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Evaluation the inflammatory marks in a specimen of Iraqi smokers.

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ABSTRACT

This study included 60 person (Iraqi) were they divided into three groups: heavy smokers (20 persons) who smoking more than 20 cigarettes / day, mild smokers (20 person) who smoking less than 20 cigarettes/day, and third group non -smokers (20 persons) as a control group. In each group the concentration IL-6 of CRP levels were evaluate in sera of smokers and control by ELISA technique. The analysis results show a significant difference ($p<0.05$) in IL-6 and CRP concentration between studied groups. We suggest from result that IL-6 and CRP as important marks and had a role in inducing diseases in smoker's person.

Keywords: smoking, inflammatory marks, IL-6, cytokine

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INTRODUCTION

Cigarette smoking is a complex mixture of chemicals such as carbon monoxide ,hydrogen cyanide ,it effects on the human health ,immunological changes in smokers are crucial in the pathogenesis of smoking related disorder (1).Nicotine is one of the main constitute of cigarette smokers, which is suppress the immune system (2).The effect of smoking on the immune system not only occurs in active smokers ,but also in those exposed to smoke passively in the contaminated environment (3).The Nicotine in cigarette smoke may suppress the immune system but might have therapeutic potential as a neuroprotective and anti-inflammatory agent (3).other study showed that smoking cession is most effective method of prophylaxis of treatment of diseases related to tobacco smoking (1).smoking induced inflammation of immune system modulation are emerging as potential important mechanism to develop cancer, smoking may increase the number of macrophages ,neutrophil and alter macrophage and neutrophil function(4).smoking triggers immunological response. CRP and WBC were increased ,also there are changes in level of total cholesterol ,high density lipoprotein cholesterol ,triglyceride ,systolic blood pressure and diabetes(5).other study showed WBC was increased but not CRP level and smoking cessation didn't reduce CRP level (6).CRP were not associated with smoking especially in current smokers (7).from the above ,the present study was done to elevated the inflammatory marks in a sample of Iraqi smokers.

MATERIALS AND METHOD

This study conducted on (60) individuals of smokers ,and divided into three groups according to number of cigarettes (heavy smokers> 20,mild <20 and nonsmoker as a control). IL-6, CRP were evaluated according to manufacture company ;Abcam,DRG respectively by ELISA.

RESULTS AND DISCUSSION

The present study showed significant differences in concentration of IL-6 as in table (1) between studied groups.

Table (1) concentration of IL-6 in studied groups

IL-6 pg/ml	Heavy smokers	Mild smokers	Control(nonsmoker)	p-value
	48±0.01	43±0.21	12±0.31	P=0.02

The result of this study showed also significant differences at $p < 0.05$ in concentration of CRP as in table (2).

Table (2) CRP concentration in studied group

CRP mg/l	Heavy smokers	Mild smokers	Control
	6.133±0.21	**4.75±0.091**	**3.55±0.018**

The inflammation is associated with different chronic conditions ,reducing the inflammation may help to prevent the disease .CRP at IL-6aid to prognosis the inflammation , is a part of immune reactions leads to release CRP into blood ,IL-6 is the major factor driving the elevation of CRP(8).

Smoking is risk factor of chronic condition such as lung cancer .The potential significant IL-6 of CRP has been suggested in growth of progression of many malignancies (9).

Other study showed decrease IL-6concentration this impairment is related to both decreased production at antigenic protein(10).Serum CRP ,the main acute phase protein ,is a sensitive markers for systematic inflammation in human.it produced by liver in response to pro inflammatory cytokine induced by inflammatory stimuli cigarettes smoking is a classical and a major risk factor for development of condition which can be assessed by serum CRP(11).

IL-8,IL-6 and CRP are associated with lung cancer may be a marker to predicting subsequent lung cancer (12).

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