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Original Article

Influence of Lifestyle and Clinical Risk Factors on Carotid Atherosclerotic Changes in Adults Undergoing Doppler Sonography

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ABSTRACT

Background: Hypertension is a major risk factor for atherosclerotic cardiovascular disease, and carotid intima-media thickness is a useful surrogate marker for early atherosclerotic changes. This study aimed to assess the relationship between lifestyle factors and clinical risk factors with carotid arterial parameters in hypertensive patients compared to normotensive controls. **Methods & Materials:** This cross-sectional comparative study was conducted at Mymensingh Medical College Hospital from July 2019 to June 2021. In this study, 84 participants (42 hypertensive patients and 42 normotensive controls) aged 20-70 years were recruited for comprehensive carotid Doppler sonography on a Medison SonoAce X8 ultrasound machine. IMT measurements and resistive index were obtained along with lifestyle factors that included tobacco use, dietary habits, physical activity, and family history of the participants. The data were analyzed using SPSS, version 26. **Results:** The hypertensive patients had a higher mean IMT (0.88 ± 0.20 mm vs 0.66 ± 0.10 mm, p=0.001) and RI (0.70 ± 0.05 vs 0.62 ± 0.05, p=0.001) compared with normotensive controls. Carotid plaque was found in 5 hypertensive subjects (11.9%) and none in the control group (p=0.005). The factors that showed statistically significant associations with plaque formation were tobacco use (OR=9.17, 95%CI 1.01-83.1, p=0.012), unhealthy diet (OR=7.70, 95%CI 0.88-66.4, p=0.018), and family history of hypertension (OR=5.88, 95%CI 0.65-52.5, p=0.041). **Conclusion:** Hypertension significantly contributes to early atherosclerotic changes in carotid arteries, as manifested by increased IMT and RI. Modifiable lifestyle factors like tobacco use and dietary habits substantially increase the risk of carotid plaque formation, thus emphasizing comprehensive cardiovascular risk management in hypertensive patients.

Keywords: *Hypertension, Carotid Intima-Media Thickness, Doppler Sonography, Atherosclerosis*

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INTRODUCTION

Cardiovascular diseases are the leading causes of global morbidity and mortality. Hypertension is considered one of the major modifiable risk factors [1]. Because of an increasing global trend, hypertension affects over a billion people worldwide and contributes significantly to the burden of atherosclerotic cardiovascular disease, stroke, and chronic kidney disease [2]. Detection of subclinical atherosclerosis in hypertensive conditions is of great importance for early intervention and prevention of cardiovascular events. The measurement of carotid intima-media thickness, or CIMT, has emerged as a well-established, non-invasive surrogate marker for the early stages of subclinical atherosclerosis [3]. B-mode ultrasonographic measurement of CIMT yields important information regarding arterial wall changes that precede the clinical manifestations of cardiovascular disease [4]. Many studies have indicated that increased CIMT is strongly associated with the presence of coronary atherosclerosis and predicts future cardiovascular events, including myocardial infarction and stroke [5]. The technique offers excellent reproducibility and can identify individuals at risk of future clinical endpoints earlier than traditional risk factor assessment alone. The pathophysiological mechanisms through which hypertension accelerates the atherosclerotic process include endothelial dysfunction, increased oxidative stress, chronic inflammation, and arterial wall remodeling. The association between blood pressure levels and CIMT has been well established, with systolic blood pressure being strongly associated with carotid arterial thickening. Recent evidence indicates that hypertensive patients have higher values of CIMT and RI than normotensive controls, indicating greater vascular resistance and lower arterial compliance. The resistive index, computed from velocity measurements by Doppler ultrasound, gives complementary hemodynamic information on the level of downstream resistance and stiffness affecting arterial circulation. Beyond hypertension itself, various lifestyle and behavioral factors contribute substantially to atherosclerotic risk. Cigarette smoking is considered one of the most powerful modifiable risk factors for cardiovascular disease because it promotes endothelial dysfunction, inflammation, oxidative stress, and plaque formation through multifactorial mechanisms [9]. It increases the pace of atherosclerosis by damaging the arterial endothelium, enhancing platelet aggregability, and promoting the development of carotid atherosclerotic lesions [10]. Dietary patterns also play an important role: diets high in saturated fats, trans fats, and sodium are associated with an increased atherosclerotic burden and heightened blood pressure [11]. Another major modifiable risk factor for hypertension and cardiovascular disease is physical inactivity. Regular physical activity improves endothelial function, reduces arterial stiffness, lowers blood pressure, and decreases overall cardiovascular risk [12]. In contrast, sedentary behavior is linked to unfavorable metabolic profiles, increasing body weight, and higher rates of hypertension and atherosclerotic disease. A family history of hypertension is a significant non-modifiable risk factor, reflecting both genetic predisposition and shared environmental exposures [13]. Recent genome-wide association studies have highlighted several genetic variants predisposing to hypertension, and individuals having first-degree relatives with hypertension exhibit significantly increased lifetime risk of developing the condition itself [14]. Despite the large volume of data on various individual risk factors, comprehensive studies on the combined impact of lifestyle factors, family history, and clinical parameters on carotid atherosclerotic changes among hypertensive populations remain limited, especially within the South Asian population. Understanding the relative contributions of these factors will

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help develop targeted preventive strategies, with the overall aim of risk stratification. Therefore, this study was designed to assess the relationship between lifestyle factors and clinical risk factors with carotid arterial parameters in hypertensive patients compared to normotensive controls.

METHODS & MATERIALS

This cross-sectional comparative study was conducted at Mymensingh Medical College Hospital, Bangladesh, over a period of two years from July 2019 to June 2021, to evaluate carotid atherosclerotic changes among hypertensive adults compared with normotensive controls using duplex ultrasonography. A purposive sampling technique was used to recruit 84 adults aged 20 to 70 years, comprising 42 hypertensive patients and 42 normotensive individuals. Hypertension was defined as clinically measured blood pressure ≥140/90 mmHg. Participants were eligible if they consented to the examination, while those with diabetes mellitus, malignant hypertension, previous cerebrovascular accidents, or inability to complete the protocol were excluded to reduce confounding influences. Data were obtained by using structured interviews evaluating demographic, clinical, and lifestyle characteristics, followed by standardized ultrasonographic evaluation. All examinations were performed with a Medison SonoAce X8 ultrasound machine equipped with a 5-12 MHz linear array probe. Subjects were examined supine, with slight neck extension to optimize scan conditions. The common carotid artery was scanned in transverse and longitudinal planes, and intima-media thickness was measured 0.5-1.0 cm proximal to the carotid bulb along the far wall. Colour Doppler imaging, with a 3.5-5 MHz frequency, maintaining the Doppler angle below 60 degrees, was used to measure peak systolic velocity, end-diastolic velocity, and the resistive index calculated from spectral waveforms; flow patterns and morphological features were documented. Measurements were carried out in a blind manner by two trained examiners, and mean values were calculated for analysis. Data analysis was done using SPSS version 26. All variables were summarized using descriptive statistics. Bivariate comparisons were made using independent t-tests for continuous variables and chi-square tests for categorical variables. A $p<0.05$ was considered significant for the analysis.

RESULTS

Table I represents the baseline demographics of the study population. The ages for the hypertensive participants ranged between 20-70 years, with the mean age being 50.29 ± 13.57 years. Normotensive controls had an age range between 20 and 65 years, with a mean age of 47.60 ± 12.99 years. The highest percentage of the hypertensive patients, 30.9%, was over 60, while a majority of normotensive individuals belonged to the 51-60-year age group, numbering 31%. The gender distribution indicated male predominance in the hypertensive group, 52.4%, as compared to 42.9% in the normotensive group, whereas females numbered 47.6% and 57.1%, respectively.

Table I
Baseline Demographic Characteristics of Study Participants (*n* = 84).

Category	Hypertensive (n = 42)	Normotensive (n = 42)
Age (in years)	n (%)	n (%)
≤30	4 (9.6%)	6 (14.3%)
31-40	7 (16.8%)	7 (16.8%)
41-50	9 (21.6%)	8 (19%)
51-60	9 (21.6%)	13 (31%)
>60	13 (30.9%)	8 (19.1%)
Mean ± SD	50.29 ± 13.57	47.60 ± 12.99
Range (min-max)	20-70	20-65
Sex	n (%)	n (%)
Male	22 (52.4%)	18 (42.9%)
Female	20 (47.6%)	24 (57.1%)

Table II shows the clinical profile of the study population. In hypertensive patients, the mean systolic blood pressure was 136.76 mmHg, while in controls it was 117.38 mmHg. For diastolic, the mean was 88.10 mmHg and 69.76 mmHg, respectively. Regarding the duration of hypertension among hypertensive participants, 31.0% reported 2 years, followed by 28.6% who described 3 years of disease duration. Among all the cases, the mean duration of hypertension was 2.83 ± 1.2 years, indicating recent diagnosis in most of them.

Table II
Clinical Profile of Study Participants.

Variable	Hypertensive (n = 42)	Normotensive (n = 42)
Blood Pressure (Mean mmHg)	-	-
Systolic	136.76	117.38
Diastolic	88.10	69.76
Duration of Hypertension (years)	n (%)	
1	5 (11.9%)	
2	13 (31.0%)	
3	12 (28.6%)	
4	7 (16.6%)	
5	5 (11.9%)	
Mean ± SD	2.83 ± 1.2	

Table III depicts the carotid artery ultrasonographic parameters. The mean intima-media thickness was markedly higher among hypertensive patients, 0.88 ± 0.20 mm, compared to that of normotensive controls, 0.66 ± 0.10 mm, and this difference was highly statistically significant, p=0.001. The range of IMT in hypertensive patients was 0.5-1.6 mm versus 0.4-0.9 mm in controls. The resistive index was also significantly higher in hypertensive individuals, 0.70 ± 0.05, than in normotensive subjects, 0.62 ± 0.05, once again reaching high statistical significance, p=0.001, with ranges of 0.56-0.81 and 0.54-0.76, respectively.

Table III
Carotid Arteries Ultrasonographic Parameters.

Parameter	Hypertensive (n = 42) Mean ± SD	Normotensive (n = 42) Mean ± SD	p-value
Intima-Media Thickness (IMT, mm)	0.88 ± 0.20	0.66 ± 0.10	0.001
Range	0.5-1.6	0.4-0.9	-
Resistive Index (RI)	0.70 ± 0.05	0.62 ± 0.05	0.001
Range	0.56-0.81	0.54-0.76	-

Table IV demonstrates the lifestyle and family history factors of the study population. 76.2% of the hypertensive patients had a family history of hypertension, while in controls, only 9.5% had that record. A large proportion of hypertensive patients (71.4%) followed unhealthy dietary habits, while in the normotensive patients, only 2.4% did so. Tobacco consumption was noticed among 61.9% of the hypertensive subjects and only 4.8% of the controls. In addition, adequate physical activity was lower among the hypertensive subjects (34.1%) as compared to the normotensive controls (50%), indicating a sedentary pattern in the hypertensive population.

Table IV
Lifestyle and Family History Factors of the Study Population.

Risk Factor	Hypertensive (n = 42)	Normotensive (n = 42)
Family history of hypertension	32 (76.2%)	4 (9.5%)
Unhealthy diet	30 (71.4%)	1 (2.4%)
Tobacco use	26 (61.9%)	2 (4.8%)
Adequate physical activity	14 (34.1%)	21 (50.0%)

Table V summarizes the association of lifestyle and clinical risk factors with carotid plaque formation. Plaques were detected in 5 individuals belonging to the hypertensive group-whereas 79 had no plaque formation. Hypertension itself revealed a significant association with plaque presence, p=0.005. Tobacco use demonstrated a strong association, p=0.012: 4 out of 28 tobacco users developed plaques versus 1 out of 56 non-users. An unhealthy diet was significantly associated with plaque formation, p=0.018: plaque formation occurred in 4 of 31 individuals with poor dietary habits. Family history of hypertension showed a significant correlation, p=0.041: among 36 individuals with a positive family history, 4 plaques were recorded. Physical activity did not reach statistical significance, p=0.298.

Table V
Association of Lifestyle and Clinical Risk Factors with Carotid Plaque Formation.

Risk Factor	Total (N)	Plaque Present (n=5)	Plaque Absent (n=79)	p-value
Hypertension	Hypertensive (n=42)	5	37	0.005
	Normotensive (n=42)	0	42	
Tobacco use	Yes (n=28)	4	24	0.012
	No (n=56)	1	55	
Unhealthy diet	Yes (n=31)	4	27	0.018
	No (n=53)	1	52	
Family history of HTN	Present (n=36)	4	32	0.041
	Absent (n=48)	1	47	
Physical activity	Adequate (n=35)	1	34	0.298
	Inadequate (n=49)	4	45	

Table VI denotes the odds ratio for risk factors associated with carotid plaque formation. The highest risk was conferred by tobacco use (OR=9.17, 95%CI 1.01-83.1, p=0.012), indicating more than nine-fold increased odds of plaque formation among tobacco users. An unhealthy diet showed substantial risk elevation (OR=7.70, 95%CI 0.88-66.4, p=0.018). Family history of hypertension was associated with nearly six-fold increased odds (OR=5.88, 95%CI 0.65-52.5, p=0.041). Inadequate physical activity demonstrated elevated risk, though the estimates have wide CIs and did not achieve statistical significance (OR=3.02, 95%CI 0.33-26.9, p=0.298), perhaps because of limited sample size.

Table VI
Odds Ratios for Risk Factors Associated with Carotid Plaque Formation.

Risk Factor	Odds Ratio (OR)	95% CI	p-value
Tobacco use	9.17	1.01 - 83.1	0.012
Unhealthy diet	7.70	0.88 - 66.4	0.018
Family history of HTN	5.88	0.65 - 52.5	0.041
Inadequate physical activity	3.02	0.33 - 26.9	0.298

DISCUSSION

This study elucidates significant relations of hypertension and lifestyle factors with early carotid atherosclerotic changes as evidenced by ultrasonographic evaluation. Hypertensive patients had significantly higher carotid intima-media thickness and resistive index compared to normotensive controls, indicating early vascular remodeling. The mean value of IMT observed in this study is 0.88 mm among hypertensives versus 0.66 mm in controls. This clinically significant increase matches the previously demonstrated evidence that for every 1 mmHg increase in systolic blood pressure, there is approximately a 0.002 mm increase in CIMT [15]. Kaur et al. found that IMT values are constantly higher in hypertensive populations than in normotensive ones [16]. Additionally, the RI was higher in hypertensive patients (0.70 vs. 0.62), which implies an increased downstream vascular resistance and decreased arterial compliance, characteristics of hypertensive arteriopathy [17]. Calabia et al. also noted that the association of carotid RI with systemic haemodynamics and arterial stiffness, and target organ damage, making it a useful and practical Doppler-based risk assessment tool [18]. A strong association was found between tobacco use and carotid plaque development, as smoking conferred more than nine-fold increased odds for plaque development to occur (OR = 9.17). This is in close agreement with Benowitz et al., who reported that the promotion of atherosclerosis through mechanisms involving endothelial dysfunction, oxidative stress, chronic inflammation, and hypercoagulability [19]. Shovon et al. suggested that cigarette smoke reduces nitric oxide bioavailability, increases reactive oxygen species, and activates inflammatory cascades, including Toll-like receptor signaling and inflammasome pathways, all contributing to accelerating plaque development [20]. Of note, evidence exists indicating that smoking cessation can slow the progression of carotid atherosclerosis. These findings emphasize the reversible component of smoking-related vascular injury and underscore the need for smoking cessation programs in hypertensive patients [21]. Dietary patterns represented another strong predictor of early atherosclerotic change in this investigation, where poor diet was associated with seven-fold higher odds ratios for plaque development. This finding agrees with well-established links among poor dietary quality, dyslipidemia, systemic inflammation, and hypertension [22]. Norouzzadeh et al. indicated that poor dietary patterns/smoking combinations may produce synergistic negative effects, thereby further elevating cardiovascular and stroke risk [23]. Taken together, these results point to the importance of standardized dietary counseling based on salt restriction, increased fruit and vegetable consumption, and intake of saturated and trans fats as key elements of cardiovascular risk factor modification. Family history of hypertension was also significantly associated with carotid plaque formation, with an OR of 5.88. Contemporary genome-wide association studies have identified multiple loci involved in blood pressure regulation, confirming the genetic predisposition to hypertension susceptibility [24]. First-degree relatives of subjects with early-onset hypertension carry a significantly higher lifetime risk, estimated to be from two to four times higher than the general population [25]. Genetic factors contribute to about 30-50% of the variation in blood pressure, influencing vascular resistance, fluid balance, and renal sodium handling through intricate polygenic mechanisms [26]. Though genetic predisposition is a non-modifiable risk factor, evidence shows that tailored lifestyle modifications can reduce inherited risk, and thus early identification and preventive counselling are indicated among those with a positive family history. While in this study, physical inactivity did not reach a level of statistical significance, the elevated odds ratios were directionally congruent with the extensive epidemiological evidence. Regular physical activity increases endothelial function, reduces arterial stiffness, lowers blood pressure, and decreases cardiovascular risk [27]. Sedentary behavior is repeatedly associated with hypertension and atherosclerotic cardiovascular disease, placing inactivity as a major modifiable determinant of vascular health [28]. The presence of carotid plaque exclusively in hypertensive subjects (11.9% versus 0%) further underlines the accelerated atherosclerotic course seen with elevated blood pressure. Community-based prospective studies also demonstrate that CIMT predicts stroke risk in hypertensive cohorts, especially in conjunction with blood pressure for enhanced risk stratification [29]. In addition to measurement of IMT, carotid ultrasound provides an assessment of plaque morphology, burden, and distribution, all with prognostic implications. These findings, in summary, support critical clinical implications: the identification of modifiable lifestyle factors contributing to early atherosclerosis supports comprehensive risk-reduction programs in hypertensive patients; smoking cessation merits priority for its strong interaction, while dietary modifications are also imperative. Regarding family history, individuals should be proactively observed and involved in preventive measures. Carotid Doppler studies may be included in routine risk assessment to enable early detection and timely intensive preventive measures.

LIMITATIONS

This was a cross-sectional study, and the establishment of temporal relationships and causality between risk factors and atherosclerotic changes is not possible. The relatively small sample size and single-center recruitment may limit the statistics for detecting modest effect sizes and restrict the generalizability of findings to broader populations.

CONCLUSION

This study revealed that hypertension is significantly associated with increased carotid intima-media thickness and resistive index, reflecting early atherosclerotic vascular changes. Modifiable lifestyle factors such as smoking and unhealthy dietary habits substantially increase the risk of carotid plaque formation among hypertensive subjects. Family history of hypertension represents an important non-modifiable risk factor identifying individuals in need of intensive preventive interventions. Comprehensive cardiovascular risk management incorporating aggressive lifestyle modification should be prioritized in hypertensive populations to prevent further progression of atherosclerotic disease.

RECOMMENDATIONS

Larger multi-center prospective longitudinal studies are required to establish the temporal progression of carotid atherosclerotic changes and to determine the real effects of lifestyle interventions on the progression of IMT and plaque development over time. Further studies should address the additive value of carotid Doppler parameters to current

cardiovascular risk prediction models and the cost-effectiveness of routine carotid screening in hypertensive populations.

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CONFLICT OF INTEREST
None declared

ETHICAL APPROVAL
The study was approved by the Institutional Ethics Committee

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