The effect of smoking on platelet count and platelets parameters.

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Abstract:

Background: Smokers may have increased levels of platelets and platelet agreeability, which may initiate clot formation, or decreased levels, which lead to uncontrolled bleeding.

Aim: The study aimed to investigate the effect of smoking on platelet counts and platelet parameters, mean platelet volume (MPV), platelet distribution width (PDW), Platelets Large cell ratio (P-LCR), and Platelet Crite (PCT).

Method: A cross-sectional study was conducted in Al-Khums, Libya, from June to August 2022, involving 94 participants, including 44 smokers and 50 non-smokers aged 18-40. The smokers consumed at least five cigarettes daily for at least a year without any influencing factors.

Result: This study showed that there is no significant impact of cigarette smoking on platelet count and platelet parameters when comparing smokers to non-smokers (p=0.12) Additionally, the duration of smoking (1-5, 5-10, and over 10 years) and the amount of cigars smoked per day

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did not have a significant effect on platelet count or parameters in smokers, with p-values of 0.10 and 0.25, respectively.

Conclusion: The study found no significant difference in platelet count and parameters between smokers and non-smokers, as well as among different smoking durations and quantities.

Keywords: Platelets, smokers and non- smokers.

الملخص:

الخلفية: قد يعاني المدخنون من ارتفاع في مستويات الصفائح الدموية وقابلية متزايدة لتجمعها، مما قد يُحفز تكوين الجلطات، أو انخفاض في مستوياتها، مما يؤدي إلى نزيف لا يمكن السيطرة عليه.

الهدف: هدفت الدراسة إلى دراسة تأثير التدخين على تعداد الصفائح الدموية ومؤشراتها، بما في ذلك الحجم الوسيط للصفائح الدموية (MPV)، وعرض توزيع الصفائح (PDW)، ونسبة الخلايا الكبيرة من الصفائح الدموية (P-LCR)، والحجم الكلي للصفائح الدموية (PCT).

المنهجية: أُجريت دراسة مقطعية في مدينة الخمس، ليبيا، خلال الفترة من يونيو إلى أغسطس 2022، شملت 94 مشاركًا، من بينهم 44 مدخنًا و50 غير مدخن، تتراوح أعمارهم بين 18 و40 عامًا. كان المدخنون يستهلكون ما لا يقل عن خمس سجائر يوميًا لمدة لا تقل عن سنة واحدة، دون وجود أي عوامل مؤثرة أخرى.

النتائج: أظهرت هذه الدراسة عدم وجود تأثير ذو دلالة إحصائية لتدخين السجائر على تعداد الصفائح الدموية أو مؤشراتها عند مقارنة المدخنين بغير المدخنين (القيمة الاحتمالية 0.12 = p). بالإضافة إلى ذلك، لم يكن لمدة التدخين (من 1 إلى 5 سنوات، من 5 إلى 10 سنوات، وأكثر من 10 سنوات) أو كمية السجائر المدخنة يوميًا تأثيرا ذو دلالة على تعداد الصفائح أو مؤشراتها لدى المدخنين، حيث بلغت القيم الاحتمالية 0.10 و0.25 على التوالي .

الاستنتاج: لم تجد الدراسة فروقًا ذات دلالة إحصائية في عدد الصفائح الدموية أو مؤشراتها بين المدخنين وغير المدخنين، وكذلك بين فترات وكميات التدخين المختلفة.

الكلمات المفتاحية: الصفائح الدموية، المدخنون وغير المدخنين.

Introduction:

The history of cigarette smoking started in the 15th century when Columbus imported tobacco to Europe. For the popular use of tobacco, the name "nicotine" originated from the debtor's nickname, Jaen Nicot Villeman.[1]

Heavy smoking is the most common cause of ischemic heart disease and death in 30-40-year-olds who are likely to be free from myocardial risk factors.[2]

The biomarkers most widely used to quantify tobacco exposure Include various body fluids. Blood serves as a matrix for many biomarkers that monitor tobacco exposure or effects. It can be measured using widely available procedures and equipment.[3]

Agents in smoke have a direct irritant effect on the tracheobronchial mucosa, producing inflammation, atherosclerosis, and myocardial infarction. It has also been strongly linked to cigarette smoking; causal factors include increased platelet aggregation.[4]

Platelets are regarded as the primary cardiovascular risk factor. This is due to their significant involvement in the development of acute thrombotic events and atherosclerosis.[5]

The hypercoagulable state in smokers may be the result of increased platelet aggregation and increased platelet activity, which initiate clot formation, leading to occlusive vascular disease. The most aggregating agents elevated in smokers are epinephrine and norepinephrine, and this is due to nicotine-induced stimulation of the adrenal medulla. Epinephrine binds to a specific receptor on the platelet and stimulates prostaglandin synthesis from the platelets.[2]

According to a total of 88 references published from 1974–2017 in 10 different languages and including 90 study populations with 533,242

individuals, 222,887 current smokers, 92,001 ex-smokers, and 222,538 non-smokers between the ages of 15 and 89 from 34 countries were analyzed. Meta-analyses showed that the mean of the complete blood count (CBC) was higher in smokers and ex-smokers compared to non-smokers. Differences were larger comparing smokers to non-smokers than comparing ex-smokers to non-smokers, and larger comparing heavy smokers to non-smokers than comparing light smokers to non-smokers. All analyses presented high heterogeneity in the main and sensitivity analyses but no overall publication bias.[6] Although several studies were conducted on the effect of smoking on platelet count, there are only a few reports on this topic that showed no significant difference in the platelet count and its parameters.[7] Otherwise, a 2013 study by Varol et al. found that smokers had higher MPV rates than non-smokers. Thus, the effect of smoking on the platelet count is still controversial, and it is wondered if smoking affects platelet count and platelet parameters, so this study was performed as a cross-sectional study to compare the platelet count and platelet parameters in a sample of people who are smokers and non-smokers.[8]

Methods:

An analytical cross-sectional study was conducted to evaluate the effect of smoking on platelet count and parameters among male voluntary population smokers in Al-Khoums city, Libya, from June to August 2022. Ninety-four Libyan male smokers and non-smokers aged 18 to 40 participated in the study. They were divided into 44 cigarette smokers in the test group and 50 non-smokers in the control group. The smokers were also classified based on the quantity of cigarette smoking into five groups: chain smokers (3 packs/day), heavy smokers (2 packs/day), moderate

smokers (1-2 packs/day), and light smokers (1 pack/day). Otherwise, according to the duration of smoking, it can be divided into three groups: 1–5 years, 5–10 years, and more than 10 years. To avoid any disease or medication that may affect the platelet count or parameters, people who had high blood pressure had diabetes, or had been taking antiplatelet drugs for at least 10 days prior to blood collection were excluded from the study. In addition, those with a history of blood disorders and those suffering from cardiac and/or pulmonary disorders were also excluded.

The data was collected by direct interview through a designed questionnaire, which included the age, number of cigarettes per day, duration of smoking, and excluded criteria such as diseases or medications that may affect the platelet count or parameters. After filling out and verifying the personal details and data specified in the research questionnaire form, 2.5 ml of blood was collected in an EDTA.K3 tube from each study participant at Al-Khums Hospital. The platelet counts, and platelet parameters were analyzed by an automated hematological analyzer (Sysmex XN-330) in all EDTA blood samples taken.

The study was approved by the local ethics committee of Al-Khums Hospital. Written informed consent was obtained from all participants in the study. The aim of the research was explained to all study participants, and the confidentiality and privacy of the sample and clinical data collected from the subjects were considered.

Results

Demographic features of 94 participants included 50 participants (53.2%) in the non-smokers group and 44 participants (46.8%) in the smokers group. According to the duration of smoking, the smokers were divided into three groups: 1–5 years, 5–10 years, and >10 years, with percentages

of 21.3%, 9.6%, and 46.8%, respectively. The smokers will also be divided into 4 study groups based on the daily amount of cigarettes, which include heavy smokers, moderate smokers, light smokers, and casual smokers, with percentages of 6.4%, 12.8%, 19.1%, and 8.5%, respectively, as shown in figure 1.
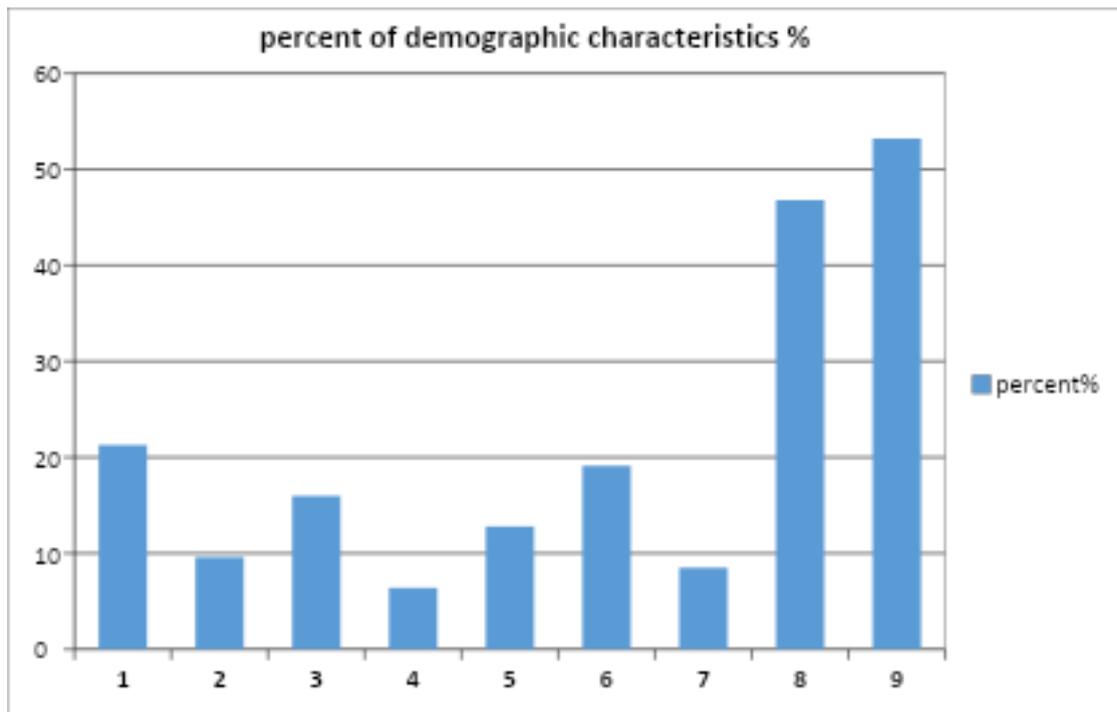

Figure 1. Descriptive statistics for demographic characteristics percentage among the study participants.

According to the Kolmogorov-Smirnov test, all variable data had a parametric normal distribution (P value > 0.05), which was shown as the mean of the platelet parameters in Table 1.

Table 1. The descriptive statistic of the platelet parameter:

Descriptive Statistics						
Platelets parameter	N	Min	Max	Mean	SD	P-value
Platelet Count	94	130	375	241.81	52.985	.200*
PDW (fL)	94	9.9	20.5	13.156	2.1465	.200*
MPV (fL)	94	8.9	12.5	10.551	.9217	.150*
PCT %	94	0.14	0.39	.2522	.04797	.200*

In table 2, normality statistical tests were applied to compare platelet parameters between smoking and non-smoking groups using the T-test.

Table 2. A T-test table shows a comparison of the platelet parameters between the non-smoking group and the smoking group.

Variables	Knowledge		P-value
	Smoker	Non-smoker	
	Mean± SD	Mean± SD	
Platelet Count	232.30±53.124	250.18±51.952	0.876*
PDW (fL)	13.377±2.1048	12.962±2.1852	0.731*
MPV (fL)	10.791±0.9437	10.340±0.8567	0.367*
PCT %	0.2473±0.05032	0.2566±0.04588	0.495*
P-LCR %	30.280±8.3096	27.924±6.5649	0.136*

The independent samples T statistic results showed that there was no statistically significant difference in platelet count and platelet parameters in the smokers group compared to the non-smokers group, as shown in figure2.
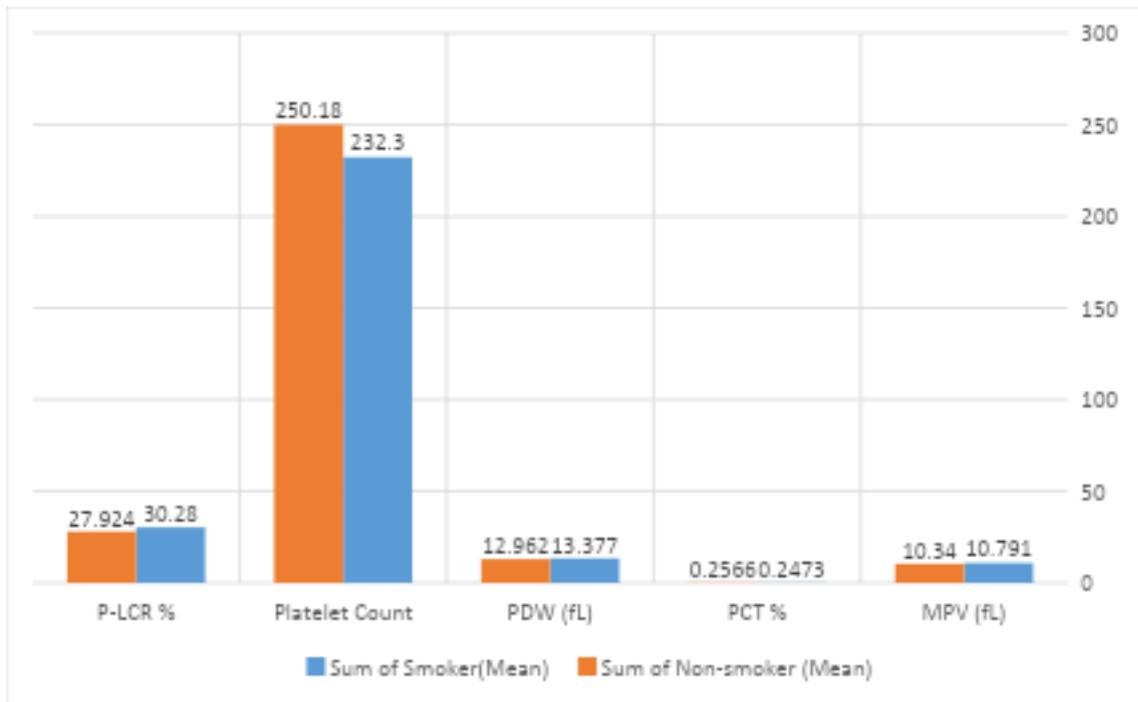
Figure 2. Comparative analysis of platelet parameters between smokers and non-smokers.

One of the aims of this study was to determine whether there was a difference in the effect of the quantity of smoking with chain-smoking, heavy smoking, moderate smoking, light smoking, and causal smoking on platelet parameters. A one-way ANOVA test was used to determine whether there

was a statistically significant difference in platelet parameters according to the quantity of smoking per day in the smokers and non-smokers groups, as shown in Table 3.

In Table 3, the p-value results were greater than 0.05, indicating that there was no statistically significant difference in platelet parameters between the study groups, both between the non-smoking and the smoking groups. In addition, there was no statistically significant difference between the smoker groups with the different amounts of smoking. The reason for the non- statistically significant differences in platelet parameters between the

study groups may be due to the convergence of mean values between the study groups.

Table 3. ANOVA test results for the effect of variation in the amount of smoking on platelet parameters.

Variable	Cigarette quantity/day	Mean	S.D	f-value	P-value	Interpretation
Platelet Count	Non-smoking	250.18	51.952			
	Heavy(2 packs /day)	216.83	56.559			
	Moderate(1-2packs /day)	223.25	55.286	1.185	0.323	Non-significant
	Light(1packs /day)	245.50	54.539			
	Causal(few cigarettes a day)	227.75	46.976			
PDW (fL)	Non-smoking	12.962	2.1852			
	Heavy(2 packs /day)	13.133	1.7535			
	Moderate(1-2packs /day)	13.583	1.9780	0.340	0.850	Non-significant
	Light(1packs /day)	13.178	2.3559			
	Causal(few cigarettes a day)	13.700	2.2463			
MPV (fL)	Non-smoking	10.340	0.8567			
	Heavy(2 packs /day)	10.817	0.9411			
	Moderate(1-2packs /day)	10.925	0.8874	1.651	0.168	Non-significant
	Light(1packs /day)	10.644	1.0303			
	Causal(few cigarettes a day)	10.900	0.9607			
PCT %	Non-smoking	0.2566	0.04588			
	Heavy(2 packs /day)	0.2317	0.05307			
	Moderate(1-2packs /day)	0.2400	0.05592	0.656	0.624	Non-significant
	Light(1packs /day)	0.2578	0.04821			
	Causal(few cigarettes a day)	0.2463	0.04897			
P-LCR %	Non-smoking	27.924	6.5649			
	Heavy (2 packs /day)	30.900	6.6399			
	Moderate (1-2packs /day)	29.067	10.1496	0.804	0.526	Non-significant
	Light (1packs /day)	30.011	8.1124			
	Causal (few cigarettes a day)	32.238	7.9714			

A one-way ANOVA was also used to compare three categorical smoking groups (1–5 years, 5–10 years, and more than 10 years of smoking) in order to determine the effect of smoking duration on platelet parameters, as shown in Table 4.

Table 4. ANOVA test results for the effect of smoking duration on platelet parameters

Variable	Duration of smoking	Mean	Std. Deviation	f-value	P-value	Interpretation
Platelet Count	Non-smoking	250.18	51.952	2.464	0.067	Non-significant
	1-5 years	250.60	47.952			
	5-10 years	215.22	45.063			
	>10 years	218.13	59.211			
PDW (fL)	Non-smoking	12.962	2.1852	0.767	0.516	Non-significant
	1-5 years	13.570	2.0676			
	5-10 years	12.611	1.9859			
	>10 years	13.580	2.2533			
MPV (fL)	Non-smoking	10.340	0.8567	2.250	0.088	Non-significant
	1-5 years	10.865	0.9571			
	5-10 years	10.533	1.0700			
	>10 years	10.847	0.8855			
PCT %	Non-smoking	0.2566	0.04588	3.262	0.025	Significant
	1-5 years	0.2695	0.04501			
	5-10 years	0.2211	0.02667			
	>10 years	0.2333	0.05715			
P-LCR %	Non-smoking	27.924	6.5649	1.113	0.348	Non-significant
	1-5 years	29.995	9.5211			
	5-10 years	28.589	8.1204			
	>10 years	31.673	6.8804			

Considering the results in Table 4, all of the platelet parameters except platelet crit (PCT) showed no statistically significant difference among

smoker groups, whereas the PCT showed a statistically significant change with the variation in the duration of smoking.

Discussion:

Smoking may have an effect on platelet count, and platelet biomarkers that include PDW, MPV, PCT%, and P-LCR% were evaluated in the smokers in this study. The main purpose of the current study was to determine and compare the platelet parameters (PlT, PDW, MPV, PCT%, and P-LCR%) in smokers and non-smokers, as well as to determine the effect of quantity and duration of smoking on the platelet parameters. Some of the studies showed a significant increase, while others showed a significant decrease or no significant difference in platelet count and platelet parameters. The results of this study showed that all of the platelet parameters showed no statistically significant difference in smokers when compared with non-smokers.

According to the findings illustrated in the previous study by Sirwansaksri et al. (2004), which showed that there were no statistically significant differences in the platelet count and platelet parameters, this result was consistent with the previous study conducted by Butkiewicz et al. (2006), which showed no significant change in Plt count, MPV, and P-LCR%.[7,9] In addition, the results of this study also agreed with the results of the study of Sirwansaksri et al. (2004) and Butkiewicz et al. (2006), where this study showed that there were no statistically significant differences in the platelet count and platelet parameters. This study also agreed with the results of the previous study by Arslan et al. (2008) since both studies showed no statistical difference in MPV between smokers and non-smokers.[10]

Otherwise, these findings in previous results disagreed with the study conducted by Gitte et al. (2004), whose study groups consisted of 120 healthy male smokers and 120 healthy male non-smokers, showing a significantly increased platelet count in smokers.[11]

Also, the results of this study on the PLT count did not agree with the study by Aghaji et al. (1990), which included 176 smokers and 176 non-smokers.[12] A study by Aghaji et al. (1990) showed that smokers had higher platelet counts than non-smokers, whereas the present study showed no significant differences in platelet count and its parameters between the smokers and non-smokers groups.[12]

Another study conducted in 2013 by Varol et al. showed that smoking was statistically significant in MPV and lower statistically significant in Plt count, which was not in agreement with the results of this study. The result of the Varol et al. (2013) study was consistent with the results from PDW in this study.[8]

The study revealed that heavy and long-term smokers exhibited lower platelet counts in comparison to non-smokers, which contrasts with some previous research findings.[9,13] This discrepancy may be attributed to chronic inflammation, oxidative stress, and the cytotoxic effects on bone marrow cells, which impair platelet production over time.[14] While acute smoking is known to increase platelet aggregation and consumption, chronic exposure disrupts the equilibrium between platelet production and destruction, leading to accelerated turnover without adequate compensation. Furthermore, genetic and metabolic variability among individuals may influence platelet behavior, with some individuals being more susceptible to bone marrow suppression or platelet destruction.[7]

Heavy smokers, defined as those who consume two or more packs of cigarettes per day, demonstrated lower platelet counts compared to lighter smokers or non-smokers. This outcome is likely due to the toxic effects of high nicotine levels, which can damage platelets or inhibit their production.[15] Chronic heavy smokers may develop compensatory mechanisms, such as the upregulation of anti-inflammatory cytokines, which mitigate excessive platelet activation and consequently reduce platelet counts over time. [8] It is also important to consider that undiagnosed conditions, such as anemia or infections, and lifestyle factors, including diet and alcohol consumption, could further confound the results.[12]

Conclusion: The results of this study concluded that cigarette smoking had no statistically significant effect on platelet count and platelet parameters (PDW, PCT, MPV, P-LCR) in smokers compared with non-smokers. Furthermore, differences in the amount of cigarette smoking with an increasing number of cigarettes smoked per day, as well as differences in smoking periods, had no statistically significant effect on platelet count and platelet parameters.

Based on the study conclusion, further prospective studies with high sample numbers are needed to explain this issue and determine the relationship between cigarette smoking and platelet function disorders, whether or not cigarette smoking activates platelets, and increase confidence in this study's findings.

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