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The Effect of Cigarette and Water Pipe Smoking on Some of Blood Parameter

Amal K. Abbas*, Layla Fouad Ali

Department of Biology, College of Science, University of Baghdad, Baghdad, Iraq

Abstract:

Current study aimed to investigate the effect of cigarette and water pipe smoking on smoker immunity comparing to non smoker. Twenty blood samples were collected from smoker students and twenty blood samples from non smoker students from Baghdad university in 2014. The results showed that there was increase in W.B.Cs total count in smoker group comparing to non smoker in a significant differences $P < 0.05$. The differential W.B.Cs count results shows that there are increase was neutrophiles and Monocytes while there is decrease in lymphocytes in smoker group comparing to non smokers in a significant differences $P < 0.05$. Also the results revealed that there is an increase in percentage of phagocytosis to *Staphylococcus aureus* bacteria by phagocytic cells in smoker group comparing to non smoker group.

Keywords: cigarette, smoking, immunity.

تأثير تدخين السكائر والنركيلة في بعض معايير الدم

أمال خضير عباس*، ليلي فؤاد علي

قسم علوم الحياة، كلية العلوم، جامعة بغداد، بغداد، العراق

الخلاصة

تهدف الدراسة الحالية إلى دراسة تأثير السكائر والنركيلة على مناعة المدخنين مقارنة مع غير المدخنين. تم جمع عشرين عينة دم من طلاب مدخنين وعشرين عينة دم من طلاب غير مدخنين من جامعة بغداد في 2014. أظهرت النتائج أن هناك زيادة في مجموع عدد الكريات البيضاء في مجموعة المدخنين مقارنة مع غير المدخنين بفروق معنوية. كما بينت الدراسة أن هناك زيادة في عدد الخلايا العدلة و الخلايا وحيدة النواة في حين أن هناك انخفاض في عدد الخلايا اللمفاوية في المدخنين مقارنة مع غير المدخنين وبفروق معنوية. كما أظهرت النتائج أن هناك زيادة في النسبة المئوية في عملية البلعمة لبكتريا المكورات العنقودية من قبل الخلايا البلعمية في مجموعة المدخنين مقارنة مع غير المدخنين وبفروق معنوية.

Introduction

The effects of smoking are the circumstances, mechanisms, and factors by which tobacco consumption affect on human health [1]. Tobacco is the greatest cause of preventable death globally [2]. Tobacco use leads to diseases affecting the heart, liver and lungs. Smoking is a major risk factor for heart attacks, chronic obstructive pulmonary disease (including emphysema and chronic bronchitis), larynx, mouth and pancreatic cancer. It also causes peripheral vascular disease and hypertension. The effects depend on the how many years that a person smokes and on how much the person smokes. Starting smoking earlier in life and smoking cigarettes increases the risk of these

*Email: Amalk.abbas50@yahoo.com

diseases [3-4]. The World Health Organization (WHO) estimates that tobacco resulted in 5.4 million deaths in 2004 and 100 million deaths over the course of the 20th century [5-6].

Ingesting of nicotine compound by smoking is one of the rapid and efficient methods of introducing it into the bloodstream. It takes nearly ten seconds for the substance to reach the brain. The efficiency of this delivery system, smokers they are unable to cease. For those who attempt cessation for three months without succumbing to nicotine are able to remain smoke-free for the rest of their lives [7]. Smoking is being a major risk factor for myocardial infarction heart attacks, emphysema, and cancer [8].

Smoking suppresses the immune host responses. Hemorrhagic response of the periodontal tissue is decrease in smokers. Though studies state that smokers have increased number of neutrophils which is the first line of defence against bacterial infection but they have decreased the activity of neutrophils including chemotaxis, phagocytosis, adherence and cytokines production. Immunoglobulins particularly IgG2, an important antibody against gram negative pathogens is decreased in smokers when compared to non-smokers [9]. Tobacco smoke exposure to neutrophils elevates the oxidative burst causing tissue destruction by a direct toxic effect [10]. Smoking also affects a number of biomarkers which have observed to affect periodontal tissues, for example smokers have reduced the levels of prostaglandin (PG) E2, lactoferrin, lactate dehydrogenase and alkaline phosphatase [11]. Smoking has a detrimental effect on cytokines, as it reduces concentration of interleukin (IL)-1, IL-1 β in gingival crevicular fluid [12].

This study was done to estimate some blood parameters and phagocytotic activity in cigarette and water pipe smokers to find out the effects of smoking.

Materials and Methods:

This study was performed using (40) samples of blood collected from Baghdad university healthy students. Twenty samples were collected from smoker students within two months represent the experimental group and the other twenty were collected from non smoker students who were not suffering from any chronic inflammation as a control group. The samples were subjected to W.B.Cs total count, W.B.Cs differential count test [13]. The blood samples (320microliter) from EDTA tube were put in automated analyzer for hematology (analysis system) called CELL-DYN RUBY; list number 08H56-02 which made by USA, and the phagocytosis was performed in vitro and had been done according to *Furth et al.*, (1985) as follows [14].

Equal volumes (250 μ l) of heparinized blood and bacterial suspension of *Staphylococcus aureus* (106/ml) (1:1) were mixed into sterile test tube. The mixture was incubated in water bath at 37°C for 30 min with continuously shaking. Smear had been prepared by taking a drop of the mixture on the slide; duplicate slides were made for each tube. Slides had been air dried, fixed by absolute methanol, stained by giemsa stain for 10 minutes and then washed by D.W. The slides had been examined by oil immersion lens to calculate the number of neutrophils engulfed microorganism. The percentage of phagocytic cells had been calculated.

Statistical Analysis

The statistical analysis had been performed by analysis of variance (ANOVA test). The comparison between the groups was performed using one-way analysis of the variance. In order to find the source of this difference, the least significant of difference (LSD) was used.

Results and Discussion:

The results of this study are illustrated in three tables-1, -2 and -3 as shown below and the figure-1 show phagocytosis in phagocytic cells.

Table 1-The mean values of W.B.Cs. count

Group	Mean of W.B.Cs count cell/mml ³ M \pm SE
Experimental	9761 \pm 378.16*
Control	8566 \pm 435

*P \leq 0.05

Table 2-The mean values of W.B.Cs differential count

Group	Neutrophile M ± SE	Lymphocyte M ± SE	Monocyte M ± SE	Eiosinophile M ± SE	Basophile M ± SE
Experimental	65.25 ±1.18*	33.2 ±0.74*	9.25 ±0.47*	1.03 ±0.05	0.50 ±0.05
Control	58 ±1.24	38.5 ±0.95	7.22 ±0.47	0.50 ±0.50	0

*P ≤ 0.05

Table 3-The mean values of Phagocytosis%

Group	Phagocytosis%
Experimental	40.66±2.05*
Control	27.93±1.78

P≤0.05

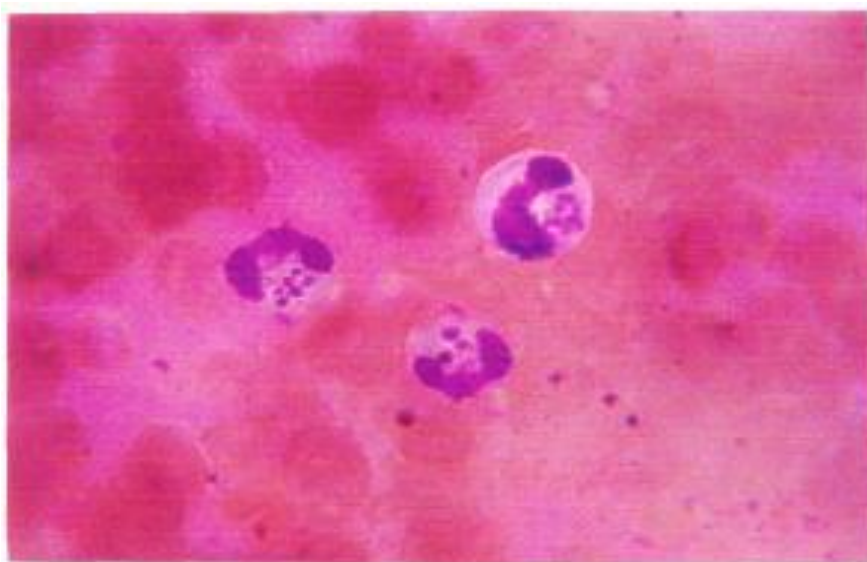


Figure 1-Phagocytosis of *S. aureus* by phagocytic cell.

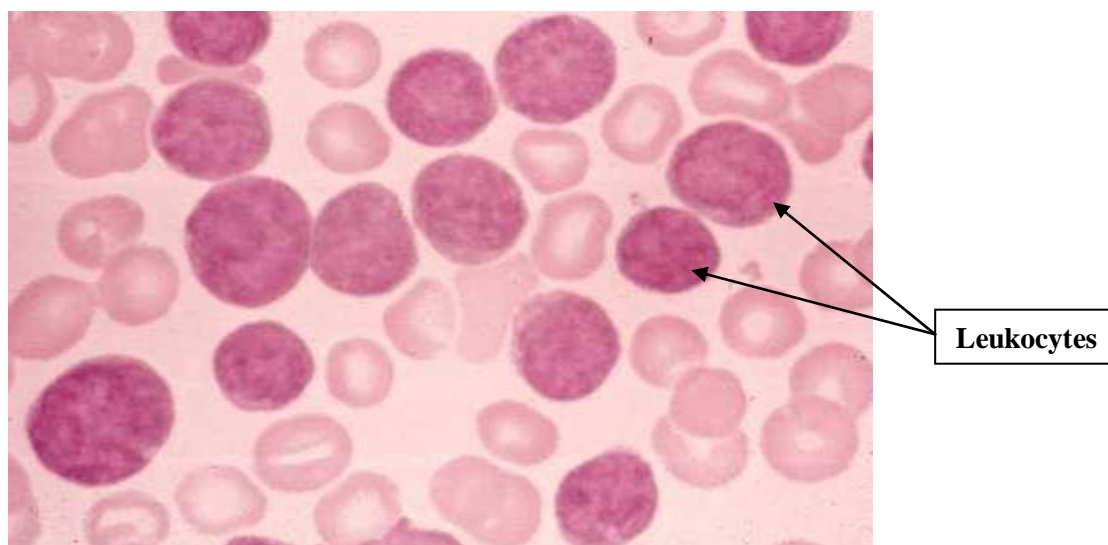


Figure 2-Leucocytes in smoker blood sample.

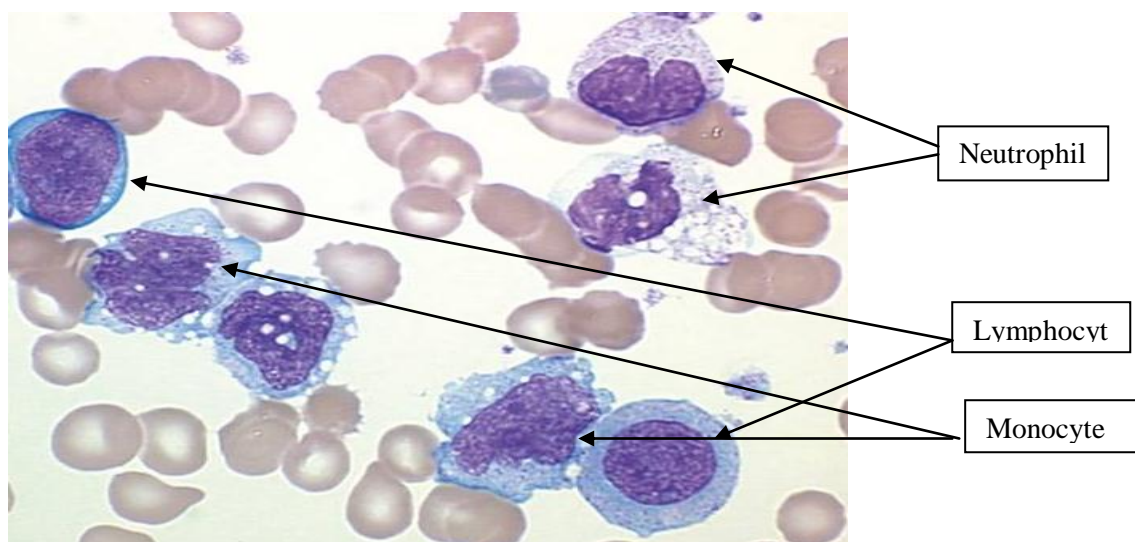


Figure 3-White blood cells in smoker blood sample.

The results showed that there is increase in W.B.Cs total count in smoker group comparing to non smoker in a significant differences $P < 0.05$. The study results are agreed with another study which revealed the higher white blood cell counts in smokers compared with nonsmokers [15]. Biologic mechanisms for a persistent effect of smoking in white blood cell counts have been proposed, such as a residual chronic inflammation of the bronchial tree due to smoking [16]. Another study showed that for adults, white blood cell count (WBC) is significantly higher in current cigarette smokers than in nonsmokers or former smokers [17]. Although these associations do not necessarily indicate causality, one hypothesis that has been put forward is that increased WBC reflects cellular injury of importance in both CVD and cancer [18].

The differential W.B.Cs count results shows that there are increase in neutrophils and Monocytes while there is decrease in lymphocytes in smoker group comparing to non smoker in a significant differences $P < 0.05$. Also the results revealed that there is an increase in phagocytosis% in smoking group comparing to non smoker group. These results are agreed with the study of Susan *et al.* in 2014, which indicate that individuals showed significantly higher increases in neutrophils [19]. Smoking influences the total number of lymphocytes and the relative distribution of the different lymphocyte subtypes and smoking may impair lymphocyte function. The relation of lymphocyte numbers to lung function and the immediate changes in lymphocyte numbers after the cessation of smoking has not been explored [20]. Smoking may cause substantial changes in the distribution of lymphocyte subtypes and impairment of lymphocyte function. The decrease in the lymphocyte count after smoking cessation suggested a direct influence of nicotine on numbers of lymphocytes [21]. Cigarette smoking adversely affects the immune system, and is a risk factor for developing osteoporosis. Since lymphocytes help maintain bone homeostasis and lymphocyte depletion results in bone loss, one potential mechanism for how smoke exposure promotes osteoporosis is by reducing bone marrow lymphocytes. Chronic cigarette smoking stimulates the bone marrow, increases the size of the mitotic and postmitotic pools of PMN, and reduces the time PMN spend in the postmitotic pool in the marrow. These changes may contribute to the leukocytosis seen in cigarette smokers [23]. The present study revealed that phagocytosis% was elevated in smoker students. In a study, one of the most common chronic diseases associated with cigarette smoking, is characterized by neutrophil-dominant inflammation. Lymphocytes levels in peripheral blood were significantly higher in blood of non-smoking compared to smokers which could be resulted from lymphocyte migration to the damaged sites to to [24].

This study concludes that Cigarette and water pipe smoking exerts a great influence on the immune system. The smokers have increased total white blood cell count, decreased lymphocytes ratios and increase in phagocytosis% which play a major role in lung defense mechanisms.

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