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Original Research

Effects of Smoking on Common Carotid Intima-Media Thickness

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ABSTRACT:

Background: To assess the effects of smoking on common carotid intima-media thickness. **Materials & methods:**In total, 50 smokers who were currently smoking and 50 non-smokers were included in the study. Individuals who had smoked during the last month or had consumed about 100 cigarettes during their lifetime were classified as current smokers. Any thickness of CIMT greater than 0.8 mm was considered abnormal. Every outcome was entered into a Microsoft Excel spreadsheet, and then SPSS software was used for statistical analysis. The student t test is employed to assess the degree of significance. **Results:**The subjects in the CIMT group ranged in age from 43.5 to 48.5 years on average. Males made up the majority of the subjects in both research groups. The current smokers' and non-smokers' mean CIMT values were 0.645 and 0.540, respectively. **Conclusion:**Smoking causes morphological changes in CCA, as evidenced by changed CIMT.

Keywords: Smoking, Carotid Intima-Media.

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INTRODUCTION

Cigarette smoking is a causative factor for premature atherosclerosis.[1,2] However, the exact mechanism of smoking-induced damage to the arterial wall and its relation to the atherosclerotic process is still largely obscure. The adverse effects of smoking on vascular wall structure has been historically evaluated by autopsy studies.[3] Clinically applicable diagnostic measurements such as ultrasound imaging are widely utilized in recent years. [4]

Cardiovascular disease (CVD) is one of the leading causes of morbidity and mortality on a global scale. [5] Atherosclerosis, the major underlying pathological process of CVD, is a chronic inflammatory state in which there is localized accumulation of lipids and inflammatory cells that make up atherosclerotic plaques. This process begins in early life and has a long latent and asymptomatic period before developing into a clinical condition, such as stroke, myocardial infarction (MI), and other CVD. [6]A well-used method of assessing atherosclerosis at the subclinical stage is the measurement of the thickness of the intimal and medial layers of the carotid artery wall, termed carotid intima-media thickness (cIMT). [7] The value of cIMT in research has been widely used as a surrogate measure of the burden of carotid atherosclerosis and a predictor of CVD for primary prevention. [8,9] Meanwhile, the observed deceleration of cIMT progression resulting from therapeutic intervention implies a positive correlation with the reduction of CVD risk. [10,11]However, this has posed a major issue to the standardization of cIMT measurement protocol. [12] A careful analysis of cIMT research reveals considerable inconsistencies in cIMT measurement, including the carotid segments evaluated (common carotid artery [CCA-IMT], internal carotid artery [ICA-IMT], carotid bifurcation [bif-IMT], or the combined segments [combined-IMT]), the measurement of the far or near walls of the segments, the type of measurements made (mean or maximum of single measurements, mean of the mean, or mean of the maximum for multiple measurements), and whether or not plaques were included in the cIMT measurement. [13]

Carotid-wall intima–media thickness is a surrogate measure of atherosclerosis associated with cardiovascular risk factors and with cardiovascular outcomes.[14,15] The intima–media thickness is the distance from the lumen–intima interface to the media–adventitia interface of the artery wall, as measured on noninvasively acquired ultrasonographic images of the carotid arteries. Increased intima–media

thickness of the common carotid artery represents a form of atherosclerosis that is manifested as diffuse arterial-wall thickening, whereas increased intima–media thickness of the proximal internal carotid artery is a surrogate for focal atherosclerotic plaque.[16] The mean intima–media thickness of the common carotid artery is a more reproducible measure than the intima–media thickness of the internal carotid artery and is believed to be better suited for cardiovascular risk assessment and intervention studies. [17] Hence, this study was conducted to assess the effects of smoking on common carotid intima-media thickness.

MATERIALS & METHODS

In total, 50 smokers who were currently smoking and 50 non-smokers were included in the study. Individuals who had smoked during the last month or had consumed about 100 cigarettes during their lifetime were classified as current smokers. Any thickness of CIMT greater than 0.8 mm was considered abnormal. The plaque was recognized as a localized protrusion of the vessel wall that expanded by ≥1.5 mm into the lumen. We obtained bilateral CIMT values. First, transverse images of bilateral CCAs were examined, starting at the origin and moving toward the vessel bifurcation. Color Doppler was employed for both CCAs, with a sample volume approximately half that of the vascular diameter and a Doppler angle between 45^0 and 60^0.When the color barely touched the vessel's interior surface, that was the perfect setting for the color increase. The spectral Doppler tracings were performed using the same artery segment as was used for the CIMT measurements. Every outcome was entered into a Microsoft Excel spreadsheet, and then SPSS software was used for statistical analysis. The student t test is employed to assess the degree of significance.

RESULTS

The subjects in the CIMT group ranged in age from 43.5 to 48.5 years on average. Males made up the majority of the subjects in both research groups. The current smokers' and non-smokers' mean CIMT values were 0.645 and 0.540, respectively. It was discovered that smokers' mean CIMT levels were noticeably higher than non-smokers'. Comparing revealed that the outcomes were significant.

Table 1: Comparison of CIMT (in mm)

CIMT (mm)	Current smokers	Non-smokers	p-value
Right	0.64	0.50	0.00*
Left	0.64	0.49	0.00*
Mean	0.645	0.540	0.00*

*: Significant

DISCUSSION

Active smoking is a major risk factor in the development of atherosclerosis and cardiovascular events.[18] Studies have found an excess risk up to ten years after smoking cessation, indicating the involvement of chronic processes besides acute effects in the mechanisms connecting smoking to cardiovascular disease. [19] The mediating mechanisms include vascular dysfunction, impaired endothelial and platelet function, and altered lipid profiles.[20] Furthermore, inflammatory mechanisms have been shown to be important in the development and progression of the atherosclerotic disease process, even though there are also negative studies.[21] Since smoking has been shown to be associated with a variety of markers of low grade inflammation, low grade inflammation is one possible link between smoking and progression of atherosclerosis.[22] Hence, this study was conducted to assess the effects of smoking on common carotid intima-media thickness.

In the present study, 50 smokers who were currently smoking and 50 non-smokers were included in the study. Individuals who had smoked during the last month or had consumed about 100 cigarettes during their lifetime were classified as current smokers. Any thickness of CIMT greater than 0.8 mm was considered abnormal. The plaque was recognized as a localized protrusion of the vessel wall that expanded by ≥1.5 mm into the lumen. We obtained bilateral CIMT values. First, transverse images of bilateral CCAs were examined, starting at the origin and moving toward the vessel bifurcation. A study by Fan AZ et al, the smoking effect on different echogenic components of carotid arterial wall measured by ultrasound is not well elucidated.Middle-aged men and women who had IMT measurement > or = 0.7 mm at baseline and follow-up were included (N = 413, age 40-60 years at baseline in 1995). Intima-media thickness of common carotid artery (CCA-IMT) and its components (echogenic and echolucent layers) were measured at baseline and in the follow-up examination 3 years later. IMT and its components were compared across current, former and never smokers. Individual growth models were used to examine how smoking status was related to the baseline and progression of overall IMT and IMT components.For both men and women, current smoking was associated with thicker echogenic layer than never smokers; former smokers exhibited thinner echogenic layer than current smokers after adjustment for cigarette pack-years. Among women, current smoking was also associated with a thinned echolucent layer that resulted in a non-significant overall association of current smoking with IMT for women.Cigarette smoking is associated with carotid artery morphological changes and the association is

sex-dependent. The atherogenic effect of smoking appears to be partly reversible among former smokers. IMT measurement alone may not be adequate to detect carotid atherosclerosis associated with cigarette smoking among middle-age women.[23]

In the present study, Color Doppler was employed for both CCAs, with a sample volume approximately half that of the vascular diameter and a Doppler angle between 45^0 and 60^0. When the color barely touched the vessel's interior surface, that was the perfect setting for the color increase. The spectral Doppler tracings were performed using the same artery segment as was used for the CIMT measurements. Every outcome was entered into a Microsoft Excel spreadsheet, and then SPSS software was used for statistical analysis. The student t test is employed to assess the degree of significance. Another study by Hansen K et al, investigated the long-term associations between smoking habits, environmental tobacco smoke exposure (ETS), carotid intima-media thickness (IMT) progression rate, and rate of lumen diameter reduction in the carotid artery during a 16-year follow-up. Another objective was to investigate if an effect of smoking on progression rate could be explained by increased low grade inflammation.The study population included 2992 middle-aged men and women in the 1991-1994 (baseline) and the 2007-2012 (re-examination) investigation of the Malmö Diet and Cancer Study cardiovascular cohort. Associations between smoking, progression of carotid IMT and lumen diameter reduction due to plaque protrusion were assessed by linear regression.IMT progression rates and rate of lumen diameter reduction increased from never smokers with no ETS through former, moderate and heavy smokers, even after adjustment for traditional risk factors (e.g., differences in yearly progression rates (mm/year) of maximal IMT in the carotid bifurcation compared to never smokers; former smokers 0.0074 (95% CI: 0.0018-0.0129), moderate smokers 0.0106 (95% CI: 0.0038-0.0175), and heavy smokers 0.0146 (95% CI: 0.0061-0.0230)). Former smokers showed distinct lowering of progression rates after more than five years since smoking cessation. Smoking and former smoking was associated with increased low grade inflammation, however, the effect of smoking on atherosclerotic progression rate remained fairly unchanged after such adjustment.The effect of smoking and former smoking on carotid IMT progression rates and change in lumen reduction due to plaque protrusion could not be explained by differences in traditional risk factors or low grade inflammation.[24]Shu D et al, thicker carotid intima-media thickness (CIMT) has been a valid predictor for atherosclerosis development. A significant association between environmental tobacco smoke (ETS) and thickening of CIMT has been demonstrated in adults, whereas such association has scarcely been reviewed in paediatric population. Newcastle-Ottawa scale was used to assess the quality of the included studies. Qualitative synthesis analyses were performed on the selected studies. 331 articles were retrieved, and 4 were finally selected. All four studies investigated the association between postnatal ETS and CIMT in children, and three of them reported a statistically significant positive association. Three studies investigated the association between prenatal maternal ETS and CIMT, and one of the three found a positive association. Two studies explored the association between postnatal maternal ETS and CIMT, one reported a positive association. Two studies used serum cotinine measurement to quantify ETS and demonstrated potential dose-response relationship with CIMT. ETS exposure may play an independent role in the development of cardiovascular risks in healthy children and adolescents. In the consideration of the great burden of respiratory and cardiovascular diseases, there is an urgent need of effective surveillance for paediatric population's ETS exposure to reduce smoke exposure.[25]Although cIMT has been used extensively in cardiovascular research over the years, there is a lack of standard protocol for how this technology should be used as a research tool. Some study groups, however, have attempted to create cIMT consensus protocols. The Mannheim consensus, established in 2004 and amended in 2006 and 2011, proposed that cIMT be measured preferably on the far wall of the CCA at least 5 mm below the bulb within an area free of plaque with a clearly identifiable double-line pattern. [26] The American Society of Echocardiography consensus statement published in 2008 recommended that cIMT measurements should be restricted to the far wall of the CCA and supplemented by a thorough scan of the extracranial carotid arteries for the presence of carotid plaque to increase sensitivity for identifying subclinical vascular disease.[27] In addition, The ARIC protocol recommends using mean IMT on the far wall of the 3 segments (CCA, ICA, and bif) in both the right and left arteries. In our meta-analysis, 1 study adopted the Mannheim consensus, 3 studies adopted ARIC protocol, but no other established consensus was followed by the remaining studies. This is certainly a major source of heterogeneity. In fact, the 2013 Cardiovascular Risk Clinical Practice Guidelines highlighted that having a standardized measurement was a major challenge and that routine measurement of cIMT was not advised in clinical practice for risk assessment for a first atherosclerotic CVD event. [28]

CONCLUSION

Smoking causes morphological changes in CCA, as evidenced by changed CIMT.

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