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EFFECTS OF SMOKING ON CARDIAC FUNCTION TESTS AMONG MALE SAUDI YOUNGSTERS

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ABSTRACT

The present study was designed to provide additional information on the effects of smoking on cardiac function tests with serum lipid profile. As a cross-sectional case-control, this study was designed to include 50 healthy and non-smoking young males and 50 young smokers. All participants were in age and body-mass-index matched. After informed consent was taken, blood was collected in fasting status to determine serum cardiac markers along with lipid profile. The results of smokers comparing to non-smoker group showed insignificant reduction in cardiac Troponin I [cTnI] ($p > 0.05$), but a significant rise in creatine kinase [CK-MB], C-reactive protein [CRP], homocysteine [Hyc] ($p < 0.001$). Moreover, the lipid results showed that only total cholesterol was increased significantly in smokers ($p < 0.001$) although the other lipid results were insignificantly changed in the serum of smokers. All these results, especially, the low cTnI level with high CK-MB and high cholesterol with normal HDL could not serve as a predictive or prognostic index for cardiac evaluation, and still it is not clear by which mechanism smoking exerts its detrimental effects on cardiovascular disease.

Key Words: Smoking, Troponin I, C-reactive protein, homocysteine, CK-MB, cholesterol, triglyceride, HDL, LDL

INTRODUCTION

The prevalence of smoking in Saudi Arabia, according to the most recent nationally representative study, was 12.2% in 2013. Smoking prevalence was 27.9% among males (Algabbani *et al*., 2018). Official figures from 2012 indicate there were somewhere around six million smokers in Saudi Arabia, including eight hundred thousand students in intermediate and high school. Smoking in Saudi Arabia is banned in all places associated with religion, education, public events as well as government facilities, restaurants and cafés (Habib, 2012, Moradi-Lakeh *et al*., 2015). A study of Bassiony (Bassiony, 2009) found smoking in all age groups was widespread. About 13.5% of smokers are university students and about 25% are elderly people.

Cigarette smoke has c.4000 chemicals-none beneficial (Ezeugwunne *et al*., 2018).

Smoking is a major risk factor for the development of chronic diseases, including cardiovascular diseases and lung cancers. People who smoke are almost double as supposed to have a cardiac attack as opposed to individuals with no history of smoke. It is recognized by the World Health Organization as the second leading risk factor for death Worldwide (Liu *et al*., 2016). Coronary artery disease (CAD) is relatively rare in young persons, occurring in 6–10% of all patients less than 40 years of age; however, there has recently been an increase in incidence (Maroszynska-Dmoch *et al*., 2016).

The influence of smoking on the cardiovascular system is mediated by several pathophysiological pathways, including (but not limited to) vasomotor dysfunction, inflammation, and smooth muscle proliferation, as well as platelet dysfunction and alterations of antithrombotic and prothrombotic factors (Lyngbakken *et al*., 2016). Smoking destroys the lining of the arteries, resulting in accumulation of fatty materials (atheroma) which narrows the artery. This can lead to angina, a cardiac attack or a stroke. The nicotine in cigarettes stimulates the body to produce adrenaline, which affects heart functions negatively (Liu *et al*., 2016). It has been postulated that many of the adverse effects of smoking could be caused by oxidative damage to critical biological substances (Pasupathi *et al*., 2009).

The prevalence of smoking among adolescents is steadily increasing in Saudi Arabia. The initiation of smoking occurs mainly during adolescence, and is significantly associated with male gender, stress and family members or friends who smoke (Al-Jdani *et al*., 2018). The aim of this study was to estimate some of the cardiac markers in serum of the smoker young Saudi males and compare them with non-smokers.

MATERIALS AND METHODS

This study was designed as a cross-sectional case control study. One hundred volunteers participated in the study. They were in age of 18 to 23 years and Body Mass Index (BMI) of 23 to 25 divided into smokers (n=50) and non-smokers (n=50). Demographic characters of the subject are presented in Table 1. Persons those refused to participate in the study were excluded as well as those with fetal malformation, serious or chronic illness, ex-smokers, endocrine disorders, and immoderate obesity or leanness; also those already have diagnosed with cardiac abnormalities, pulmonary system diseases, neurological disease, and metabolic diseases. The study was approved by the ethics committee of Faculty of Medical Laboratory Sciences of Omdurman Islamic University. Pre-prepared questionnaire including data concerning participants and their smoking information (such as age, family history, duration of smoking, and number of cigarettes per day) was employed. In addition, the objectives and procedures of the study were explained to all participants and written informed consent forms were obtained. The BMI was calculated by dividing the body weight by the square of body height (MacKay, 2010) (Table 1). Venous blood samples were withdrawn from all subjects when they were fasting during the daytime between 9-11 am. Six mL venous blood was obtained from the antecubital vein by standard venipuncture techniques without venous stasis (Nikolac *et al*., 2013), in SST gel separated serum tubes. Following collection, samples were left to clot, then centrifuged for 5 min at 15000 RPM and serum was separated and stored at - 4°C till time of analysis. Then serum was used to determine cardiac function tests which includes creatine kinase-MB [CK-MB], C-reactive protein [CRP], total homocysteine [Hyc] and troponin I [cTnI] using commercially available ELISA kits from Fine Biotech Company; Wuhan, China. Also the serum was used to estimate cholesterol [CHOL], triglyceride [TG], high density lipoprotein [HDL], and low density lipoprotein [LDL] using commercially available enzymatic kits from Randox Laboratories Ltd; Crumlin, United Kingdom.

Statistical Analysis: The level of significance was determined using repeated measures ANOVA followed by Tukey's post-test. $P<0.05$ was considered significant. Data analysis was performed using SPSS version 22 (SPSS Inc., Chicago, IL, USA). All values are mean ± SD.

RESULTS AND DISCUSSION

Smoking is the most important and preventable cause of morbidity and premature mortality in the developed and developing world. The overall smoking rate in Saudi Arabia has slowly diminished over the past decades, transforming the habit from a cultural centerpiece to a target of social exclusion. Despite frequent and repeated warnings about the dangers of smoking, many smokers reduce the risk of getting one, especially cardiovascular disease (Lyngbakken *et al*., 2016).

In the present study the levels of CK-MB, Hyc and CRP in smoker young men showed significant increasing when compared to non-smoker control persons ($p <0.0001$). On the other hand, serum cTnI showed insignificant decreasing level in smokers compared to control (Table 2). Willerson *et al*. (2004) explained this in-correlation as that one or more components of tobacco smoke reduce the rate of low-grade cardiomyocyte turnover, which results in lower circulating cardiac troponin levels, potentially via inhibition of the immune activation characteristic of atherosclerosis and heart failure. Both reduced left ventricular mass and myocardial fibrosis may partly explain the lower levels of cTnI in current smokers (Lyngbakken *et al*., 2016). Another study of Safdar *et al*. (2014) indicated that a normal troponin with an elevated creatine kinase (CK)-MB imparts poor prognosis for the cardiovascular syndrome.

Table 1. Demographic characteristics of the participants (n=50 in each group).

	Control	smokers	P value
Age (years)	21.6 ± 2.51	21.1 ± 4.68	0.5533
BMI	23.8 ± 7.09	24 ± 6.22	0.8937
Student	25 (50%)	25 (50%)	NA
Employee	25 (50%)	25 (50%)	NA
Married	30 (60%)	37 (74%)	NA
Not married	20 (40%)	13 (26%)	NA
No. cigarettes/day	-	5 - 16	NA
Duration of smoking (years)	-	1 - 3	NA

Values are showed in mean±SD

Table 2. Cardiac markers levels between smokers and non-smokers (n=50 in each group).

	Control	Smokers	P value
Troponin (ng/L)	3.2 ± 0.58	2.7 ± 1.12	0.3292
CRP (mg/L)	1.69 ± 0.51	4.49 ± 2.67	< 0.0001
Homocysteine (µmol/L)	9.07 ± 2.91	17.51 ± 5.37	< 0.0001
CK-MB (U/L)	22.94 ± 7.43	55.57 ± 10.48	< 0.0001

Values are showed in mean ± SD

Table 3. Lipid profile levels between smokers and non-smokers (n=50 in each group).

	Control	Smokers	P value
Cholesterol (mmol/l)	3.74 ± 1.32	6.08 ± 1.56	< 0.0001
TG (mmol/l)	1.12 ± 0.88	1.35 ± 0.91	0.4592
HDL (mmol/l)	1.04 ± 0.72	0.94 ± 0.44	0.1466
LDL (mmol/l)	3.68 ± 1.17	3.81 ± 0.92	0.2172

Values are showed in mean ± SD

This study revealed that serum cholesterol level in the smoker group was increased significantly when compared to the control group ($P < 0.0001$). Furthermore, serum levels of triglyceride and LDL of the smoker group were insignificantly increased comparing to the control group ($p > 0.05$). While the level of HDL was decreased insignificantly comparing to the control group ($p > 0.05$) (Table 3). Although these findings were in the same line of many studies (Maroszynska-Dmoch *et al*., 2016, Pasupathi *et al*., 2009), but some studies showed a significant increase in TG and LDL levels associated with smoking (Neki NS, 2002) but elevant to the duration of smoking and number of cigarettes consumed in a day.

Cigarette smoking substantially increases the risk of coronary heart disease and ischaemic stroke, but it is likely that this will not be directly caused unless other health factors such as diabetes, hypertension, and obesity interfere. The increased risk of heart disease which often interferes with smoking is taking place by altering the lipids concentration in the blood (Neki NS, 2002). Hyperlipidemia is a well-known risk factor for the development of cardiovascular disease. Evidence suggests that oxidatively modified LDL contribute to the pathogenesis of atherosclerosis. Increased oxidative stress and the generation of the free oxygen radicals can result in modification of LDL to oxidized LDL that could lead to heart problems (Pasupathi *et al*., 2009).

Conclusion:

Smoking cigarettes altered cardiac markers (CRP, Hyc, CK-MB) significantly along with serum cholesterol. On the other hand, smoking also decreased cardiac troponin I insignificantly. These findings may suggest that smoking may increase the incidence of coronary heart disease but not as an only factor. Hence, smoking abstain is needed as a factor in the control of heart diseases.

Conflict of Interests

The author stated no conflict of interest about this article.

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