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ORIGINAL

Doppler Ultrasound Assessment of Smoking-Induced Hemodynamic Alterations in the Common Carotid Artery

Evaluación por Ultrasonido Doppler de las Alteraciones Hemodinámicas Inducidas por el Tabaquismo en la Arteria Carótida Común

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ABSTRACT

Introduction: vascular conditions, particularly those involving the common carotid artery, are increasingly associated with tobacco use. Smoking raises fibrinogen levels and platelet production, increasing blood viscosity and the risk of clotting. Nicotine also promotes atherosclerosis and arterial narrowing, disrupting blood flow and raising the risk of stroke or heart attack.
Objective: this study investigates the effects of smoking on carotid artery blood flow.
Method: a quantitative descriptive analysis was conducted on 39 individuals (smokers and nonsmokers), averaging 30 years of age. Carotid arteries were examined using B-mode and Doppler sonography with a 7-MHz linear transducer, following standard techniques. Participants also completed a detailed medical history survey. Key measurements included arterial narrowing, wall thickness, and blood flow velocity.
Results: significant differences were observed between smokers and nonsmokers, with smokers showing higher rates of vascular changes. Notably, the right and left common and internal carotid arteries (RCCA, RICA, LCCA, LICA) exhibited signs of altered blood flow. Atherosclerosis indicators were found in 28 % of smokers.
Conclusion: the study demonstrates a strong link between tobacco use and plaque buildup in the carotid artery, along with noticeable changes in blood flow. These findings highlight the urgent need to raise awareness about the cardiovascular dangers of smoking.

Keywords: Smoking; Carotid Arteries; Atherosclerosis; Hemodynamics; Ultrasonography; Doppler.

RESUMEN

Introducción: las enfermedades vasculares, especialmente aquellas que afectan a la arteria carótida común, están cada vez más asociadas con el consumo de tabaco. Fumar eleva los niveles de fibrinógeno y la

producción de plaquetas, lo que incrementa la viscosidad de la sangre y el riesgo de coagulación. La nicotina también favorece la aterosclerosis y el estrechamiento arterial, lo que interfiere con el flujo sanguíneo y aumenta el riesgo de accidente cerebrovascular o infarto.
Objetivo: este estudio investiga los efectos del tabaquismo sobre el flujo sanguíneo en la arteria carótida.
Método: se realizó un análisis descriptivo cuantitativo en 39 individuos (fumadores y no fumadores), con una edad promedio de 30 años. Las arterias carótidas fueron examinadas mediante ecografía en modo B y Doppler, utilizando un transductor lineal de 7 MHz, siguiendo técnicas estándar. Los participantes también completaron una encuesta detallada sobre antecedentes médicos. Las mediciones clave incluyeron el estrechamiento arterial, el grosor de la pared y la velocidad del flujo sanguíneo.
Resultados: se observaron diferencias significativas entre fumadores y no fumadores, siendo los fumadores quienes presentaron mayores alteraciones vasculares. En particular, las arterias carótidas comunes e internas derecha e izquierda (RCCA, RICA, LCCA, LICA) mostraron signos de alteración del flujo sanguíneo. Se encontraron indicadores de aterosclerosis en el 28 % de los fumadores.
Conclusión: El estudio demuestra una fuerte relación entre el consumo de tabaco y la formación de placas en la arteria carótida, así como cambios notorios en el flujo sanguíneo. Estos hallazgos subrayan la urgente necesidad de concienciar sobre los peligros cardiovasculares del tabaquismo.

Palabras clave: Abaquismo; Arterias Carótidas; Aterosclerosis; Hemodinámica; Ultrasonografía; Doppler.

INTRODUCTION

In modern society, health conditions or illnesses associated with vascular structures have gained prevalence, particularly within the context of the common carotid artery (CCA).[1,2] Among the factors exerting the most pronounced influence on these arteries are tobacco products, precipitating heightened fibrinogen levels, and escalated platelet production.[3] This reaches its highest point in augmented blood viscosity and heightened clotting susceptibility. Furthermore, the danger of nicotine buildup along the inner arterial walls catalyzes the emergence of atherosclerosis, emphasized by stenosis within the arteries [4]. This phenomenon adversely affects blood flow dynamics, rendering it more susceptible to clot formation. Concerns regarding the potential occurrence of stroke or heart attack in the event of sudden vessel obstruction due to these accumulative processes.[5,6]

Medical imaging modalities serve as conventional tools for assessing vascular health.[7,6,7,8,9,10,11,12,13,14,15] Computed Tomography Angiography (CTA) scans deliver high-fidelity examinations;[16,17,18,19,20,21,22] however, they are limited by radiation exposure and restricted device mobility, particularly concerning minute intravascular issues.[23,24,25] Usually, Magnetic Resonance Angiography (MRA) scans might fail to offer definitive arterial images, particularly with respect to calcium deposits and smaller vessels. Additionally, challenges exist in generating separated arterial images.[26,27,28,29] It is relevant to acknowledge that patients seeking contrast enhancement may face obstacles, including allergic reactions, during both previously mentioned procedures[30,31,32,33,34]

Ultrasound (US) emerges as a modality of choice in the spectrum of vascular risk, applicable across both developed and underdeveloped contexts, due to its remarkable precision in illustrating arterial structures and their hemodynamic profiles.[35,36,37,38,39,40] This significance is further highlighted by modern methodologies, facilitating the use of 3D and 4D attributes in the imaging process.[41,42] Consequently, the US device assumes a pivotal role in pre-clinical evaluations of vascular pathologies, delivering images that closely mirror physiological reality within the arteries. The inherent safety of US devices is underscored by their applicability across diverse patient populations, including pregnant individuals or those with metallic implants.[11,43,44] The versatility of transducer adjustments to accommodate various examination requisites, coupled with the absence of contrast media necessity, further amplifies the utility of this approach.[45,46]

The correlation between tobacco consumption and cardiovascular disease is extensively researched and acknowledged within scientific and medical circles. This link, established over decades of meticulous epidemiological and clinical study, should not be perceived as a fresh discovery. Cigarette smoke comprises a complex amalgamation of hazardous substances that lead to endothelial dysfunction, oxidative stress, inflammation, and thrombogenesis—all fundamental mechanisms involved in the pathogenesis of atherosclerosis and other cardiovascular diseases. The extensive and consistent data establishes smoking as a significant modifiable risk factor for cardiovascular morbidity and mortality, which remains critically important for public health.[47,48]

This study employs Doppler ultrasound technology for the verification of carotid artery integrity and the examination of hemodynamic attributes regulating blood circulation within. The early identification of stenosis and occlusions, before the onset of cardiac and pulmonary disorders, not only accelerates patient recovery but also augments their prognostic outlook.[49] Moreover, the primary objectives of this study include measuring

abnormal hemodynamics and variations in their constants among individuals of typical health, comparing these metrics across smokers and those suffering from specific syndromes, and comprehensively analyzing the proportion between the internal carotid artery and the common carotid artery while placing side smokers against non-smokers.

METHOD

The study's methodological framework and meticulous procedures were undertaken to carry out the research objectives (quantitative research methodology). It covers a comprehensive depiction of the study methodology, the composition of the study sample, the implementation of precise study instruments, the validation of said instruments, the evaluation of their reliability, and the subsequent statistical analysis. The authors indicate that the study received approval from an institutional ethics committee or review board. This indicates that the study adhered to established ethical norms. Personal identifiers were eliminated.

Sampling

The smokers and non-smoker participants were collected randomly from the surgery department at Al-Hussein Governmental Hospital, Palestine. 39 patients were collected from ages 18 to 65 years to be included as main study samples, which contained 24 males and 15 females. 21 patients (53,8 %) were nonsmokers, while 18 patients (28,2 % and 18 %) were collected among heavy smokers and moderate smokers, respectively. The researchers obtained written consent from each participant to ensure that the patients voluntarily agreed to take part in the research, understanding the purpose and procedures involved. Ethical considerations were followed, and the patient's rights and privacy were respected throughout the study.

Study Design

The evaluation of the impact of intense smoking on the hemodynamics of the CCA and its ramifications hinged on cross-sectional assessments. These assessments required the measurement of peak systolic velocity (PSV), intima-media (IM) thickness, and stenosis levels in a randomized selection of patients, covering both smokers and non-smokers. The aim was to compare the resultant effects, thereby changing the influence of smoking on the hemodynamic behavior of carotid arteries.

The survey consisting of nine sections was administered to the designated 39 participants. Each section played a distinct role in quantifying the extent of smoking's impact on the circulatory health of carotid arteries, as determined through Doppler ultrasound. These segments included various aspects: the first, requesting personal data such as gender, age, and socioeconomic status; the second, delving into the participants' medical history, including chronic illnesses and prior surgeries; the third, collecting information about smoking habits, including duration, quantity, and smoking type; and finally, the fourth, providing complex insights into the characteristics of the carotid arteries themselves.

Data Analysis

SPSS 10.0.1174 was employed for data analysis. Extracted from the questionnaire sections were statistical measures such as means, standard deviations, and response percentages of the sample. To assess the significance of variances attributed to the study's independent variables, a combination of T-test analysis and single variance analysis (one-way analysis of variance) was conducted. Additionally, calculations were carried out for Cronbach's alpha coefficient and Pearson correlation coefficient. A significance level of 5 % was adopted for all analyses.

RESULTS

The first two sections of the questionnaire were designed to assess blood flow health and hemodynamics in the main carotid artery using Doppler ultrasound. This assessment was conducted based on two variables: patients' smoking status (smoker or non-smoker) and gender (male or female). Among the outcomes, 43,6 % of smokers exhibited related symptoms, whereas 56,4 % of non-smokers did. In terms of gender, male patients experienced symptoms at a rate of 63,6 %, while females exhibited symptoms at 36,4 % (table 1).

Examining the effect of heavy smoking on artery stenosis in RCCA, four categories were explored: no stenosis, stenosis 50 %, stenosis 50 %-70 %, and over 70 % stenosis. Results indicated that 25,6 % of smokers exhibited no stenosis, 17,9 % had 50 % stenosis, 2,6 % had 50 %-70 % stenosis, and none exhibited over 70 % stenosis. For non-smokers, the corresponding values were 53,8 %, 0 %, 0 %, 0 %, and 0 %. This points to a relatively minimal prevalence of stenosis among non-smokers, which may be attributed to genetic and physiological factors.

Similarly, when evaluating the effect of excessive smoking on RICA, the study considered four categories: no stenosis, stenosis ≤ 50 %, stenosis 50 %-70 %, and over 70 % stenosis. Outcomes revealed that 15,4 % of smokers exhibited no stenosis, 23,1 % had stenosis ≤ 50 %, 5,1 % had stenosis 50 %-70 %, and 2,6 % exhibited over 70 % stenosis. Among non-smokers, 51,3 %, 2,6 %, 0 %, and 0 % fell into these categories, respectively. Additionally,

for the LCCA, 25,6 %, 15,4 %, 5,1 %, and 0 % of smokers exhibited these respective stenosis levels, while for non-smokers, the values were 51,3 %, 2,6 %, 0 %, and 0 % (table 2).

Table 1. The numbers and percentages of symptoms experienced by patients on the health and movement of blood flow in the main carotid artery using Doppler ultrasound by patient gender (male /female) and different patient conditions (smoker/non-smoker).

Paragraph	Smoker		Non-smoker		Female		Male	
	Number	Percentage	Number	Percentage	Number	Percentage	Number	Percentage
Asymptomatic	10	18,2 %	12	21,8 %	10	18,2 %	12	21,8 %
Hypertension	6	10,9 %	8	14,5 %	4	7,3 %	10	18,2 %
High cholesterol	3	5,5 %	3	5,5 %	2	3,6 %	4	7,3 %
Diabetes	4	7,3 %	5	9,1 %	2	3,6 %	7	12,7 %
Cardiovascular symptoms other	0	0,0 %	2	3,6 %	1	1,8 %	1	1,8 %
Retinal infraction	1	1,8 %	1	1,8 %	1	1,8 %	1	1,8 %
Total marks	24	43,6 %	31	56,4 %	20	36,4 %	35	63,6 %

Table 2. Numbers and percentages of the effect of excessive smoking on artery stenosis of RCCA, RICA, LCCA, and LICA according to the patient's condition (smoker or non-smoker).

Artery	Paragraph	Smoker		Non-smoker	
	Stenosis	Number	Percentage	Number	Percentage
RCCA	No Stenosis	10	25,6 %	21	53,8 %
	Stenosis ≤ 50 %	7	17,9 %	0	0,0 %
	Stenosis 50 %-70 %	1	2,6 %	0	0,0 %
	Over 70 % Stenosis	0	0,0 %	0	0,0 %
RICA	No Stenosis	6	15,4 %	20	51,3 %
	Stenosis ≤ 50 %	9	23,1 %	1	2,6 %
	Stenosis 50 %-70 %	2	5,1 %	0	0,0 %
	Over 70 % Stenosis	1	2,6 %	0	0,0 %
LCCA	No Stenosis	10	25,6 %	20	51,3 %
	Stenosis ≤ 50 %	6	15,4 %	1	2,6 %
	Stenosis 50 %-70 %	2	5,1 %	0	0,0 %
	Over 70 % Stenosis	0	0,0 %	0	0,0 %
LICA	No Stenosis	10	25,6 %	16	41,0 %
	Stenosis ≤ 50 %	4	10,3 %	4	10,3 %
	Stenosis 50 %-70 %	2	5,1 %	1	2,6 %
	Over 70 % Stenosis	2	5,1 %	0	0,0 %

The questionnaire's validity was confirmed by comparing carotid artery thickness between smokers and healthy individuals. The survey addressed the impact of smoking on artery thickness for RCCA, RICA, LCCA, and LICA, with answers categorized into three sections: 0,4, 0,4-0,8, and over 0,8. Among non-smokers, 57 % had normal RCCA thickness, while among smokers, this figure stood at 50 %. For RICA, 71 % of non-smokers exhibited normal thickness, compared to 28 % of smokers. Similarly, 62 % of non-smokers had normal LCCA thickness, compared with 25,6 % of smokers. Finally, 67 % of non-smokers had normal LICA thickness, while 26 % of smoker participants fell into this category (figure 1).

The buildup of tobacco-related residues within arteries can impede blood movement and velocity. This study, focusing on major arteries, underscored the pronounced impact of tobacco products. Notably, excessive smoking led to significant alterations in blood flow quantity, affecting RCCA, RICA, LCCA, and LICA by 46,2 %, 33,3 %, 46,2 %, and 33,3 %, respectively. In contrast, non-smokers generally exhibited normal flow characteristics in these arteries, except for LICA, where 2,6 % experienced flow disturbances. These findings confirm that tobacco products exert a substantial influence on health, especially in arteries subject to these deposits, which can block blood volume and speed. Further elaboration is available in table 3.

In assessing the relationship between RCCA and RICA to differentiate between smokers and non-smokers, results were remarkable for non-smokers, with 58 % exhibiting arterial blood velocity deemed excellent. This relationship yielded a satisfaction rate of 42 % among smoker participants, indicating that 72 % of them were unaffected by atherosclerosis (table 4).

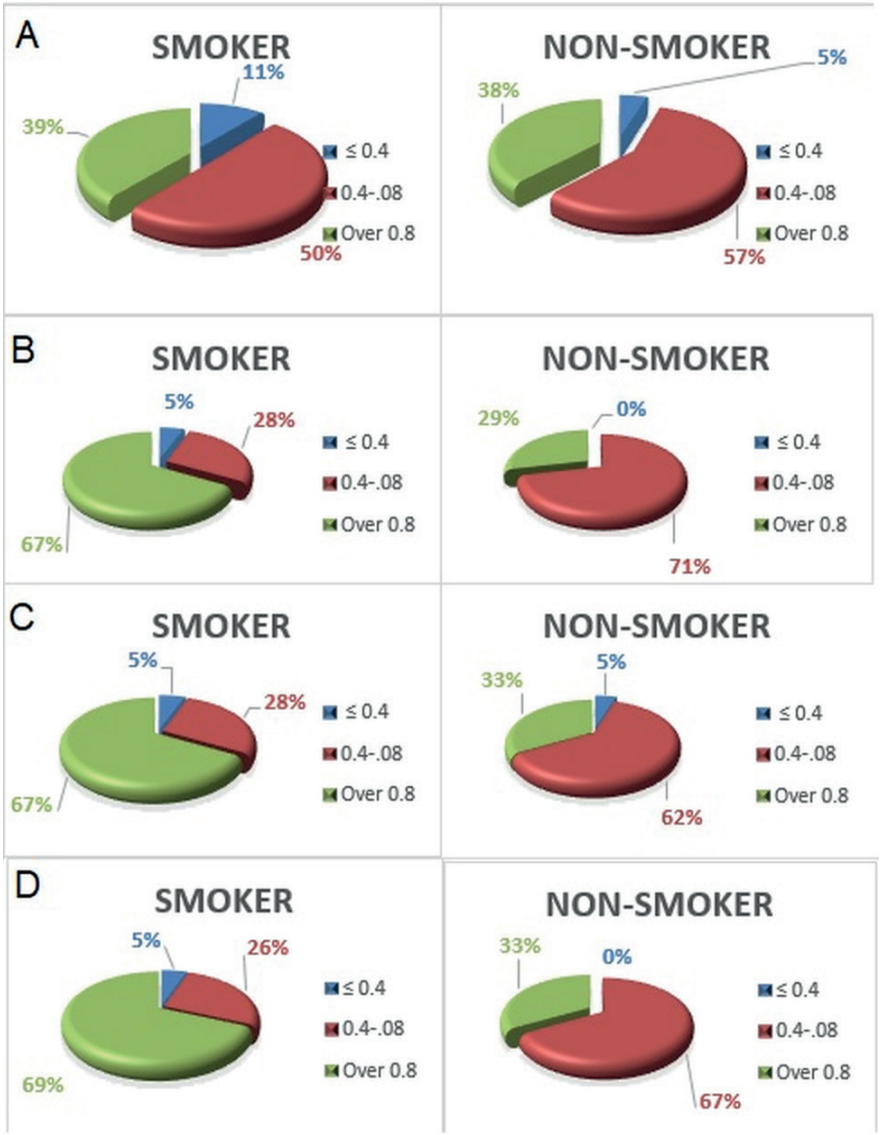

Figure 1. Illustrates the percentages depicting the impact of excessive smoking on artery thickness based on the patient's status (smokers/non-smokers). The graphical representations denoted as A, B, C, and D correspond to RCCA, RICA, LCCA, and LICA, respectively.

Similarly, the connection between LCCA and LICA revealed that 57 % of non-smokers maintained healthy arteries, while the corresponding value for smokers was 42 %. This coherence with the data from table 4 underscores that 72 % of smoker participants did not manifest symptoms of atherosclerosis (table 5).

Table 3. Numbers and percentages of the effect of excessive smoking on the blood speed in the artery (RCCA, RICA, LCCA, and LICA) according to the patient's condition (smoker/non-smoker).

Artery	Paragraph	Smoker		Non-smoker	
	Blood Speed	Number	percentage	Number	percentage
RCCA	≤ 125	18	46,2 %	21	53,8 %
	Over 125	0	0,0 %	0	0,0 %
RICA	≤ 125	13	33,3 %	21	53,8 %
	Over 125	5	12,8 %	0	0,0 %
LCCA	≤ 125	18	46,2 %	21	53,8 %
	Over 125	0	0,0 %	0	0,0 %
LICA	≤ 125	13	33,3 %	20	51,3 %
	Over 125	5	12,8 %	1	2,6 %

Table 4. Numbers and percentages of the effect of excessive smoking on arterial blood velocity of RICA compare to the artery blood velocity of RCCA, depending on the patient's condition (smoker/non-smoker).

Paragraph	Smoker				Non-smoker			
Blood velocity	Number RICA	Number RCCA	Result RICA/ RCCA	percentage	Number RICA	Number RCCA	Result RICA/ RCCA	Percentage
≤ 125	13	18	0,72	42 %	21	21	1	58 %
Over 125	5	0	0	0 %	0	0	0	0 %

Table 5. Numbers and percentages of the effect of excessive smoking on arterial blood velocity of LICA relative to arterial blood velocity of LCCA, depending on the patient's condition (smoker/non-smoker).

Paragraph	Smoker				Non-smoker			
Blood velocity	Number LICA	Number LCCA	Result LICA / LCCA	percentage	Number LICA	Number LCCA	Result LICA / LCCA	percentage
≤ 125	13	18	0,72	43 %	20	21	0,95	57 %
Over 125	5	0	0	0 %	1	0	0	0 %

DISCUSSION

Smoking, whether active or passive, remains an unusual habit within an extraordinary society. Despite its toxicity and detrimental nature, it continues to be accepted by millions globally, establishing tobacco as a pivotal element in avoidable global mortality. Rapidly, the year 2015 witnessed a staggering 7,2 million documented deaths worldwide as a consequence of this practice.[50,51,52,53]

The narrowing within the internal carotid artery (ICA) and common carotid artery (CCA) is clinically identified as carotid artery stenosis (CAS). CAS gives rise to ipsilateral retinal or cerebral ischemia, with an asymptomatic CAS prevalent in 5 %-10 % of the population. This condition is closely linked to various vascular risk factors, including smoking, age, and cholesterol levels. However, patients with asymptomatic CAS exhibit a heightened risk of subsequent stroke due to atherosclerotic disease, as demonstrated by a comprehensive study involving 1820 patients,[54,55] Furthermore, these individuals face an elevated risk of myocardial infarction (MI) and vascular death due to underlying illnesses, particularly among CAS patients.[56,57,58] Several studies underscore the vulnerability of patients with coronary heart disease (CHD) or abdominal aortic aneurysm (AAA) to injuries, MI, or vascular mortality, particularly when CAS is present.[59,60]

The outcomes of this study confirm a direct correlation between tobacco products and carotid artery plaque formation among smokers. Alongside alterations in carotid hemodynamics, 43,6 % of smokers exhibited vessel disturbances encompassing hypertension, high cholesterol, diabetes, and retinal infarction. Notably, asymptomatic CAS accounted for a higher percentage, reaching 18,2 %. These findings align with prior research, such as Mahmoud S. Babiker's study, which reported stenosis figures and percentages of 20,5 for RCCA, 30,8 for RICA, 20,5 for LCCA, and 20,5 for LICA among smoker participants. This further underscores the noticeable differences between smokers and non-smokers, prominently influenced by tobacco usage.[61]

The evaluation of blood volume traversing the CCA in both smokers and non-smokers elucidates the impact of tobacco on CCA.[62,63,64] The study affirms tobacco's contribution to CAS, culminating in atherosclerosis-induced alterations in hemodynamic blood flow. Thus, smoking emerges as a risk factor for CCA thickness. This research delved into assessing RCCA, RICA, LCCA, and LICA. The percentage of stenosis in smoker participants exceeded that of non-smokers (figure 1). These findings strengthen previous claims that arteries and vessels bear the impact of tobacco products, amplifying the vulnerability to atherosclerosis.[65,66]

Tobacco's potential to worsen atherosclerosis and consequently affect the arterial blood flow system was a focal point. This study evaluated the speed of blood flow among smokers and non-smokers, revealing that 12,8 % of smokers faced disruptions in blood flow velocity, centered around RICA and LICA. In contrast, non-smokers maintained regular blood flow velocities without interruption. The substantial contrast between smokers and non-smokers, evident through numeric and percentage analysis, underscores the profound influence of excessive smoking. However, a significant 28 % of smokers engaged with CAS. Consequently, there is a strong need to investigate the negative consequences of tobacco products, therefore promoting greater awareness among smokers and facilitating the act of stopping.[67]

The Design Ensures Valid, Reproducible, and Unbiased Results are discussed below:

Validity

The research demonstrates a definitive correlation between tobacco consumption and carotid artery stenosis (CAS), substantiated by consistent statistical evidence and comparative analysis of smokers and non-smokers. The strategy facilitates a focused evaluation of tobacco's impact on arterial thickness and blood flow by examining specific anatomical sites (RCCA, RICA, LCCA, LICA), hence enhancing internal validity. It

employs prior studies for benchmark comparisons (e.g., Babiker's research), so augmenting the credibility of its conclusions and bolstering external validity.

Reproducibility

The approach encompasses specific measurement criteria, including the percentage of stenosis and blood flow velocity, enabling other researchers to duplicate the study using comparable equipment and demographics. The utilization of quantitative data (e.g., percentage values, and blood flow velocities) guarantees that the results are amenable to statistical analysis and reproducibility in further research. References to recognized diagnostic markers such as retinal infarction and vascular risk indicators further reinforce uniformity and reliability.

CONCLUSION

In recent years, the incidence of vascular illnesses has significantly increased, particularly with conditions related to the common carotid artery (CCA). This study posits a theory indicating a direct correlation between cigarette smoking and morphological changes in the carotid artery. The vascular alterations are mostly ascribed to compromised endothelial function, a crucial initial occurrence in atherogenesis. Smoking exacerbates endothelial dysfunction via oxidative stress and inflammation, facilitating the formation of atherosclerotic plaques. The existence of these plaques leads to substantial alterations in carotid hemodynamics, encompassing modified blood flow velocity and heightened arterial stiffness. Hemodynamic changes can be quantified by Doppler ultrasonography and are acknowledged as preliminary markers of cardiovascular risk. Moreover, multiple studies have established a significant positive link between chronic tobacco consumption and the advancement of carotid artery disease, highlighting smoking as a principal modifiable risk factor. This evidence underscores the pressing necessity to improve public knowledge, especially among smokers, regarding the harmful vascular effects of smoking. Advocating for smoking cessation is essential for both preventing carotid artery pathology and alleviating the overall burden of cardiovascular disease.

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CONFLICT OF INTEREST

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