

# Relationship between Lipid profile and Carotid artery parameters among Mgbuoba community dwellers, Port Harcourt, Nigeria: A random survey

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#### Abstract

**Background:** An important variable in apparently normal individuals that may affect findings on duplex carotid scan is their lipid numbers. In developing countries like Nigeria, routine health checks are not upheld and where available are not exhaustive. Carotid intima media thickness (cIMT) and cholesterol have been implicated as risk factors for stroke. Studies in diseased states have shown an association between lipid numbers and carotid IMT. Asymptomatic apparently healthy subjects are rarely targets for carotid evaluations except in limited circumstances. Some apparently healthy individuals may already have abnormal lipid numbers; thus this study seeks to assess the association between carotid parameters and lipid profile amongst apparently healthy subjects.

**Method:** This cross sectional, community-based study, assessed the extra-cranial common and internal carotid artery variables in 74 randomly selected apparently healthy individuals, from Mgbuoba community, Port Harcourt Rivers State. Four extra cranial vessels: left common carotid (LCC) and right common carotid (RCC) left internal carotid (LIC) and right internal carotid (RIC) were evaluated using a predefined protocol to assess parameters such as vessel diameters, peak systolic velocities (PSV), end diastolic velocities (EDV), resistivity index (RI) and intimal media thickness (cIMT). Fasting venous blood samples were collected and analysed using a Colour Spectrophotometer for Total Cholesterol (TC), High Density Lipoprotein (HDL), Low Density Lipoprotein (LDL) and Triglyceride (TG). The mean values for the common carotid artery variables, Total Cholesterol (TC), Low density Lipoprotein (LDL), High Density Lipoprotein (HDL) and Triglycerides (TG).

**Result:** Data was collected on Excel spread sheet and analyzed using SPSS 23 statistical software package. Seventy-four (74) subjects comprising 30 (41%) males and 44(59%) females respectively were studied. Their mean age  $\pm$  SD was 38.60  $\pm$ 12.56yrs. Mean Systolic blood  $\pm$ SD and diastolic blood pressure $\pm$ SD were 122.94  $\pm$  12.64mmHg and 75.41  $\pm$  9.20 mmHg respectively. There was negative correlation between total cholesterol and LCC PSV (-.31,.03), negative correlation between HDL and LCC diameter (-.29,.03) and positive correlation between LDL cholesterol and IMT (.30,.04). On the RCC there was a negative correlation between HDL and RI (-.38,.02). on the LIC there was negative correlation between TG (-0.33,.05)). Plaques were identified in 2 with only mild stenosis.

**Conclusion:** The impact of cholesterol on the carotid artery parameters is not just in symptomatic or diseased states but also in apparently normal individuals. It is an important area and more studies are encouraged. It will be pertinent to explore this in a larger study population. An important area would be the impact of HDL on vessel distensibility.

#### Keywords

Carotid artery, Lipids, Cholesterol, cIMT, Triglycerides, HDL, LDL, TC, Plaques

#### Introduction

Carotid IMT, plaque and dyslipidaemia are individual risk factors for stroke and other cardiovascular diseases<sup>1,2</sup>. The American college of cardiology (ACC) has not recommended screening using carotid scan, for asymptomatic healthy adult population<sup>3</sup>. Several factors

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have been shown to increase the risk for carotid artery stenosis, including older age, male sex, hypertension, smoking, hypercholesterolemia, diabetes, and heart diseases<sup>4.5</sup>. Studies have shown the relationship between lipid numbers and carotid IMT amongst diseased patients<sup>6,7</sup>. However, studies on the relationship with cholesterol numbers and other carotid artery variables in apparently normal individuals are few. This study assessed the relationship between Carotid IMT and other doppler variables with lipid numbers. Carotid vascular scan is cheap, readily available and considering the huge burden of stroke in the Nigeria population, there is a growing need to include this investigation as a screening tool to prevent debilitating consequences of atherosclerotic disease in patients at high risk of cardiovascular disease.

# Method

**Study design:** This is a cross-sectional survey that took place in ad-hoc laboratory created in a room at the community hall after ethical clearance gotten from the Community leaders. Held in Mgbuoba community from September 2018 to October 2018. Using the Sonoscape SS1 8000 which is a duplex high-definition scanner with a 712(5mHz -12mHz) transducer.

**Sample Size:** the sample size for the population of Mgbuoba community was obtained using the Taro Yamani Formula<sup>8</sup>  $n = \frac{N}{1 + Ne^2}$  Where, n= sample size required, N= the finite population, 1= constant, e= level of precision or sampling error (0.05).

The population of the Mgbuoba community from a projected estimate for 2019 was 7303.9

from the above formula with finite N= 7303 $7303/1 + 7303(0.05)^2 = 40$ 

*Subjects:* The study assessed the extra-cranial common carotid artery variables in 74 randomly selected apparently normal individuals, from Mgbuoba in Obio/Akpor community of Rivers State, two extra cranial vessels: left common carotid, and right common carotid were evaluated using a predefined protocol to involve the vessel diameters, the peak systolic velocities (PSV), the end diastolic velocities (EDV), resistivity index (RI) and intimal media thickness (IMT)

Subjects had their demographic data and their anthropometric measurements (patients had their weight and height assessed and body mass indices calculated by dividing their weights in kilograms by the square of their heights). Blood pressure was measured, and blood glucose measured to exclude diabetes mellitus.

All subjects were educated on the procedure and consent obtained required to fill a questionnaire with

information on baseline demographics and history for smoking and alcohol consumption noted. Blood glucose was carried out on all subjects and blood pressure measured

*Inclusion Criteria:* Non hypertensives, non-diabetics, non-smokers were recruited.

Duplex carotid vascular scan was performed using a high frequency linear probe that gives high resolution (7-15mHz). Scanning employed both 2D real time imaging and still M-mode cuts for structural evaluation and the added value of colour flow and spectral that aids elimination of artefacts and help unmask hypoechoic plaques.

**Scanning Procedure:** Examination was started proximally in transverse and followed distally to the bifurcation and beyond this to assess the presence of any intimal thickening or plaque. This was repeated in longitudinal plane to follow the course and measure intimal thickness as well as velocity of flow. Then colour flow was used to aid in assessing patency and direction of flow. The origins of the ICA were noted, and their course followed. The pulse wave doppler and IMT was taken as the mid part of each vessel. Two values for Vessel diameter and IMT was taken, and two successive spectral wave was taken for PSV, EDV, RI and S/D and the average calculated.

Mosaic flow patterns, filling defects, retrograde flow, high peak systolic flow velocities, thickening of the intimal medial thickness (IMT), vascular plaques and absence of flow through a vessel are pointers to vascular diseases<sup>10-12</sup>. Care was needed to interpret the presence of a plaque; usually more than 1 of the points were employed in interrogation of a plaque<sup>13</sup>. Plaques can be defined as hypoechoic (echolucent) or hyperechoic (echogenic) depending on the echogenicity14,15. The echogenicity usually is a measure of its calcium content<sup>16</sup>. It can also be classified as homogeneous or heterogeneous depending on the uniformity of its echogenic pattern. Plaque stability has been judged by its echogenicity. Hypoechoic or Ecolucent plaques have been shown to be associated with increased stroke<sup>14,15</sup>. Data was collected on Excel spread sheet and analysed using SPSS 23 statistical software package.

*Statistical Analysis:* Data was collected on Excel spread sheet and analysed using SPSS 23 statistical software package. The general mean for the population and sex differences was obtained for carotid artery variables and the mean cardiovascular parameters and fasting lipid numbers. They were expressed as tables. Pearson correlation coefficient was used to assess the

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presence of	plaques was		Variable	Min.	Max.	Mean ±SD
			RCC/DIAM (cm)	.46	.93	$0.70 \pm 0.01$
e of abnorr	nal lipid vai	hables was also	RCC/IMT (cm)	.02	.11	$0.07\pm0.02$
			RCC/PSV (cm/s)	.40	88.63	58.29 ±18.56
			RCC/EDV (cm/s)	3.61	29.80	$11.20 \pm 5.25$
			RCC/RI	0.50	0.98	$0.76 \pm 0.09$
ean age of	the particip	ants was 38.60	RCC/SD	2.31	9.80	4.78 ±1.69
ere 127.29	± 9.97 and	$1\ 76.04\ \pm\ 8.15$	LIC/DIAM (cm)	.46	1.15	0.75 ±.15
there was significant difference between mean ages for the sexes at 95% confidence interval (1.58,6.03) (The males were older with mean age $37.29\pm11.23$ yrs and				.040	.16	$.065 \pm .02$
				19.03	86.99	44.52±.32
			LIC/EDV	4.00	21.29	$10.79 \pm .13$
: Mean of	Study Var	iables:	LIC/RI	.55	10.77	.94 ± 1.42
Min.	Max.	Mean	—LIC/SD	2.20	10.79	$4.16 \pm .70$
			RIC/DIAM	.58	.97	.75 ± .10
17.63				.02	.11	$0.06 \pm .02$
				10.02	0140	
				19.83	94.18	$42.68 \pm 15.98$
				2.25	25.27	$11.27 \pm 5.67$
			·	2.23	23.27	11.2/ ± 3.0/
			· · ·	02	91	$0.69 \pm .17$
	35.32 .98	$13.65 \pm 5.50$ $0.76 \pm 0.08$	RIC/SD		11.59	$4.54 \pm 2.44$
.59				1.00		
	subjects considered above alles accourse ean age of systolic blo ere 127.29 by. For the int differen confidence with mean confidence with mean co.48yrs.) show any s : Mean of Min.	subjects consented to hales accounting for 53 ean age of the particip bystolic blood pressure ere $127.29 \pm 9.97$ and by. For the cardiovascu int difference between confidence interval (1 with mean age 37.29 to.48yrs.) others the show any significant d the	confidence interval (1.58,6.03) (The with mean age 37.29 $\pm$ 11.23yrs and (6.48yrs.) others cardiovascular tshow any significant differences. (See <b>Min.</b> Max. Mean $\pm$ SD19.090.038.60 $\pm$ 12.5617.6337.6526.11 $\pm$ 4.5490.0143.0122.94 $\pm$ 12.6458.092.075.41 $\pm$ 9.20.470.8700.67 $\pm$ 0.09.020.1800.07 $\pm$ .0220.2897.8157.11 $\pm$ 15.88	bresence of plaques was noted and the e study population computed, in ge of abnormal lipid variables was also subjects consented to the study; 30 males accounting for 53% and 46.9% ean age of the participants was 38.60 bystolic blood pressure and diastolic ere 127.29 $\pm$ 9.97 and 76.04 $\pm$ 8.15 by. For the cardiovascular parameters int difference between mean ages for confidence interval (1.58,6.03) (The with mean age 37.29 $\pm$ 11.23yrs and c6.48yrs.) others cardiovascular is show any significant differences. (See <b>IIC/PSV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/s) <b>IIC/EDV</b> (cm/	bresence of plaques was noted and the e study population computed, in ge of abnormal lipid variables was also RCC/DIAM (cm) .46   RCC/IMT (cm) .02   RCC/IMT (cm) .02   RCC/EDV (cm/s) .40   RCC/EDV (cm/s) .40   RCC/EDV (cm/s) 3.61   subjects consented to the study; 30 RCC/RI 0.50   ales accounting for 53% and 46.9% RCC/SD 2.31   ean age of the participants was 38.60 RCC/SD 2.31   bystolic blood pressure and diastolic ere 127.29 $\pm$ 9.97 and 76.04 $\pm$ 8.15 LIC/DIAM .46   (cm) LIC/IMT .040   (cm) .1C/PSV 19.03   c6.48yrs.) others cardiovascular cm/s)   t show any significant differences. (See LIC/PSV 19.03   (cm/s) LIC/RI .55   Min. Max. Mean LIC/SD 2.20   19.0 90.0 38.60 $\pm$ 12.56 (cm) .55   19.0 90.0 38.60 $\pm$ 12.64 (cm) .58   19.0 90.0 38.60 $\pm$ 12.64 (cm) .58   19.0 <t< td=""><td><math display="block">\begin{array}{c c c c c c c c c c c c c c c c c c c </math></td></t<>	$\begin{array}{c c c c c c c c c c c c c c c c c c c $

Table 2. Comparison between Male and Female Clinical Parameters

Clinical Parameters	Mean ± SD Males	Mean ± SD Females	SE of Mean	t	Sig (2 tailed)
AGE	37.29 ±11.23	$33.48 \pm 6.48$	1.09	3.49	.002
$BMI(kg/m^2)$	25.45 ± 3.45	26.60 ±4.99	-1.07	1.07	.293
PULSE(beats/min)	$71.69 \pm 9.86$	$78.41 \pm 9.22$	2.39	-2.81	.009
SBP(mmHg)	$122.46 \pm 11.74$	$122.07 \pm 14.36$	3.32	.12	.907
DBP(mmHg)	$74.00\pm9.75$	$75.68 \pm 9.66$	2.31	73	.473



The mean  $\pm$  SD for all parameters is as shown on table 1. Mean  $\pm$  SD of LCC IMT for the population was 0.07  $\pm$  0.02cm, mean  $\pm$  SD for LIC; 0.75  $\pm$ .15cm, and mean  $\pm$  SD of RCC IMT; 0.07  $\pm$  0.02. mean  $\pm$  Sd for RIC; .07  $\pm$  .02cm.

However, some subjects showed IMT greater than 0.09cm: 7 had IMT greater than 0.09cm on the LCC (4 males and 3 females). Similarly, on RCC 7 also had IMT greater than 0.09 (5 females and 2 males). (See table) The mean total cholesterol was  $4.25 \pm 0.84$ mmol/l with 10(12.8%) having values higher than normal range) and mean triglycerides was  $2.42 \pm 1.0$ mmol/l with 1 (1.3%) having a value higher than normal range and mean low density lipoprotein cholesterol (LDL) was  $2.24 \pm 0.67$ mmol/l with 13 (16.7%,) subjects having values higher than normal range and the mean High density lipoprotein cholesterol (HDL)was  $1.45 \pm 0.38$ mmol/l with 19 (23.3%,) subjects having values lower than normal range, (See Table 3). Plaques were only seen in two who had elevated TC and LDL with hyperechoic plaques.

Tables four to six (4-6) demonstrates the correlation between Lipid numbers and Carotid artery variables. **Table 3.** Percentage with abnormal Cholesterol in population studied

Cholesterol(mmol/l)	No with mean > or < than normal for Population	Total No studied	Percentage of Study Population
Total Cholesterol	10	74	12.8%
Triglycerides	1	74	1.3%
Low Density Lipoprotein	13	74	16.7%
High Density Lipoprotein	19(<)	74	19%

#### Table 4. Correlation between lipids and LCC parameters

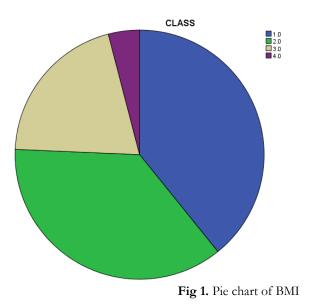
		LCC/DIA M (cm)	LCC/IM T (cm)	LCC/PSV (cm/s)	LCC/E DV (cm/s)	LCC/RI	LCC/SD
AGE (yrs)	Pearson Correlation	.113	.194	224	027	107	032
	Sig. (2-tailed)	.344	.118	.060	.833	.417	.803
BMI(kg/m <sup>2</sup> )	Pearson Correlation	.170	.094	206	129	130	044
	Sig. (2-tailed)	.150	.450	.083	.306	.319	.729
TC(mmol/l)	Pearson Correlation	178	.127	310*	.014	054	038
	Sig. (2-tailed)	.232	.402	.034	.926	.735	.809
TG(mmol/l)	Pearson Correlation	158	039	127	.026	068	120
	Sig. (2-tailed)	.288	.799	.394	.864	.667	.448
LDL(mmol/l)	Pearson Correlation	.014	.304*	249	132	.125	.208
	Sig. (2-tailed)	.924	.040	.091	.381	.429	.187
HDL(mmol/l)	Pearson Correlation	293*	139	143	.131	091	112
	Sig. (2-tailed)	.046	.359	.338	.387	.565	.482



#### Table 5. Correlation between lipids and RCC parameters

		RCC/DIA M (cm)	RCC/IM T (cm)	RCC/P SV (cm/s)	RCC/ED V (cm/s)	RCC/ RI	RCC/ SD
AGE(yrs)	Pearson Correlation	.239	.333*	083	.125	071	114
	Sig. (2-tailed)	.076	.014	.541	.356	.601	.402
BMI(kg/m <sup>2</sup> )	Pearson Correlation	.025	.219	225	.081	287*	317*
	Sig. (2-tailed)	.854	.112	.093	.548	.030	.017
TC(mmol/l)	Pearson Correlation	084	.266	.037	.271	227	233
	Sig. (2-tailed)	.616	.107	.821	.095	.164	.154
TG(mmol/l)	Pearson Correlation	.007	.087	182	.079	298	279
	Sig. (2-tailed)	.965	.602	.267	.633	.066	.086
LDL(mmol/l)	Pearson Correlation	049	.071	.191	.147	.095	014
	Sig. (2-tailed)	.771	.670	.244	.373	.566	.931
HDL(mmol/l)	Pearson Correlation	094	.185	082	.170	377*	219
	Sig. (2-tailed)	.574	.266	.622	.302	.018	.181

Table 6. Correlation between lipids and LIC parameters





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		LIC/DI	LIC/I	LIC/PSV	LIC/ED	LIC/R	LIC/SD
		AM	MT	(cm/s)	V	I	
		(cm)	(cm)		(cm/s)		
BMI(kg/m <sup>2</sup> )	Pearson Correlation	.224	.026	.017	.038	035	264
	Sig. (2-tailed)	.076	.850	.902	.784	.809	.064
AGE(yrs)	Pearson Correlation	.270*	.192	.095	028	.251	.095
	Sig. (2-tailed)	.031	.156	.492	.840	.079	.510
TC(mmol/l)	Pearson Correlation	.227	051	.035	.083	117	008
	Sig. (2-tailed)	.153	.763	.838	.627	.518	.967
TG(mmol/l)	Pearson Correlation	.031	.067	226	330*	.098	054
	Sig. (2-tailed)	.847	.688	.178	.046	.587	.765
LDL(mmol/ l)	Pearson Correlation	.284	024	.184	.191	083	.193
	Sig. (2-tailed)	.072	.887	.276	.257	.645	.282
HDL(mmol /l)	Pearson Correlation	.091	.027	.028	.006	156	.053
	Sig. (2-tailed)	.572	.873	.871	.973	.386	.769

The BMI of the subjects were classified into 5 Classes: Class 0: those with BM<  $18.5 \text{kg/m}^2$ , Class 1: BMI  $18.5 \text{kg/m} - 24.9 \text{kg/m}^2$ , Class 2: BMI  $25-29.9 \text{kgm}^2$  and Class 3: BMI:  $30 - 34.9 \text{kg/m}^2$  and Class  $4 \ge 35 \text{kg/m}^2$  Graphs were used to demonstrate the impact of BMI on the Carotid artery diameters and IMT. Sex differences of Carotid parameters is also demonstrated as graphs.

<b>Table 7.</b> Conclation between Explos and ECC parameters	Table 7. Correlation	between Li	pids and LC	C parameters
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Variable		RIC/DIA M (cm)	RIC/IM T (cm)	RIC/PSV (cm/s)	RIC/ED V (cm/s)	RIC/RI	RIC/S D
Age (yrs)	Pearson Correlation	.413**	.417**	035	302*	.203	.398**
	Sig. (2-tailed)	.003	.004	.821	.046	.187	.007
BMI(kg/m <sup>2)</sup>	Pearson Correlation	142	035	.030	057	.033	.119
	Sig. (2-tailed)	.319	.816	.849	.713	.830	.442
TC(mmol/l)	Pearson Correlation	008	.052	036	.158	233	051
	Sig. (2-tailed)	.964	.778	.851	.404	.216	.791
TG(mmol/l)	Pearson Correlation	.132	197	021	010	.007	066
	Sig. (2-tailed)	.458	.280	.914	.958	.971	.728

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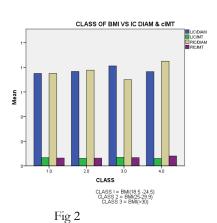
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LDL(mmol/l)	Pearson Correlation Sig. (2-tailed)	.164 .353	036 .845	.118 .534	.149 .433	084 .660	.013 .944	
HDL(mmol/l)	Pearson Correlation Sig. (2-tailed)	231 .189	.137 .455	.038 .842	.055 .772	077 .685	040 .833	

Table 10. Representation of population by class of BMI

	_				
		Frequency	Percent	Valid Percent	Cumulative
			(%)	(%)	Percent (%)
	1.0	33	42.3	42.3	42.3
	2.0	27	34.6	36.5	75.7
	3.0	15	19.2	20.3	95.9
	4.0	3	3.8	4.1	100.0
Total		78	100.0		



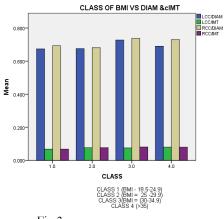
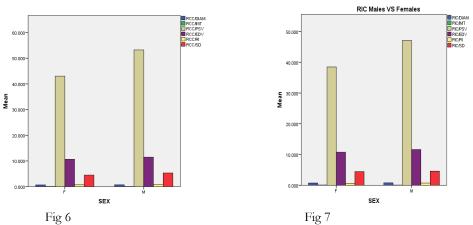




Fig 4

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#### Discussion

Carotid artery parameters like the PSV, EDV, the RI and SD have been associated with cardiovascular disease <sup>16,17,18</sup>. There is the need to further explore the use of carotid doppler as a screening tool in apparently healthy individuals with dyslipidaemia and understand the relationship between cholesterol and carotid vascular disease, this will further explain the impact of cholesterol on cardiovascular disease. Carotid IMT has been as a surrogate for cardiovascular disease<sup>17</sup>. The mechanisms by which cholesterol affects the carotid doppler velocities not yet fully understood. The present study examined the relationship between different cholesterols and carotid artery parameters, to include doppler velocities.

Due to the cross-sectional nature of this study, no causal relationship could be proved, as this will require longitudinal investigation, and this represents one limitation of our research<sup>19</sup>. Another limitation was the small sample size. This study demonstrated that apparently normal individuals showed abnormal lipid numbers in varying proportions of the types of cholesterol. 19% had abnormally low HDL cholesterol, high LDL was found in 16.7%, high Total Cholesterol was high in 12.8% and high triglycerides found in 1.2%. This can be further buttressed by the higher percentage of study population of 53% being with BMI equal or greater than 25.

The males had larger vessel diameters, thicker cIMT and higher doppler velocity as has been corroborated in other studies. There was negative correlation between total cholesterol and LCC PSV (-.31,.03), negative correlation between HDL and LCC diameter (-.29,.03) and positive correlation between LDL cholesterol and IMT (.30,.04). On the RCC there was a negative corelation between HDL and RI (-.38,.02) on the LIC there was negative corelation between TG (-0.33,.05)). Plaques were identified in 2 with only mild stenosis.

The Chinese study by Hou et al <sup>20</sup> showed that lipid parameters related more with plaques from a univariate analysis, but a multivariate analysis from the same study showed that gender, age diastolic blood pressure and total cholesterol showed more relationship with carotid plaques and age, gender systolic blood pressure related more with carotid IMT. This could imply how different cardiovascular parameters affects the formation of plaques and increased IMT differently. Understanding these individual predictors will guide better understanding of disease and possibly management modalities.

Studies in the past have shown the atherogenic nature of LDL and anti-atherogenic nature of HDL<sup>21</sup>. Triglycerides has also been implicated as a predictor of mortality and morbidity in coronary heart disease.<sup>22</sup> Wei et al<sup>23</sup> demonstrated that the formation of atherosclerosis was from an imbalance between the pro-atherosclerotic LDL and the anti-atherosclerotic HDL. They were able to demonstrate that in low stress environment, the increase in LDL outweighs the increase in HDL resulting in atherosclerosis and that in high stress environment, the increase in LDL with resultant protective effect against atherosclerosis. The high stress and low stress environments in this study was determined by the flow velocities of the vessels.

The Wei research study showed the relationship between peak velocities with LDL and HDL. This would

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favour the thought that flow velocities would show a relationship with LDL and HDL.

This was corroborated by the index study that showed a negative correlation between the peak systolic velocity of the left carotid artery and the total cholesterol. HDL showed a negative corelation with the LCC diameter and increased LDL was associated with increase in cIMT. This corroborates the protective nature of HDL cholesterol and the atherogenic effect of LDL.

The protective role of HDL will be an interesting area, other studies performed in children have assessed the role of HDL in vascular distension<sup>24</sup>. The negative correlation may imply a role of HDL in vascular distensibility and its anti-atherogenic function a preventing vascular stiffness.

## Conclusion

The impact of cholesterol on the carotid artery parameters is not just in symptomatic or diseased states but also in apparently normal individuals. It is an important area and more studies are encouraged. It will be pertinent to explore this in a larger study population. An important area would be the impact of HDL on vessel distensibility.

#### Authors' contribution

Please provide roles played by each author around conceptualization, planning, data collection, interpretation of the result, manuscript preparation, proofreading, and approval of the final manuscript.

# **Conflict of Interest**

Authors declared no conflict of interest.

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