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ORIGINAL ARTICLE

Acute effects of cigarette smoking in habitual smokers, a focus on endothelial function

Nasser M. Taha, Mohammed A. Abdel Wahab, Amr S. Amin *

Cardiology Department, Al Minia University, Egypt

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KEYWORDS
Cigarette smoking;
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Abstract *Background:* The chronic effect of cigarette (cig.) smoking is well established. The acute effect of smoking abolishes the concept, argued by heavy smokers, to decrease the number of smoked cigarettes instead of quitting.
Aim: To detect the acute effects of cigarette smoking and the duration of these effects.
Patients and methods: Thirty four smokers (age 21–35 years) were studied at 3 occasions; 9 h after the last cig. smoking, 5 min after one cig. smoking and 30 min after 3 cig. smoking within 30 min. They were subjected to measurement of both ventricular functions using standard and tissue Doppler imaging (TDI), aortic distensibility, stiffness and endothelial function assessment by endothelium-dependent flow-mediated dilatation (FMD) and maximum vasodilatation.
Results: After one cigarette smoking, we found a statistically significant effect on blood pressure, Heart Rate, FMD percent, Dilation Ratio, aortic distensibility ($P = 0.007$), and aortic stiffness index (ASI) ($P = 0.01$). Furthermore the LV diastolic function was significantly impaired after smoking. Despite disappearance of acute effect of 3 cig. smoking within 30 min on blood pressure, Heart Rate and aortic distensibility, a significant difference was still found as regards FMD percent and dilation ratio denoting the extension of the endothelial dysfunction for more than 30 min after the last cigarette.
Conclusion: Many acute changes occur following one cigarette smoking even in habitual smokers. Persistence of endothelial dysfunction parameters after smoking indicates the failure of circulation adaptation in response to such offense that might contribute to the precipitation of acute events in vulnerable patients.

1. Background

Smoking is one of the most important modifiable risk factors for coronary artery disease. Almost one in five adults is a current smoker in western societies and the numbers are higher in developing countries.[1]

Endothelial dysfunction, an early phenomenon in atherogenesis, has been described in brachial arteries of healthy chronic and passive smokers.[2] Long-term smoking gradually

* Corresponding author. Tel.: +20 1146555409.
E-mail address: amrsalahamin@hotmail.com (A.S. Amin).
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impairs the endothelial function in habitual and passive smokers, but it is still unclear whether smokers and nonsmokers have different acute responses regarding the endothelial dysfunction caused by cigarette smoking.[3]

2. Aim of the work

The aim of this work is to examine the acute effects of cigarette smoking on some circulatory parameters and the duration of these effects.

3. Patients and methods

This prospective, case-control study was carried out at the cardiology department, Al-Minia University Hospital, during the period from September 2009 to January 2010. Thirty four male healthy chronic smokers (Age < 35 years) were studied under 3 situations:

- Occasion 1: 9 h after last cigarette smoking.
- Occasion 2: 5 min after 1 cigarette smoking.
- Occasion 3: Half an hour after smoking 3 cigarettes within 30 min.

Subjects with any of the following criteria had been excluded from the Study:

1. Hypertension: either by history or by blood pressure measurement.
2. Other cardiovascular diseases (e.g., rheumatic, congenital, ischemic . . .etc.)
3. Diabetes: either by history or by estimation of fasting blood sugar (> 126 mg /dl).

All participants were subjected to:

- History and clinical examination for smoking index, exclusion of any cardiovascular diseases and diabetes and blood pressure measurement. Normal Bp was defined as < 140/90 mmHg.
- Laboratory assessment of fasting blood sugar and lipogram.
- Standard 12-lead ECG.
- Standard Echocardiography using General Electric vivid 3 ultrasound unit equipped with 2.5–3.5 MHz transducer. All measurements represent a mean of at least three consecutive cardiac cycles. Normal Diastolic function was defined as E/A ratio > 1. Ejection fraction (EF) was obtained by M-mode approach.
- Assessment of the Endothelial Function.

Endothelial function was assessed by measuring brachial artery flow–mediated vasodilation using a high-resolution 7.5 MHz ultrasound probe before and after one minute release from 5 min occlusion of the brachial artery[4](Fig. 1) and comparing it with total vasodilatation capacity of the blood vessel 5 min after sublingual administration of 5 mg isosorbide mono-nitrate.

4. Evaluation of aortic elastic properties

The aortic stiffness is measured using the following formulae[5]:

- Aortic diameter change (mm) = SD – DD.
- Aortic strain index = (SD − DD) × 100/DD.
- Aortic stiffness index = *ln*(SBP/DBP)/aortic strain.
- Aortic distensibility = (2 × strain)/(SBP—DBP).

Where SBP and DBP are the systolic and diastolic Bp respectively, SD is systolic aortic diameter and DD is diastolic aortic diameter measured with echocardiography 3–4 cm above the aortic valve from a transthoracic parasternal long-axis view and ln is the natural logarithm.

5. Statistical methods

Data were analyzed using Statistical Package for the Social Sciences (SPSS) v 10. Non-parametric variables were presented as numbers and % and compared using Chi-square test, while parametric data were expressed as mean ± standard deviation and compared using Student's paired *t*-test. Pearson's co-efficient (*r*) was used for correlation studies of any two variables. A probability level of $p < 0.05$ is considered significant in all tests.

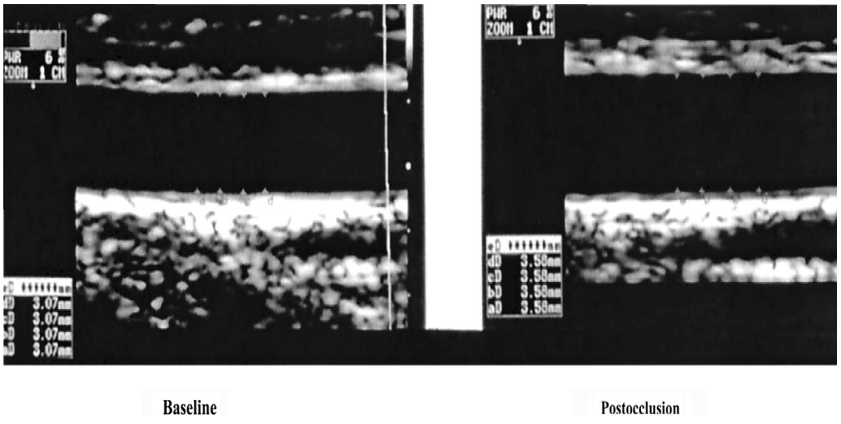

Figure 1 Ultrasound images of a brachial artery under baseline and after cuff-occlusion conditions.