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Research Article

Comparison between smoking and non-smoking in term of thrombocytes count and Coagulation: Is there any differences?

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Abstract

Introduction: Smoking is a major risk factor for cardiovascular disease, with a significant impact on platelet activation and thrombosis. This study aims to examine the effects of smoking on platelet activation and INR as a marker of thrombosis. This study aimed to investigate the influence of tobacco smoking on platelets count and coagulation tests (PT, PTT, INR) in adult males in sirte city.

Materials and methods: A case control study was carried out during 1/1/2023 to 15/2/2023. 200 males (100 smokers and 100 non-smokers) were participated in this study. with ages between 15-85 years enrolled in this study. Data were presented in median (interquartile range). Data was collected by using structural interviewing questionnaire. blood samples were collected in tri sodium citrate anti-coagulant for PT and INR, manually analyzed was using. Ethylene-di-amine-tetra-acetic acid (EDTA) anticoagulant for the platelets count, analyzed by (Sysmex, German) automated hematology analyzer.

Main results: The results showed that the Median platelets count was statistically no significantly between smokers (237) and non-smokers (256), U = 5.696.000, Z = 1.701, P = 0.089. Whereas, median PT was statistically significant between smokers (15) and non-smokers (14), U = 4.112.000, Z = -2.194, P = 0.028. Also, median INR was statistically significant between smokers (1.4) and non-smokers (1.3), U = 4.063.500, Z = -2.316, P = 0.021.

Conclusion: The present study shows that smokers likely to have lesser platelet counts, longer PT, and higher INR values in comparable to non-smokers. Thus, smoking might be related with bleeding complaints but additional examinations are required.

Keywords: smoking; cardiovascular disease; thrombocytes count; coagulation test

Introduction

Platelets are specialized blood cells formed and released into the bloodstream by precursor cells called megakaryocytes that lie within the bone marrow. They play a critical role in blood clot formation, impacting thrombosis and haemostasis. Because of their capability to become adhesive and release a range of mediators affecting coagulation at the site of injury, they are also involved in immunity, inflammation, and wound healing. Moreover, platelets are directly connected to the coagulation cascade and the humoral immune system through a diverse combination of surface receptors, adhesion proteins, integrins, and glycoproteins that are linked to multiple signalling pathways (Linden, 2013).

The coagulation cascade is a sequence of processes that occur during haemostasis following damage by activating a cascade of proteins known

as clotting factors. It has three pathways: intrinsic, extrinsic, and common. The coagulation pathway's main function is to maintain haemostasis, which is the prevention of bleeding or haemorrhage (Chaudhry *et al*, 2022). Each coagulation pathway comprises coagulation factors that have specific roles in haemostasis. The intrinsic pathway comprises factors I, II, IX, X, XI, and XII. The extrinsic pathway consists of factors I, II, VII, and X. The common pathway consists of factors I, II, V, VIII, X. Coagulation factors are proteins that form blood clots to stop bleeding in the presence of an injury or wound (Chaudhry et.al, 2022).

Moreover, platelets are critical mediators that initiate the mechanical pathway of the coagulation cascade when blood vessels are damaged. They promote primary haemostasis through three basic mechanisms: activation, adhesion, and aggregation. As the vascular endothelium's integrity is compromised, several macromolecular constituents of the vascular sub-endothelium become exposed and easily accessible to platelets (Periayah *et al*, 2017). Platelets play a significant role in the coagulation cascade by generating the initial haemostatic plug, which gives a surface for the synthesis of active coagulation factors, which in turn leads to the development of fibrin stabilized platelet aggregates and subsequent clot retraction (Palta *et al*, 2014).

A variety of factors can influence haemostasis. According to research, cigarette smoking is one of these factors. It can modify the main haemostatic systems and coagulation factors, affecting their functions. Further, smoking has been shown to reduce the expression of tissue factor pathway inhibitors and raise plasma fibrinogen levels in smokers compared to non-smokers (Elkhalifa, 2018). Moreover, the endothelium lining is damaged in smokers, causing platelets to stick to the subendothelial collagen, as a result, the number of platelets in circulation tends to fluctuate (Pujani et.al, 2021). According to the results of a study conducted in Nigeria, platelet count was significantly lower in smokers when compared to non-smokers. Additionally, platelet count was lower in older patients compared to younger individuals, and smokers who had smoked for more than ten years had a significant decline in platelet count when compared to those who had smoked for less time. (Okeke et.al, 2017).

Smoking may also influence coagulation screening tests, such as blood clotting time. In a study conducted in Sudan aimed to determine the effects of cigarette smoking on coagulation screening tests and platelet counts, the findings revealed that smokers had considerably lower mean of PT and INR compared to those who do not smoke (Elkhalifa, 2018). Moreover, cigarette smoke exposure raises the risk of acute myocardial infarction and sudden cardiac death, with thrombosis being responsible for the majority of these acute occurrences (Barua et.al, 2010; Ali AA et al, 2023). In addition to affecting haemostasis, numerous studies have connected tobacco use to a variety of fatal diseases such as coronary artery disease, chronic obstructive pulmonary disease, malignancies in every human organ system, and diabetes (Adams & Morris, 2022). Therefore, our study aimed to investigate the effect of cigarette smoking on platelets count and coagulation tests (PT, PTT, INR) in adult males in Sirte city, Libya.

Materials and Methods:

Study population and methodology

A case control study was carried out during 1/1/2023 to 15/2/2023. 200 males (100 smokers and 100 non-smokers) were participated in this study. with ages between 15-85 years recruited to take place in this study. Data were presented in median (interquartile range). Data was collected by using structural interviewing questionnaire (**Appendix.1**). Venous blood specimens were collected in tri sodium citrate anti-coagulant for PT and INR, manually analyzed was using. Ethylene-di-amine-tetra-acetic acid (EDTA) anticoagulant for the platelets count, analyzed by (Sysmex, German) automated hematology analyzer.

Statistical analysis

Data analysis carried out by Statistical Package for the Social Sciences (SPSS) version 25.0 (IBM Corp., Armonk, NY, USA). A Mann-whitne U-test was run to determine if were differences in Platelets count, PT and INR of smokers and non-smokers. Distributions of the engagement scores for smokers and non-smokers were similar, as assessed by visual inspection. Association between weigh lose and smoking status was assessed by Fisher's exact test.

Results

Comparison of age between study groups

200 males are participating in this study. The comparison of median age between smokers and non-smokers group is illustrated in Table 1. As can be seen from Table, the median age of smokers was (27) years, whereas the median age of non-smokers was (30) years. No statistical significance is observed between the age of two groups, U = 5.414.500, Z = 1.014, P = 0.311.

Variable	All	Smokers	Non-smokers	P-value
Age	28(23 – 35)	27(23 – 33)	30 (22 – 38)	0.311

Table 1. Comparison of median age between study groups

The average of platelets counts and coagulation profile (PT, INR) between study groups.

Table 2, shows the differences between two groups in platelet counts, PT and INR results. Median platelets count was statistically no significantly between smokers (237) and non-smokers (256), U = 5.696.000, Z = 1.701, P = 0.089. Median PT was statistically significant between smokers (15) and non-smokers (14), U = 4.112.000, Z = -2.194, P = 0.028. Median INR was statistically significant between smokers (1.4) and non-smokers (1.3), U = 4.063.500, Z = -2.316, P = 0.021.

Variable	All	Smokers	Non-smokers	P-value
Platelets count	245 (209 – 297)	237 (199 – 278)	256 (216 – 303)	0.089
PT	14 (13 – 16)	15 (13 – 16)	14 (13 – 15.75)	0.028
INR	1.3 (1.2 – 1.5)	1.4 (1.2 – 1.5)	1.3 (1.2 – 1.5)	0.021

Table 2. Differences in the average of platelet numbers and coagulation profile between the study groups.

Distribution of diseases between study population

As can be seen from Table 3, 168 males (84.0%) have not any disease. While 32 males (16.0%) have diseases such as hypertension, ulcer, respiratory diseases, diabetes and renal diseases.

Disease	Frequency	Percent
Hypertension	5	2.5%
Ulcer	6	3.0%
Respiratory diseases	12	6.0%
Diabetes	5	2.5%
Renal diseases	4	2.0%
No diseases	168	84.0%
Total	200	100.0%

Table 3: The percentage of diseases between the precipitants

Association between smoking status and weigh lose

200 males participated in the study, of the participants, 100 (50%) were smokers and 100 (50%) were non-smokers. For smokers, 36 (83.7%)) were have weigh lose, while for non-smokers, 7(16.3%)) were have weigh lose. There was statistically significant association between smoking status and weigh lose as assessed by Fisher's exact test, P = 0.000 as show that in table 4.

Variable	All n (%)	Smokers n (%)	Non-smokers n (%)	P value
Weigh lose	43 (100%)	36(83.7%)	7(16.3%)	0.000

Table 4: Association between smoking status and weigh lose

Discussion

Coagulation is the process in which vascular damage is followed by successive platelet adhesion to sites of vessel injury and the initiation of the coagulation cascade. According to research, smoking causes a state of hypercoagulability, which affects the normal platelet physiology and haemostasis pathways (Basalingappa *et al*, 2015). The results of this study shown that the average of platelets count was lower than in smokers compared to non-smokers, however, this difference was not statistically significant. This result is comparable to findings that reported by Asif, *et al* (2013), there was a non-significant decrease in platelets count in smokers compared to those who do not smoke. The drop in platelet count could be the result of smoking damage to the endothelium lining causing platelets to adhere to the sub-endothelial collagen, which in turn causes fluctuations in the amount of platelets in the bloodstream (Pujani, *et al*., 2021). Nonetheless, our findings disagree with the study that accomplished by Ghahremanfard, *et al* (2015) that reported the smokers had a significantly higher mean platelet count compared to non-smokers.

In the study that accomplished by (Elkhalifa, 2018), it reported that the mean of PT was considerably shorter in smokers compared to non-smokers. In contrast, the results of our study showed that the average of PT was significantly longer in smokers compared to non-smokers. The present study shown that smokers had a greater INR compared to non-smokers. Different findings were reported by Elkhalifa, (2018), the average INR was significantly lower in smokers compared to the non-smoker group. Data on the impact of smoking on the INR are contradictory (Ali, *et al*., 2017). However, the majority of studies indicate that smoking reduces INR levels, although the results may differ between each individual (Stading, *et al*., 2007).

Filozof, et al., (2004) reported that weight change is predicted by younger ages, lower socioeconomic level, and heavier smoking. Regarding the results of the Fisher's exact test, the findings of the present study demonstrated that there was association between smoking status and weigh lose.

Conclusion

The main aim of this research is to investigate how smoking effects on platelets count and coagulation tests. The results indicate that smoker's group had a significantly longer PT and INR value, and a slightly decrease platelet counts, in comparison to non-smokers group. Also, there was statistically significant association between smoking status and weigh lose. Therefore, smoking might be related with bleeding complaints but more examinations are required.

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Appendix

Personal data

- Age……
- Are you smoker ? ……
- How long Smoking period? .....
- How many cigarettes ?…….

Diseases	Yes	No
Atherosclerosis		
High blood pressure		
Stomach ulcer		
Kidney diseases		
Respiratory or heart diseases		
Are you take blood thinners?		
Are you suffering from weight loss or gain		

Do you suffer from one the following diseases?

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