Changes of PLT and MPV in Smokers Comparing With Non-Smokers

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ABSTRACT

Tobacco cigarette smoking is one of causes of cardiovascular diseases, including coronary artery disease (CAD), myocardial infarction (MI), and stroke.however, the accurate of mechanisms of these disorders resulted from smoking cigarettes are still unknown. Therefore, the present study aims to investigates the relationship between effects of smoking amount (number of cigarettes smoked per day) andPLT/MPV indices. 355 subjects participated in this study including males and females with age over 17 years old. Blood sampling and (CBC) test wereconducted using MEDONIC device at Tishreen University Hospital,LatakiaCity.The results of this research showed that the smokers had significantly higher levels of (PLT) and (MPV) comparing by nonsmokers (p≤0.05). Additionally, the results showed significant relation between the smoking amount and the extent of its effects on the studied parameters.

Keywords*:* Smoking, Red blood cell, hemoglobin, hematocrit, smokers, non – smoker

I. INTRODUCTION

According to the World Health *Organization*, smoking leads to approximately 6 million preventable deaths worldwide, each year. As of 2030, it is estimated that this number will be more than 8 million [1].Although tobacco has dangerous effect on human health, it still highly consumed throughout the world. Smoking is one of the most common addictions of modern times. Its etiological agent for various chronic diseases, including a variety of infections, cancers, heart diseases and respiratory illnesses [2]. Cigarette smoke (CS) contains over 4000 compounds, including at least 200 toxicant, 80 known or suspected carcinogens. Moreover, cigarette smoking generates many toxic and carcinogenic compounds harmful to the health, such as nicotine, nitrogen oxides, carbon monoxide, hydrogen cyanide and free radicals [3]. Nicotine is commonly consumed via smoking cigarettes, cigars or pipes [4].Carbon monoxide in tobacco smoke exerts a negative effect on the heart by reducing the blood's ability to carry oxygen. Although cigarette smoking is a strong risk factor for cardiovascular disease, its relationship with hypertension remains unclear [5].The main way for the arrival of cigarette smoke into the smoker's blood by gaseous exchange that occurs in the lungs, so the lungs damaged larger than the rest of the members of the body that's why we need to discover the damages incurred in blood by smoking to save our bodies [6]. Platelets are formed from bone marrow megakaryocytes.

They are nonnucleated and possess little viable mitochondria, glycogen, many types of morphologically variant granules (dense core granules and lysosomes), and a complex membranous system. The granules contain adhesion molecules, which help in platelet–platelet interactions and platelet interactions with other blood cells, mitogenic factors, plasma proteins, some coagulation factors, and fibronectin. Platelets are important for hemostasis, wound healing, and inflammation [7].The increased platelet activity and thrombus formation and thromboembolic diseases are among the major cause of mortality in developed countries. Successful management of these diseases relies on early detection of progressive activation of coagulation. Recently, many reliable markers which play a role in the activation of coagulation, such as prothrombin fragment 1+2 and thrombin–antithrombin complex, and involved in the platelet activation, such as b-thromboglobulin or soluble platelet P-selectin have been investigated. [8] Nonetheless, the laboratory assessment of these indices is strenuous and costly. Moreover, routine laboratory tests cannot contain the aforementioned indices [9,10].Mean platelet volume (MPV) is the indicator and the function of platelet activation [11] These platelet parameters are estimated routinely by automated blood counters.As the point that platelet activation results in morphologic variations of platelets is known, a sequence of platelet parameters measured by hematology analyzers have been applied by several researchers. The MPV is perhaps the most widely studied platelet activation parameter. [12,13]There are a very few studies relating the effect of smoking on platelets. [14,15]. In addition, many of the studies have not compared the data with those of

the nonsmoking control groups Kario et al. [16] found elevated MPV in smoking patients, which reduced after the patients stopped smoking. However, Butkiewicz et al. [17,18] studied the impact of smoking on platelet activation and few other morphological indices including MPV and found no effect on MPV by smoking. Thus, studies on this have reported conflicting results. [19] Hence, this work was undertaken to study the effect of cigarette smoking on platelet parameters. We postulated the hypothesis that smoking contributesto heightened platelet reactivity and, hence, will result in an increase in the platelet volume indices.UU

II. MATERIAL AND METHODS

In this cross-sectional study. Samples were randomly collected for 355 female and male volunteers aged over 17 years. The study included healthy smokers and non-smokers, CBC analyzes were conducted at Tishreen University Hospital in Lattakia during the period of sampling from September 2018 to February 2019. The socioeconomic status, age, height, weight, and daily activity were comparable between the study and the control groups. Subjects with history of coagulation disorders, diabetes, hyperlipidemia, hypertension, peripheral vascular disease, chronic renal disease, hypertension, and any infectious or debilitating illness and those who are on any medication such as aspirin or nonsteroidal anti-inflammatory drugs (NSAIDs)were excluded from this study.

All subjects were free from other habits such as tobacco chewing and alcohol intake. The subjects who were passive smokers and ex-smokers and those who underwent radiotherapy were also excluded. A detailed history regarding current smoking status, number of cigarettes smoked per day. Nonsmokers were the respondents who affirmed that they have not smoked yet. The samples were divided into two main groups: smokers(N=50) and non-smokers (N=305). The smokers group included three groups depending on the amount of smoking High smokers (H.S N=129) (more than 20 cigarette), Moderate smokers (M.S N=88) (10-20 cigarette), Light smokers (L.S N=88) (2-10 cigarette).[20,21]The subjects underwent the following tests: blood pressure examination to rule out hypertension, complete blood count and platelet parameters such as platelet count (PLT),(MPV) wereconducted using MEDONIC device at Tishreen University Hospital, Latakia City

Statistical Analysis

All the results of laboratory investigations were loaded in Software SPSS (version 22), and statistical significancewere analyzed using(ANOVA test-One-way analysis of variance, arithmetic mean test, and standard deviation). Results were expressed as mean ± standarddeviation (SD). The P value of ≤ 0.05 has been considered as significant.

III. RESULTS

Table 1: compares the anthropometric parameters between smokers and nonsmokers, which shows that there is no significant difference between the two groups.

Parameters	Group I, smokers (N = 305) (mean ± SD)	Group II, control subjects (N = 50) (mean ± SD)	T- test P ≤0.05
Age(year)	57.98 ± 10.42	47.50 ± 10.26	0.213
Hight(cm)	158 ± 11	161 ± 8.8	0.226
Weight (Kg)	65 ± 10.9	64.7 ± 11.6	0.926
BMI (Kg/m^2)	26.06 ± 4.03	24.81 ± 4.24	0.404

Table 2: compares the platelet parameters between smokers and nonsmokers and shows a statistically significant increase in MPV and PLT in the smokers.

No.	Blood parameters	Non-smokers N=50 Mean ± S.td	Smokers (N = 305) (mean ± SD)	*ANOVA.T* p≤0.05
1	PLT	222.42 ± 45.18	261.16 ± 71.45	*0.000***
2	MPV	8.67 ± 0.50	9.26 ± 0.09	*0.000***

Table 3: compares the platelet parameters between Light, moderate, and heavy smokers, which shows that the parameters MPV, PLT were significantly increased in heavy smokers. The abnormalities of platelet parameters were more significant when the smoking intensity increases.

Blood parameters	Light smokers N=129 Mean ± S.td	Moderate smokers N=88 Mean ± S.td	Heavy smokers N=88 Mean ± S.td	F	P≤0.05
PLT	256.00 ± 76.15	284.20 ± 73.15	301.41 ± 66.52	6.146	0.005*
MPV	8.07±0.84	8.28 ± 0.91	8.35 ± 0.90	25.03	0.000***

IV. DISCUSSION

Tobacco smoking has been associated to be a reason for various major morphological and biochemical complications in individuals. In this study, we compared the platelet parameters between smokers and nonsmokers**. [Table 1]** shows that both the groups were comparable. The experimental results showeddifferences in platelet parameters such as MPV and PLT, which were significantly high (P ≤ 0.05) in smokers when compared with nonsmokers **[Table 2]**. MPV and PLT increased during platelet activation.To achieve a larger surface area, platelets modify their shapes during activation. Their shape changes from discoid to spherical. Pseudopodia are

formed as well. On the basis of impedance technology, the hematology analyzers estimate the platelet volume by the distortion of electrical field, which depends on the platelet vertical diameter.

In acute thrombotic events, platelet activation can beassessed by an increase in MPV, a well-known marker. It is alleged that carbon monoxide (CO) establishes a vital role in the cigarette smoke-induced cardiovascular diseases. The researchers found significant correlations between MPV and COHb levels ($r = 0.55$, $P = 0.0001$) and between MPV and lactate levels ($r = 0.65$, $P = 0.0001$) after smoking and have shown that 1-h exposure to passive smoking enhances the platelet activation, which may be the mechanism that leads to an enhanced risk of thrombotic events in healthy people. It is probable that continued exposure to passive smoking might pose even higher impacts. Passive smoking exposure should be avoided by healthy people in order to prevent from increasing risk of thrombotic events. [22, 23]Varol et al. have shown that chronic smoking causes platelet activation and smoking cessation improves platelet function. However, Arslan et al. [24] investigated the effects of smoking on MPV in young healthy male population (smokers, 56; nonsmokers, 46), and they found no significant difference in MPV between the smoking and nonsmoking healthy male participants. The increase of MPV among the smokers in this study may be attributed to platelet activation Ihara et al. [25] and Khandekar et al. [26] found the same observation in patients with ischemic heart disease. The MPV, PLT were significantly increased as the intensity of smoking increases, as shown in **[Table 3]** This increase of MPV and PLT comparatively suggests that young platelets are released into circulation, which are comparatively more reactive. [27] For decades, epidemiological data have demonstrated the association of smoking with the incidence of coronary heart disease, myocardial infarction, and stroke. In majority of the acute clinical scenarios, there isincidence of thrombotic occlusion of the vessel, a process that is habitually related to platelets. Therefore, the definition of the relation between platelets and smoking seems important [28]. Hung et al. [29] demonstrated smoking-stimulated platelet aggregate formation in habitual smokers. Another effect of smoking found by different groups is an increase in the serum fibrinogen levels. In addition, platelets shape and volume might differ, even in healthy persons. Thus, sequential assessment of MPV and PLT might be beneficial but unrealistic for the identification of progressive platelet activation. Instead, concurrent enhancement of MPV and PLT might suggest platelet activation, as shown by Vagdatliet al. [30] Platelet activation by cigarette smoking is related to thrombosis formation, which may lead to initiation of myocardial infarction. A paucity of studies that estimate all of the platelet parameters, including MPV, PLT, exists in the literature. Some limitations of our study include the relatively small sample size.

V. CONCLUSION

In our study, cigarette smoking in healthy men and women was accompanied by significant effects on platelet indices, such as increase in the mean MPV and PLT values in comparison with nonsmokers, which were also pronounced in heavy smokers along with increase in MPV.

We propose that these platelet parameters should be routinely reported with other hematological parameters in complete blood count reports. In light of the adverse effects on platelet function, cessation of smoking should be encouraged. Future research should be carried out with larger sample sizes

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VII. REFERENCES

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