a direct branch of the thoracic aorta while the right common carotid artery (RCCA) is a branch of the brachiocephalic artery which is a direct branch of the thoracic aorta. Hence, differences any have existed in arterial growth between the two sides and/or that flow-mediated mechanical forces applied to the carotid wall also differ between the right and the left sides. However, the right and left CCALD values documented by Cookey, *et al*[29] in Port Harcourt, are higher than that of the index study. The possible reason for this variation could as a result of differences in the sample size, measurement techniques, diet, study design, duration of the disease condition, medication and lifestyle among other factors.

This study also revealed that the mean CCALD value in male was higher than those of females in both the hypertensive and normotensive subjects. This finding is in tandem with the studies by Cipolli et al,[30] who reported mean CCALD values of 6.7±0.1 mm and 6.1±0.1 mm for male and female hypertensive subjects. Similar findings on normotensive subjects have been reported by some researchers.[31,32,33] None of the reviewed literatures has reported a contrary finding in terms of gender. This might be explained by the gender difference in the development of atherosclerosis. These findings imply that the chance of developing atherosclerosis was higher in males compared to females, although the precise mechanism of the gender difference carotid artery dimensions cannot be elucidated from the present data and remain unclear but might be attributed to the fact that males are more exposed to emotional psychological and environmental stress than females.[34]

Very weak positive and negative correlations were observed between age and hypertensive and normotensive subjects respectively. This finding is somehow similar to that of Bénétos, et al[35] who reported a weak negative correlation between CCALD and age among hypertensive subjects. On the contrary, some other studies normotensive subjects have reported that the CCALD increases with age significantly in the general population.[36,37,38] This variation in observations might be due to differences in participant sample size, methodology, diet, lifestyle or age range. The reported increase in CCALD with age may be secondary to endogenous physiological mechanisms that favour enlargement of the arterial.

Conclusion

This study revealed that the mean CCALD value in hypertensive subjects is higher than those of normotensives. The study also noted that the relationship between the age and CCALD was very weak in both hypertensive and normotensive subjects. High resolution ultrasound is a cheap, affordable, non-invasive, reliable, readily available and reliable imaging modality that is useful in monitoring HTN and its associated complications.

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Confects of interest

There are no conflicts of interest.

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