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RESEARCH ARTICLE
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FREQUENCY OF INCREASED CAROTID ARTERY INTIMAL MEDIAL THICKNESS AMONG PATIENTS WITH STROKE

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Abstract

Objective: To examine the Frequency of increased carotid artery intimal medial thickness among patients with stroke

Methods: This cross-sectional study was carried out at the department of neurology, Hayatabad medical complex Peshawar from January 2022 to January 2023. 200 participants were included in a cross-sectional study that was used to diagnose intimal medial thickness (IIMT). It was assessed how frequently such people experienced strokes. High-resolution ultrasound was used to assess the intima and media of the common carotid artery noninvasively in all patients. Statistical analysis was performed using SPSS 24.

Results: A cross-sectional research with 200 individuals was conducted to diagnose intimal medial thickness (IIMT).The incidence of strokes in such individuals was evaluated. All patients underwent noninvasive evaluation of the common carotid artery's intima and media thickness using high-resolution ultrasonography.

Conclusion: Utilizing noninvasive ultrasound technology, increased IMT thickness of the common carotid artery is linked to stroke.

Keywords: Carotid artery, intimal medial thickness (IMT), Stroke

Introduction

Stroke is a type of cerebrovascular disease that arises from chronic diseases that cause pathological changes in the cerebral vessels. The most common type of stroke, with an 85% prevalence rate, is an ischemic stroke, whose pathophysiology can be acute, such as occlusion by emboli, or chronic, resulting from atherosclerosis. The early stage of atherosclerosis is vessel injury induced by multiple conditions that injure the vessels directly or indirectly. The most common cause of vessel injury is hypertension, which is caused by high blood pressure, a major risk factor in stroke. The pathophysiology of an ischemic stroke can be acute, such as occlusion by emboli, or chronic, due to atherosclerosis.

Atherosclerosis brought on by hypertension causes blood arteries to shrink, stiffen, and lose their compliance. Blood flow through the arteries is accelerated by high blood pressure. This causes an

increase in shear stress, which in turn causes endothelial cells to produce more endothelium-derived relaxing factor (EDRF). This comprises prostacyclin, prostaglandin E, and nitric oxide. These vasomotor activators decrease the permeability of the arteries and increase the generation of superoxide. As a leukocyte is activated during an injury, the endothelial cells will produce more pro-inflammatory cytokines. This also causes vasoactive chemicals like nitric oxide and prostacycline to rise, which ultimately results in total endothelium damage.

Change in the common-carotid-artery intima—media thickness is thought to be an indicator of generalized atherosclerosis. It has also been adopted as an intermediate end point for determining cardiovascular morbidity and as a surrogate end point to evaluate the success of lipid lowering drug interventions [4,5]. The increase in intravascular pressure that results in hypertension causes stress on the vessel wall, which alters the vessel wall thickness through a process known as vascular remodeling. This reduction of the blood vessel's diameter is caused by hypertrophy (hypertrophic outer remodeling or hypertrophic inner remodeling) or through a eutrophic inner remodeling process. Measurements of the carotid artery media and intima thickness have been made using high-resolution carotid ultrasonography. Research conducted in western nations has demonstrated prospective associations as well as cross-sectional correlations between the incidence of cardiovascular and cerebrovascular illness and the thickness of the common carotid artery intima media [1-3]. There are currently few research in Asia, particularly in Pakistan, that demonstrate a link between stroke and increasing carotid artery intima—media thickness. The idea that there is a direct correlation between the incidence of stroke and the thickness of the carotid artery intima—media was examined in this study.

Methodology

Subjects and study design

This cross-sectional study was carried out at the department of neurology, Hayatabad medical complex Peshawar from January 2022 to January 2023. Patients with ages ranging from 30 to 80 who were receiving care at department of neurology, Hayatabad medical complex Peshawar served as the study participants. High-resolution B-mode ultrasound was used to assess each patient's carotid arteries using a cross-sectional approach. A single carotid artery longitudinal picture was obtained. Readers who were blind to all clinical information completed the measurements. The primary variable in establishing the relationship between intima—media thickness and stroke was the maximum rather than the mean intima—media thickness.

The mean of the maximal intima—media thickness of the near and far walls on both the left and right sides—defines the maximal intima—media thickness of the common carotid artery. If the intima-media thickness exceeded 1 mm, it was deemed abnormal.

Statistical analysis

The statistical analysis was carried out utilizing the SPSS 24. Chi-square statistics were utilized to assess the relationship between the variables. When necessary, two statistics and an independent t-test were employed to assess the significance of the differences between the background variables that were examined.

Results

The base-line characteristics of the 200 patients are given in Table 1. Other risk factors such as smoking and hypertension were analyzed to rule out the bias in determining the correlation between IMT and stroke. Using chi-square test for statistical analysis, we found there were no statistical difference between both patients according to hypertension and smoking. We can therefore conclude that the correlation of IMT and stroke were statistically significant (p= 0.005) (Table 2).

The gender distribution of the population studiesd was 122 (61%) males and 78 (39%) females. (Fig 1)

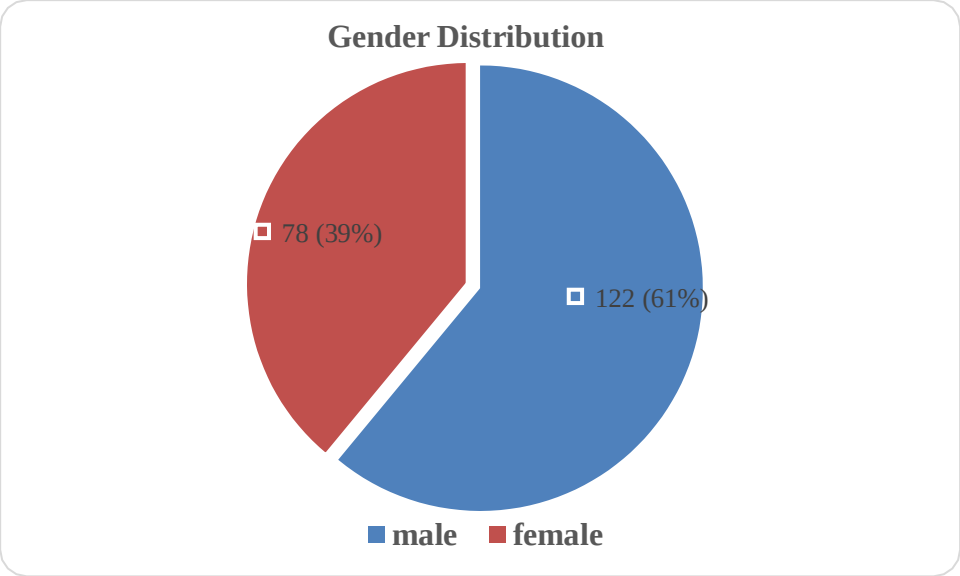

Figure 1gender distribution number and % of participants

Table 1: Mean age and risk factors of the participants

Mean age years (SD)	55.5 (9.8)
Hypertension n (%)	80 (40%)
Type-2 Diabetes n (%)	40 (20%)
Dyslipidemia n (%)	35 (17.5%)
Smoking n (%)	30 (15%)
IMT n (%)	200(100%)

Severity of IMT	**n (%)**	**Stroke n**	***p*-value**
Normal IMT (<8 mm)	100 (50%)	25	0.009
Mild IMT (8 mm to <12 mm)	45 (22.5%)	20	0.007
Moderate IMT (12 mm to <16 mm)	35 (17.5%)	22	0.008
Severe IMT (≥16 mm)	20 (10%)	18	< 0.001
Total	200 (100%)	85	0.005

Discussion

The positive correlation between carotid artery intima-media thickness and cardiovascular risk factors, as well as the positive correlation between carotid artery intima-media thickness and the incidence of myocardial infarction and hearty stroke in the Caucasian population, have been previously documented in numerous journals [6], [7]. According to this study, there is a significant correlation between stroke and intima-media thickness in the Pakistani population (p = 0.005). Because intima-media thickness is a measure of widespread atherosclerosis, there is a direct association between the two. This pathologic vascular phenomenon explains the correlation between IMT and stroke and plays a significant role in the pathophysiology of cerebro and cardiovascular events like stroke. [9, 10]

The potential relationship between the occurrence of cardiovascular events and carotid-artery intima-media thickness has been investigated in five further studies. Three of these investigations used common carotid artery measures to publish their findings. In a study of 1257 middle-aged population, a correlation between cardiac events and the thickness of the intima-media in the common carotid artery. This observation was based on 24 incidents during the course of a year-long follow-up. A single-center, prospective study of illness and disability in the elderly, the Rotterdam Elderly Study included 7983 participants who were 55 years of age or older. They conducted a case-control study

in a subset of their population and found a correlation between the risk of myocardial infarction and stroke and the thickness of the intima-media in the common carotid artery. [6-8]
The racial makeup of our population sample, our research's small subject group, and our methodology may account for some of the disparities between their study and ours. Furthermore, our data confirm their findings by demonstrating a significant association between the incidence of stroke and the thickness of the intima-media in the common carotid artery. This suggests that the thickness of the intima media in the common carotid artery may be utilized to predict cerebrovascular occurrences. There are a few more restrictions on this study. Among the study's shortcomings include its retrospective approach, small sample size, and few information on the patients' attributes. Nonetheless, our work might serve as a pilot study to ascertain the relationship between Asian individuals' intima-media thickness and stroke, particularly in the Pakistani community.

Conclusion

Utilizing noninvasive ultrasound technology, increased IMT thickness of the common carotid artery is significantly linked to stroke. The thickness of IMT is positively correlated with stroke.

References

1. Touboul PJ, Labreuche J, Vicaut E, Amarenco P. Carotid intima-media thickness, plaques, and Framingham risk score as independent determinants of stroke risk. Stroke. 2005 Aug 1;36(8):1741-5.
2. Kitamura A, Iso H, Imano H, Ohira T, Okada T, Sato S, Kiyama M, Tanigawa T, Yamagishi K, Shimamoto T. Carotid intima-media thickness and plaque characteristics as a risk factor for stroke in Japanese elderly men. Stroke. 2004 Dec 1;35(12):2788-94.
3. Hollander M, Hak AE, Koudstaal PJ, Bots ML, Grobbee DE, Hofman A, Witteman JC, Breteler MM. Comparison between measures of atherosclerosis and risk of stroke: the Rotterdam Study. Stroke. 2003 Oct 1;34(10):2367-72.
4. Hunt KJ, Evans GW, Folsom AR, Sharrett AR, Chambless LE, Tegeler CH, Heiss G. Acoustic shadowing on B-mode ultrasound of the carotid artery predicts ischemic stroke: the Atherosclerosis Risk in Communities (ARIC) study. Stroke. 2001 May;32(5):1120-6.
5. Chambless LE, Folsom AR, Clegg LX, Sharrett AR, Shahar E, Nieto FJ, Rosamond WD, Evans G. Carotid wall thickness is predictive of incident clinical stroke: the Atherosclerosis Risk in Communities (ARIC) study. American journal of epidemiology. 2000 Mar 1;151(5):478-87.
6. O'leary DH, Polak JF, Kronmal RA, Manolio TA, Burke GL, Wolfson Jr SK. Carotid-artery intima and media thickness as a risk factor for myocardial infarction and stroke in older adults. New England Journal of Medicine. 1999 Jan 7;340(1):14-22.
7. Petty GW, Brown Jr RD, Whisnant JP, Sicks JD, O'Fallon WM, Wiebers DO. Ischemic stroke subtypes: a population-based study of incidence and risk factors. Stroke. 1999 Dec;30(12):2513-6.
8. Bots ML, Hoes AW, Koudstaal PJ, Hofman A, Grobbee DE. Common carotid intima-media thickness and risk of stroke and myocardial infarction: the Rotterdam Study. Circulation. 1997 Sep 2;96(5):1432-7.
9. Bots ML, Hofman A, Grobbee DE. Increased common carotid intima-media thickness: adaptive response or a reflection of atherosclerosis? Findings from the Rotterdam Study. Stroke. 1997 Dec;28(12):2442-7.
10. Handa N, Matsumoto M, Maeda H, Hougaku H, Kamada T. Ischemic stroke events and carotid atherosclerosis: results of the Osaka Follow-up Study for Ultrasonographic Assessment of Carotid Atherosclerosis (the OSACA Study). Stroke. 1995 Oct;26(10):1781-6.