Original Article

Magnetic Resonance Imaging Infarct Volume Correlates with Carotid Intima–Media Thickness and Plaque Echotexture in Ischemic Stroke

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Abstract

Objective: To determine the correlation between cerebral infarct volume, carotid intima–media thickness (CIMT), and plaque echotexture in patients with ischemic stroke. **Materials and Methods:** This was a cross-sectional study of 70 patients with ischemic stroke who were imaged using the head coil of a 1.5T Toshiba magnetic resonance machine. The volumes of infarcts were documented and calculated using the manual tracing of the infarct perimeter method. The common carotid CIMT was measured on ultrasound using a linear high-frequency 7.5 MHz transducer. **Results:** Seventy subjects were evaluated. The mean magnetic resonance imaging cerebral infarct volume was 8.07% volume. Hyperechoic plaques were the most prevalent (36.7%) compared to the hypoechoic (33.3%) and isoechoic (30%) plaques. There was a moderate positive correlation between CIMT and infarct volume ($r = 0.70$; $P = 0.001$) in the entire study population. Similarly, positive correlations between CIMT and infarct volume were recorded in both the male ($r = 0.73$; $P = 0.001$) and female ($r = 0.67$; $P = 0.001$) subjects. Furthermore, subjects who presented in the acute phase (1–3 days) of ictus showed a moderate positive correlation ($r = 0.621$; $P = 0.0001$) between CIMT and infarct volume, while there was a strong positive correlation ($r = 0.74$; $P = 0.0001$) in subjects that presented in the subacute phase (4–7 days). **Conclusion:** Common carotid artery CIMT correlated positively with cerebral infarct volume in patients with ischemic stroke. Furthermore, hyperechoic plaques were associated with significantly larger infarct volumes compared to hypoechoic and isoechoic plaques.

Keywords: *Carotid intima–media thickness, hypertension, infarct volume, stroke*

Introduction

Systemic hypertension (HTN) is a known cause of atherosclerosis that leads to thickening of the arterial walls. Arterial wall thickening is an important cause of end-organ damage (myocardial infarction, stroke, etc).[1]

Carotid intima–media thickness (CIMT) is an established index with a strong independent positive correlation to HTN. CIMT increases with the severity and duration of HTN.[2] The normal CIMT values for young (20–30 years) and older (>60 years) individuals are ≤0.5 and ≤0.9 mm, respectively. Values between 0.9 and 1.4 mm are regarded as thickening, while CIMT > 1.4 mm indicates atheromatous plaques.[3] Increased CIMT is a marker of subclinical atherosclerosis and a reliable independent marker of cardiovascular disease.[1,4]

The 11th revision [International classification of diseases (ICD)-11] of the World Health Organisation's International Classification of Diseases (a general framework for health information and reporting used by 194 countries) defines stroke as an acute neurologic dysfunction secondary to cerebral ischaemic stroke, intracerebral hemorrhage, subarachnoid hemorrhage, and stroke not known to be ischaemic or hemorrhagic.[5,6] The World health organisation (WHO) ICD-11 further defines cerebral ischaemic stroke as "acute focal neurological dysfunction caused by focal infarction at single or multiple sites of the brain," evidenced by symptom duration lasting >24 h or neuroimaging or other technique in the clinically relevant area of the brain.[6] Silent cerebral infarcts, cerebral microinfarcts, and monocular blindness with transient ischemia of the retina/retinal infarct were excluded from the definition of stroke in the WHO ICD-11.[6]

As of 2019, there were 12.2 million incident cases of stroke (150.8 per 100,000 people), 101 million prevalent cases of stroke (1240.3

How to cite this article: Ogholoh OD, Enyi AC, Idowu BM, Ogbeide AO, Ikubor JE, Nwafor NN, *et al.* Magnetic resonance imaging infarct volume correlates with carotid intima–media thickness and plaque echotexture in ischemic stroke. J West Afr Coll Surg 2023;14:17-25.

Received: 16-Jan-2023
Accepted: 07-Aug-2023
Published: 14-Dec-2023

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Access this article online

Website: www.jwacs-jcoac.com

DOI: 10.4103/jwas.jwas_9_23

Quick Response Code:

per 100,000 people), and 6.55 million deaths from stroke (84.2 per 100,000 people).[7] Ischemic stroke constituted 62.4% of all incident strokes in 2019, intracerebral hemorrhage constituted 27.9%, while subarachnoid hemorrhage constituted 9.7%. The five leading risk factors for stroke were high systolic blood pressure, high body mass index (BMI), high fasting plasma glucose, ambient particulate matter pollution, and smoking.[7] The prevalence of stroke in Africa is 1,460 per 100,000 people, with an annual incidence rate of up to 316 per 100,000 people.[8] Sub-Saharan Africa is now the region with the highest burden of stroke in the world, with its associated healthcare cost, morbidity, and mortality.[9-11]

Thrombotic or ischemic cerebral infarction results from the atherosclerotic obstruction of large cervical or cerebral arteries, with ischemia in all or part of the territory of the occluded artery. This can be due to occlusion at the site of the main atherosclerotic lesion or embolism from this site to more distal cerebral arteries.[6] The volume of the infarct could be a pointer to the predisposing risk factor(s) and eventual outcome and may be considered in determining possible intervention.[12]

Magnetic resonance imaging (MRI) protocols such as diffusion-weighted imaging (DWI) stroke volume and infarct core volume, which measure the infarct volume in acute ischemic stroke, show a high inter-reader reliability.[13] Manual tracing of the perimeter of the infarct is the most reproducible method for measuring the volume of the infarct and total intracranial space.[13] The choice of DWI sequence is to minimize errors that may occur from the measurement of perilesional edema, thereby improving the reproducibility of this method in stroke volume estimation.[13,14] Perilesional cerebral edema does not show hyperintensity (is not restricted) on DWI like the infarct and, therefore, does not pose a challenge during measurement.[13] MRI is also ideal because it detects ischemic infarcts early, eliminates exposure to ionizing radiation, and minimizes the possibility of underdiagnosing hyperacute cerebral infarcts. MRI DWI sequence has been shown to be the most sensitive for detecting of ischemic infarcts.[15]

HTN, which significantly increases the CIMT, is the predominant risk factor for stroke worldwide[16]; thus, there is a need to investigate the relationship between CIMT and infarct volume. This study estimated ischemic stroke infarct volume within the first 2 weeks of stroke onset using DWI MRI and correlated it with the CIMT and plaque echotexture.

Materials and Methods

Study design

This prospective, cross-sectional study was done at the Department of Radiology of *Delta State University Teaching Hospital, Oghara, Delta State, Nigeria* from May 2020 to November 2020. The Ethical Committee of the hospital approved the study protocol (*HREC*/*PAN*/2019/045/0335).

Study population

The subject's next-of-kin and or caregiver granted written informed consent after explaining the aims, objectives, and benefits of the study to them. Seventy consenting patients with hypertensive ischemic stroke (within the first 2 weeks poststroke) referred for brain MRI were enrolled consecutively after fulfilling the inclusion criteria.

Inclusion and exclusion criteria

The inclusion criterion was consenting adults (>18 years old) with DWI evidence of ischemic stroke within the first 2 weeks poststroke.[17-19] Patients with stroke onset greater than 2 weeks, dyslipidemia, ferromagnetic implants, or any contraindication to MRI were excluded.

Clinical parameters and medical history of the study population

The participants' age, sex, marital status, occupation, systemic HTN status, diabetes mellitus (DM) status, family history of stroke/DM/HTN, weight, height, and BMI were documented. The weight and height of participants who were unable to stand were determined using a chair weighing scale and demi-span estimated height,[20] respectively.

Brain magnetic resonance imaging

Brain imaging was performed on a 1.5 Tesla Toshiba Excelhart MRI scanner (Toshiba Medical Systems®, Otawara, Tochigi, Japan). Participants presenting for brain MRI were interviewed to obtain their past medical history and to exclude the presence of non-MRI-compliant metallic implants. Afterward, the patient was placed in a supine position in the MRI gantry, the head coils were applied over the head, scanning sequences/protocols were selected, and scanning commenced. Images of the brain were obtained using the T1W, T2W, fluid-attenuated inversion recovery, and DWI sequences in the axial, sagittal, and coronal planes.[21] Following the completion of the above sequences, the subject dismounted the gantry.

The volume of ischemic infarct was evaluated by manual tracing of the hyperintense lesion (area of restricted diffusion) on DWI images using the institutional picture archiving and communication system (PACS) software. The PACS software measurement toolbar was used to perform a manual tracing of the perimeter of an axial 2D image, and this gave an automatic display of the area equivalent of the measured perimeter after the tracing had been completed. For each patient, the perimeter of the infarct was traced in all the slices showing the infarct [Figure 1A] and the sum of the areas determined. The total volume of the infarct was determined by multiplying the sum of the areas by the slice distance (the sum of the thickness of slices showing the infarct). In addition, manual tracing of the perimeter of the brain [Figure 1B] in all the slices that show the brain was also done to automatically get the in the area; and the volume of the brain was determined by

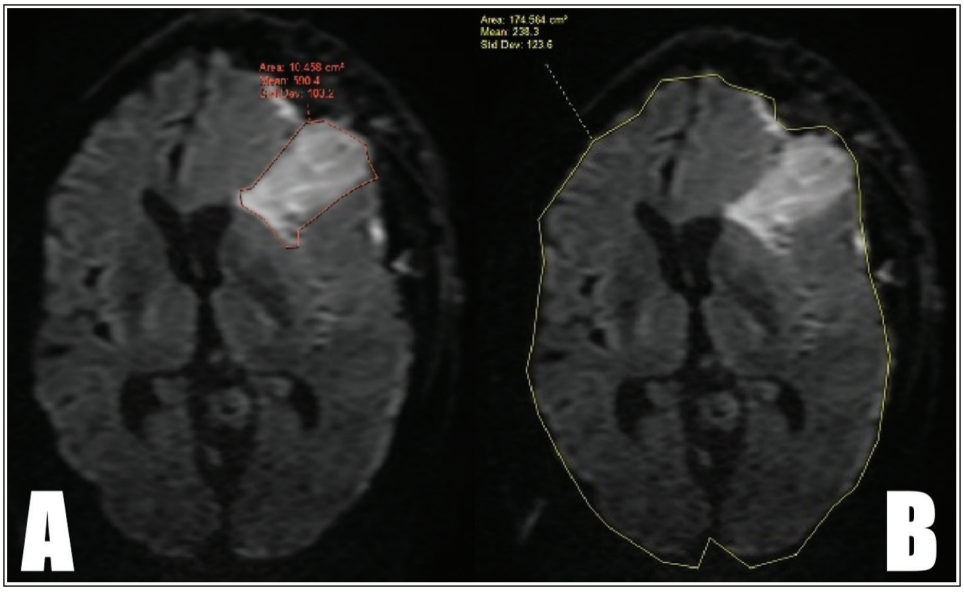

Figure 1: Axial DWI image of the brain shows an acute cerebral infarct (A). The perimeter of the infarct is traced to get the area of the infarct. This measurement is repeated for all slices with the infarct, and all are summed up to give the total area of the infarct. (B) Shows measurement of the area of the brain in one slice - the perimeter of the brain is traced as shown, repeated for all the slices that show the brain, and summed up to get the total area of the brain

multiplying the sum of all the areas by the slice distance as done for the infarct. This was done because the size of the brain varies between individuals; therefore, to get an infarct volume that is truly comparatively representative, the infarct volume was determined as a percentage of the volume of the brain. Thus, true infarct volume (%) was given as true infarct volume (%) = (volume of infarct/volume of the brain) × 100.[22] All the tracings were done by one radiologist to eliminate interobserver variation.[23]

Common carotid artery ultrasonography

Ultrasound of the carotid artery was performed on a Siemen G50 Sonoline® ultrasound machine (GM-56400A00E, model 7474722, Siemens Inc., Issaquah, Washington) with a linear high frequency 7.5 MHz transducer having spectral and color Doppler capabilities. Scanning was done with the patient in a supine position, the neck hyperextended, and the face turned to look over the shoulder of the contralateral side. The intima–media thickness was defined as the distance between the inner echogenic line representing the intima-blood interface and the outer echogenic line representing the adventitia–media junction.[24,25] Continuous longitudinal and transverse scans of the common carotid artery were done on the neck, and measurements of CIMT were taken at a point 1cm proximal to the carotid bifurcation on both sides. In order to increase the reproducibility of the measurements, each CIMT was measured three times in the longitudinal plane at the arterial far wall, and the three measurements were summed up and averaged to get the mean CIMT for each side. The measurement of the CIMT, done by one radiologist, was taken using electronic calipers after freezing the image. Magnification of the ultrasound image was used to improve the accuracy of measurements. The presence and sonographic appearance of atherosclerotic plaques was assessed and documented. A plaque was defined as a focal thickening of the intima–media layer >1.4 mm.[24,25]

Data analysis

The data were analyzed using IBM SPSS Statistics for Windows version 22 (IBM Corp., Armonk, New York). Data normality was tested using the Kolmogorov–Smirnov test. Categorical data were expressed in proportion or percentages, while continuous data were expressed as mean (± standard deviation). For categorical data, the test of association was done using the Chi-square test and Fisher's exact test, while for continuous variables, the Student's *t* test or analysis of variance was used to test for difference. The association/relationship between variables was tested using Pearson's correlation. The strength of the correlation coefficients was graded as follows: r = 0–0.2: very low/negligible and probably meaningless correlation; r = >0.2–0.4: low correlation; r = >0.4–0.6: moderate correlation; r = >0.6–0.8: high correlation; r = >0.8–1.0: excellent/very high correlation.[26] *P* values ≤0.05 were considered statistically significant at 95% confidence interval.

Results

Sociodemographics of the study population

Thirty-six (51.4%) participants were males, while the females were 34 (48.6%). The mean age of the participants was 59.9 ± 16.5 years (range = 18–90 years). The mean ages of the male and female participants were 58.8 ± 16.3 years (range = 26–90 years) and 61.1 ± 16.8 years (range = 18–90 years), respectively (P = 0.566). The other sociodemographic parameters are shown in Table 1.

Table 1: Sociodemographic characteristics of the study population

Variable	Frequency (%)			χ^2/t	df	*P*
	All (*n* = 70)	Male (*n* = 36)	Female (*n* = 34)			
Age range (years)						
0–25	2 (2.9)	0 (0.0)	2 (5.9)	15.134[a]	NA	0.011
26–35	6 (8.6)	6 (16.7)	0 (0.0)			
36–45	5 (7.1)	1 (2.8)	4 (11.8)			
46–55	9 (12.9)	5 (13.9)	4 (11.8)			
56–65	15 (21.4)	7 (19.4)	8 (23.5)			
66–75	23 (32.9)	15 (41.7)	8 (23.5)			
>75	10 (14.3)	2 (5.6)	8 (23.5)			
Mean age ± SD	59.9 ± 16.5	58.8 ± 16.3	61.1 ± 16.8	–0.577	68	0.566
Marital status						
Single	4 (5.7)	1 (2.8)	3 (8.8)	5.612[a]	NA	0.107
Married	58 (82.9)	32 (88.9)	26 (76.5)			
Widowed	2 (2.9)	2 (5.6)	0 (0.0)			
Separated	6 (8.6)	1 (2.8)	5 (14.7)			
Occupation						
Self-employed	12 (17.1)	8 (22.2)	4 (11.8)	6.273[a]	NA	0.189
Retired	38 (54.3)	18 (50.0)	20 (58.8)			
Student	2 (2.9)	0 (0.0)	2 (5.9)			
Unemployed	1 (1.4)	0 (0.0)	1 (2.9)			
Civil servant	17 (24.3)	10 (27.8)	7 (20.6)			

t: student *t*-test, df: degree of freedom, χ^2: chi square test, NA: degree of freedom not applicable in Fisher's exact test
[a]Fisher's exact test

Table 2: Medical history and clinical parameters of the study population

Frequency (%)						
Variable	**All (*n* = 70)**	**Male (*n* = 36)**	**Female (*n* = 34)**	χ^2/t	**df**	***P***
Hx of hypertension						
Yes	53 (75.7)	31 (86.1)	22 (64.7)	4.357	1	0.037
No	17 (24.3)	5 (13.9)	12 (35.3)			
Hx of DM						
Yes	18 (25.7)	8 (22.2)	10 (29.4)	0.473	1	0.492
No	52 (74.3)	28 (77.8)	24 (70.6)			
Hx of stroke						
Yes	0 (1.4)	0 (0.0)	0 (0.0)			
No	70 (100.0)	36 (100.0)	34 (100.0)			
Family hx of hypertension						
Yes	30 (42.9)	14 (38.9)	16 (47.1)	0.477	1	0.490
No	40 (57.1)	22 (61.1)	18 (52.9)			
Family hx of DM						
Yes	23 (32.9)	12 (33.3)	11 (32.4)	0.008	1	0.930
No	47 (67.1)	24 (66.7)	23 (67.6)			
Family hx of stroke						
Yes	21 (30.0)	10 (27.8)	11 (32.4)	0.174	1	0.676
No	49 (70.0)	26 (72.2)	23 (67.6)			
Height (cm)	158.2 ± 5.6	158.6 ± 4.6	157.8 ± 6.6	0.589	68	0.558
Weight (kg)	77.7 ± 7.5	77.5 ± 6.9	77.9 ± 8.2	–0.197	68	0.844
BMI (kg/m^2)	30.9 ± 2.7	30.7 ± 2.4	31.2 ± 3.1	–0.735	68	0.465

t: Student's *t* test, df: degree of freedom, χ^2: Chi-square test, BMI: body mass index, DM: diabetes mellitus, Hx: history
[a]Fisher's exact test

Clinical parameters and medical history of the study population

The mean height of all participants was 158.2 ± 5.6 cm. The male mean height was 158.6 ± 4.6 cm, while the female mean height was 157.8 ± 6.6 cm ($P = 0.558$). The mean weight of all participants was 77.7 ± 7.5 kg. The male mean weight was 77.5 ± 6.9 kg, while the female mean weight was 77.9 ± 8.2 kg ($P = 0.844$). The mean BMI was 30.9 ± 2.7 kg/m^2. Male mean BMI was 30.7 ± 2.4 kg/m^2, while female mean BMI was 31.2 ± 3.1 kg/m^2 ($P = 0.465$) [Table 2].

Fifty-three (75.7%) participants had pre-existing HTN, while subjects without a previous known history of HTN (diagnosed at presentation) were 17 (24.3%). Thirty-one (86.1%) males and 22 (64.7%) females had pre-existing HTN ($P = 0.037$). Subjects with a family history of HTN were 30 (42.9%). The males and females with a family history of HTN were 14 (38.9%) and 16 (47.1%), respectively ($P = 0.490$). Subjects with a family history of stroke were 21 (30.0%); the males and females with a family history of stroke were 10 (27.8%) and 11 (32.4%), respectively ($P = 0.676$) [Table 2].

Carotid intima-media thickness

The range of CIMT was 1.0–2.5 mm. The mean composite/aggregate bilateral CIMT of the study population was 1.16 ± 0.38 mm. The mean right CIMT of the study population was 1.18 ± 0.44 mm, while the mean left CIMT of the study population was 1.19 ± 0.43 mm ($P = 0.864$). The mean right CIMT for the male and female subjects were 1.17 ± 0.50 and 1.18 ± 0.44 mm, respectively ($P = 0.923$). The mean left CIMT for the males and females were 1.16 ± 0.37 and 1.22 ± 0.44 mm, respectively ($P = 0.617$). The male mean bilateral CIMT (1.15 ± 0.38 mm) and female mean bilateral CIMT (1.17 ± 0.39 mm) were not significantly different ($P = 0.787$) [Table 3].

Sonographic evaluation of carotid plaques

Plaques (CIMT > 1.4 mm) were detected in 60 (85.7%) subjects, comprising 31 (44.3%) men and 29 (41.4%) women. Twenty-two (36.7%), 20 (33.3%), and 18 (30.0%), subjects had hyperechoic, hypoechoic, and isoechoic plaques, respectively. Hyperechoic plaques were predominant in females, while hypoechoic plaques were predominant in males. Ten (32.3%) males had hyperechoic plaques, 13 (41.9%) had hypoechoic plaques, and eight (25.8%) had isoechoic plaques. Eleven (37.9%) females had hyperechoic plaques, eight (27.6%) had hypoechoic plaques, while 10 (34.5%) had isoechoic plaques. Hyperechoic plaques were associated with a higher mean infarct volume (17.3%), while hypoechoic and isoechoic plaques were associated with lower mean infarct volumes [Table 4].

Infarct volume in the study population

The mean infarct volume (%) for the entire study population was 8.07% ± 9.77% (range = 0.18%–26.35%). The mean infarct volume was highest among the 66–75 years age group (15.32% ± 2.25%), followed by the 56–65 years old age group (8.27% ± 1.17%). There was a statistically significant difference in mean infarct volume across the age groups [Table 5]. Furthermore, the highest mean infarct volumes among the male and female participants, 13.49% ± 2.82% and 18.75% ± 3.63%, respectively, were also recorded in the 66–75 years age group.

Infarct volume and carotid intima-media thickness

There was a moderate positive correlation between CIMT and infarct volume ($r = 0.70$; $P = 0.001$) in the entire study population. Similarly, positive correlations between

Table 3: Carotid intima–media thickness of the participants

Variable	Total ($n = 70$)	CIMT (mean ± SD)		*t*	df	*P* value
		Male ($n = 36$)	Female ($n = 34$)			
Right CIMT	1.18 ± 0.44	1.17 ± 0.50	1.18 ± 0.39	0.282	68	0.923
Left CIMT	1.19 ± 0.43	1.16 ± 0.37	1.22 ± 0.49	–0.104	68	0.617
t	–0.172	0.070	–0.316			
df	138	70	66			
P value	0.864	0.945	0.753			
Mean CIMT	1.16 ± 0.38	1.15 ± 0.38	1.17 ± 0.39	0.163	68	0.787

CIMT: carotid intima–media thickness in millimeters (mm), *t*: Student's *t* test, *P*: probability, df: degree of freedom, SD: standard deviation

Table 4: Relationship between infarct volume and plaque echotexture

Plaque echotexture	Mean infarct volume (range)	Test statistics[a]	df	*P* value
Hyperechoic	17.3 (1.85–26.32)	11.355	2	0.003
Hypoechoic	0.58 (0.31–2.68)			
Isoechoic	0.58 (0.11–5.86)			

df: degree of freedom
[a]Independent samples median test

Table 5: Infarct volume in different age groups

Age (years)	*n*	Mean infarct volume (%)	df	Test statistics	*P* value
18–25	2	0.18 ± 0.7	6	‡5.386	**0.001**
26–35	6	1.71 ± 0.61			
36–45	5	4.09 ± 3.34			
46–55	9	0.65 ± 0.30			
56–65	15	8.27 ± 1.17			
66–75	23	15.32 ± 2.25			
>75	10	5.18 ± 2.50			

‡indicates analysis of Variance

Table 6: Correlation between infarct volume and CIMT

			Cerebral infarct volume	Mean CIMT (mm)
Total	**Infarct volume**	*r*	1	0.70
		P		**0.001**
		N	70	70
	Mean CIMT (mm)	*r*	0.70	1
		P	**0.001**	
		N	70	70
Male	**Infarct volume**	*r*	1	0.73
		P		**0.001**
		N	36	36
	Mean CIMT (mm)	*r*	0.73	1
		P	**0.001**	
		N	36	36
Female	**Infarct volume**	*r*	1	0.67
		P		**0.001**
		N	34	34
	Mean CIMT (mm)	*r*	0.67	1
		P	**0.001**	
		N	34	34

r = Pearson's correlation coefficient
CIMT = carotid intima–media thickness

CIMT and infarct volume were recorded in both the male (r = 0.73; P = 0.001) and female (r = 0.67; P = 0.001) subjects [Table 6].

Furthermore, subjects who presented in the acute phase (1–3 days) of ictus showed a moderate positive correlation (r = 0.621; P = 0.0001) between CIMT and infarct volume, while there was a strong positive correlation (r = 0.74; P = 0.0001) in subjects that presented in the subacute phase (4–7 days) [Figure 2].

Discussion

Infarct volume has been shown to be useful in decision-making for thrombectomy cases,[22] improving patient selection for endovascular treatment,[13] and as a surrogate marker for stroke severity and functional outcome following ischemic stroke.[27,28] Furthermore, Daneshvari and Johansen[29] reported an association between infarct volume and the etiologies of acute ischemic stroke, such that single, large-volume strokes were associated with large artery atherosclerosis, while multiple infarcts were associated with cardioembolism.

In this study, there was a strong positive correlation between CIMT and cerebral infarct volume. This was in tandem with the findings of the MRI study by Alagoz *et al*.[30] in Turkey, but differs from that of Yıldırım *et al*.,[31] also in Turkey, who observed found no statistically significant correlation between infarct volume and CIMT (of both common carotid and internal carotid arteries). This disparity might be due to the use of computerized tomography, which has poor delineation of the infarct from the perilesional edema,[14] by Yıldırım *et al*. Also, the infarct volume was measured strictly within 72 h of the onset of stroke symptoms which could have caused an underestimation of infarct volume since it is now known that infarct volume at 7–30 days is more predictive of the eventual final infarct volume.[17-19] Our finding was that participants who presented in the acute phase (1–3 days) had a moderate positive correlation (r = 0.62) between CIMT and infarct volume, while there was a strong positive correlation (r = 0.74) in subjects that presented in the subacute phase

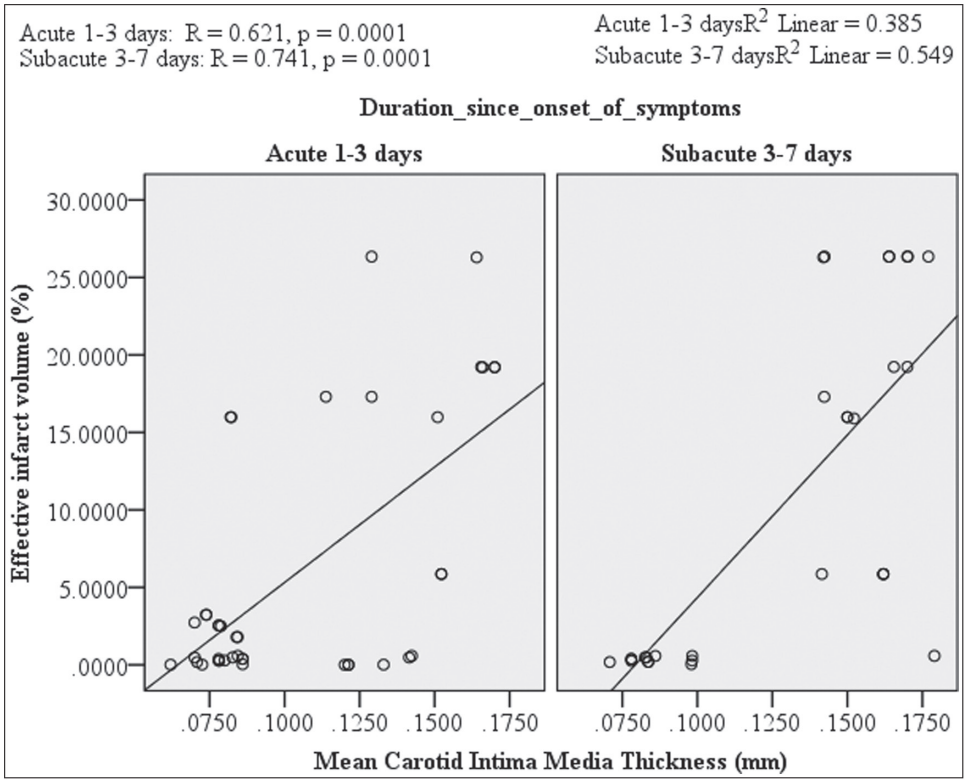

Figure 2: Scatter plot of infarct volume and carotid intima–media thickness according to duration since onset of symptoms

(4–7 days) further lends credence to the appropriateness of infarct measurement at 7–30 days.

The mean CIMT was higher in the left carotid artery than in the right in the entire study population and in the female participants. This may be due to the anatomy and/or hemodynamics of the left carotid artery being a direct branch of the aortic arch, resulting in a different level of stress conditions that predispose it to earlier and faster atherosclerotic changes. This was in agreement with the findings of Ogholoh *et al*.[32] who found CIMT to be consistently higher on the left compared to the right carotid artery. However, this finding is at variance with the findings of Asaleye *et al*.[33] who found CIMT to be the same value in both males and females and higher in the right carotid artery compared to the left. This difference may be due to the younger population studied by Asaleye *et al*.[33] who have reduced atherosclerotic changes when compared to the older age groups.

The plaque thickness (CIMT) determines the degree of carotid stenosis.[30] The same hemodynamic changes responsible for the thickening of the carotid arterial wall also cause plaque formation. In this study, subjects with hyperechoic plaques had a significantly larger mean infarct volume, but the plaque echotexture showed a weak positive correlation with infarct volume. This finding is at variance with the study by Mathiesen *et al*.[34] in Norway, which found that in comparison to echogenic plaques, echolucent plaques were more associated with a high risk of ischemic stroke. Leng *et al*.[24] in Hong Kong, China, also reported a lack of correlation between CIMT and large artery intracranial occlusive disease; however, the study correlated CIMT with findings on transcranial Doppler ultrasonography rather than with cross-sectional neuroimaging findings as done in the index study.

Plaque echotexture is the sonographically visible appearance and composition of carotid artery atherosclerotic plaques.[35-37] The plaque echotexture can offer vital information about its stability and vulnerability to rupture, which can increase the risk of ischemic stroke. Plaques with a heterogeneous echotexture (admixture of hypoechoic and hyperechoic areas) are typically regarded as more unstable and susceptible to rupture than plaques with a homogenous echotexture.[35] This is due to the fact that heterogeneous plaques frequently have a bigger lipid core, a thinner fibrous cap, and an increase in inflammatory cells, all of which contribute to an increased risk of rupture and subsequent thrombus development. Plaques with a more homogenous echotexture (uniform hyperechoic appearance) have a thicker fibrous cap, less lipid deposition, and fewer inflammatory cells, which may imply a decreased risk of rupture and subsequent thromboembolic events.[35]

Although plaque echotexture can be a valuable technique for determining the risk of ischemic stroke in individuals with carotid artery disease because it gives information

regarding plaque stability and fragility; yet, other variables such as plaque size, location, and the existence of concurrent medical problems play significant roles in predicting stroke risk and should be considered when evaluating individual individuals.[35]

In conclusion, common carotid artery IMT correlated positively with cerebral infarct volume in patients with ischemic stroke. Furthermore, hyperechoic plaques were associated with significantly larger infarct volumes compared to hypoechoic and isoechoic plaques.

Author contributions

OD Ogholoh: manuscript review and approval of final draft. AC Enyi: conception, design, literature search, data analysis, statistical analysis, manuscript preparation, manuscript editing, manuscript review, approval of final draft, and guarantor. BM Idowu: literature search, data analysis, manuscript preparation, manuscript editing, manuscript review, and approval of final draft. AO Ogbeide: manuscript review and approval of final draft. JE Ikubor: manuscript review and approval of final draft. NN Nwafor: manuscript review and approval of final draft. N Kogha: manuscript review and approval of final draft.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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