COMPARISON OF PLATELET COUNT IN SMOKERS VERSUS NON-SMOKERS

Jaideep Misra[1], Kusuma Venkatesh[2]

[1]Junior Resident, Department of Pathology, Kempegowda Institute of Medical Sciences, Bengaluru, Karnataka.
[2]Professor, Department of Pathology, Kempegowda Institute of Medical Sciences, Bengaluru, Karnataka.

ABSTRACT

BACKGROUND

Cigarette smoking is one of the major lifestyle factors influencing the health of human beings. Smoke from cigarettes can affect nearly every organ in the body by promoting cell damage and causing inflammation. The effects of smoking are many namely cancers, lung diseases, cardiovascular diseases and circulatory problems. The primary objective of this ICMR short term studentship research was to compare blood platelet count of smokers and non-smokers.

MATERIALS AND METHODS

Twenty five healthy smokers and non-smokers each in the age group of 20 to 40 years among the hospital employees and college students were studied. Subjects suffering from chronic diseases, dengue, typhoid or those taking regular medicines for the last 2 years were excluded. The platelet count was performed by obtaining 2 millilitres of anticoagulated blood samples from the subjects after taking their informed consent and with all the aseptic precautions. The platelet count was done using an automated analyser known as Sysmax Electronic Counter.

RESULTS

The platelet count was found to be higher in smokers as compared to that of non-smokers even though there was no major significant difference. The mean platelet count of smokers was higher than that calculated for nonsmokers. The mean platelet count for smokers was 221.68 thousands/μL and for nonsmokers 216.8 thousand/μL. Data of 50 subjects have been tabulated and used in calculating the mean platelet count.

CONCLUSION

After the short-term students' study research, it was found that mean platelet count of smokers was higher than that of nonsmokers, even though the difference was not very much. It was concluded that apart from smoking having ill effect on health is associated with cardiac diseases, it also has social stigma attached with adolescents of today.

KEYWORDS

Platelet Count in Smokers, Non-Smokers, Pathology.

HOW TO CITE THIS ARTICLE: Misra J, Venkatesh K. Comparison of platelet count in smokers versus non-smokers. J. Evid. Based Med. Healthc. 2018; 5(19), 1522-1528. DOI: 10.18410/jebmh/2018/320

BACKGROUND

We are fully aware of the hazards of smoking and convinced of its major medical and social problem. Smoking in different forms is a major risk factor for atherosclerosis and coronary heart disease. Smoking is the major risk factor in the developing world but falling in developed nations. In the developing world, tobacco consumption is rising by 3.4% per year. Among young teenagers between the age of 13 to 15, about one in five smokes worldwide. Between 80, 000 and 100, 000 children worldwide start smoking every day - roughly half of whom live in Asia. In the WHO, 2002 report evidence showed that around 50% of those who start smoking in adolescent years continue smoking till the age of 20 years. Smoking results in more deaths each year than due to AIDS, alcohol, cocaine, heroin, homicides, suicides, motor vehicle crashes and fires. Cigarette smoking is now acknowledged to be one of the leading causes of preventable morbidity and mortality and is one of the largest single preventable causes of ill health by the world health organization.

Despite the statutory warning on the cigarette packs "Smoking is injurious to health", smoking is still on the rise and is one of the major lifestyle factors influencing the health of the human beings. It causes serious health problem not only to smokers but also to those exposed to it what is called as passive smoking. Lung cancer is the major danger of smokers but diseases of the blood vessels and the heart account for one third of all excess death in smokers.[1]

Tobacco smoking is one of the major factors accelerating cardiac problems. Deleterious effects of smoking are associated with generation of free radicals that break down Nitric Oxide, which on the one hand enhances thromboxane synthesis but on the other hand

Financial or Other, Competing Interest: None.
Submission 15-04-2018, Peer Review 22-04-2018,
Acceptance 30-04-2018, Published 02-05-2018.
Corresponding Author:
Dr. Jaideep Misra,
#O52, Nivedita Kunj,
R. K. Puram, Sector 10,
New Delhi – 110022.
E-mail: jaideepmisra17july@gmail.com
DOI: 10.18410/jebmh/2018/320

reduces production of prostacyclin (the antithrombic action), thus leading to clotting disorders. Addicted smokers show increased potential for platelet aggregation, lower platelet survival rate and increased excretion of thromboxane metabolites. Elevated platelet aggregation induced by passive smoking may cause an increase in the risk of cardiac ischemic disease even by 34%. The importance of platelet count per se in cardiovascular disease is indicated by several studies which suggest that increased platelet counts frequently attend hyper-coagulability and thrombosis.[2]

MATERIALS AND METHODS

This cross sectional study was carried out as a short term student ship project during by MBBS course with prior permission and clearance by the ethical committee under ICMR and done with the guidance of a professor. The study duration & report preparation was 3 months. It was done in the Department of Pathology of Kempegowda Institute of Medical Sciences, Bangalore, Karnataka.

Total Number of Subjects were 50 which included 25 Smokers and 25 of Non-Smokers. The age group of subjects was between 20 to 40 years. 25 of them having the history of smoking for atleast last 3 years and 25 of them being nonsmokers. Informed consent was taken from the subject prior to conducting the study. The inclusion criteria were that male smokers, with frequency of 5 or more cigarettes per day with atleast 3 years duration of smoking were selected for the study. The relevant personal history was obtained by personal interview with the subjects. Exclusion criteria included, subjects suffering with dengue or those who were above the age group of 40 and having history of cardiac disorder. Only those subjects were considered who were neither diabetic nor hypertensive nor taking any regular drugs for more than 2 years. For the nonsmokers group also the same exclusion criteria were followed. The platelet count for these subjects was carried out in all these subjects.

The platelet count was performed on blood samples of subjects drawn from both categories namely smokers and non-smokers by obtaining 2 millilitres of blood sample in EDTA as anticoagulant with all the aseptic precautions. The skin was wiped clean with an alcohol pad, and then a needle was inserted through the area of cleansed skin into the subject's vein which was seen from skin. The blood was then pulled from the syringe and collected in a special vacuumed vial. The samples were then analysed through an automated analyser known as Sysmax Electronic Counter. The blood was well mixed and placed on a rack in the analyser. This instrument has many different components to analyse different elements in the blood. The cell counting component helps to find the number and different types of blood cells. The same results were then reviewed.

The results of an automated cell counter are very precise as large number of cells are counted compared to manual method. However, certain small cells (microcytes and fragmented red blood cells) can be mistaken for platelets as well as abnormal cells in the blood may not be identified correctly. Therefore, to confirm the platelet count the peripheral smear study was made for each subject for counter checking the automated result.

The amount of haemoglobin and all the red cell indices are measured by the automated haematology apart from counting, measuring and analysing red blood cells, white blood cells and platelets.

RESULTS

The healthy subjects were taken in the age group of 20 to 40 years who were not in the exclusion list as mentioned earlier. The blood platelet count of the subjects ie smokers and non-smokers were tabulated. Table 1 and 2 show the platelet count of all 50 subjects. Graphical representation is also shown in Figures 1 and 2.

Table 3 gives the mean platelet count of smokers and nonsmokers whereas graphical representation is shown in Figure 3.

For Smokers

Sl. No.	Smokers – 1 to 25	Platelet Count (Thousand/uL)	Age
1.	S1	244	30
2.	S2	194	26
3.	S3	253	25
4.	S4	118	28
5.	S5	195	35
6.	S6	186	21
7.	S7	225	22
8.	S8	256	24
9.	S9	248	20
10.	S10	246	20
11.	S11	245	23
12.	S12	239	24
13.	S13	237	30
14.	S14	224	22
15.	S15	241	22
16.	S16	181	26
17.	S17	230	25
18.	S18	191	34
19.	S19	242	35
20.	S20	237	21
21.	S21	252	22
22.	S22	199	23
23.	S23	234	20
24.	S24	245	20
25.	S25	180	23

Table 1. Platelet Count (thousand/uL) in Smokers)

For Non-smokers

Sl. No.	Non Smokers NSn – 1 to 25	Platelet Count (Thousand/uL)	Age
1.	NS1	182	22
2.	NS2	233	21
3.	NS3	246	30
4.	NS4	178	26
5.	NS5	237	25
6.	NS6	215	28
7.	NS7	251	35
8.	NS8	249	21
9.	NS10	226	22
10.	NS11	247	24
11.	NS12	248	20
13.	NS13	220	20
14.	NS14	229	23
15.	NS15	231	24
16.	NS16	222	22
17.	NS17	230	20
18.	NS18	212	30
19.	NS19	233	21
20.	NS20	211	29
21.	NS21	226	20
22.	NS22	213	24
23.	NS23	240	20
24.	NS24	212	24
25.	NS25	229	22

Table 2. Platelet Count (thousand/uL) in Non-smokers)

Subject	Mean Platelet Count (thousand/uL)
Smoker (S)	221.68
Non-Smoker (NS)	216.80

Table 3. Mean Platelet Count of Smokers and Non-Smokers

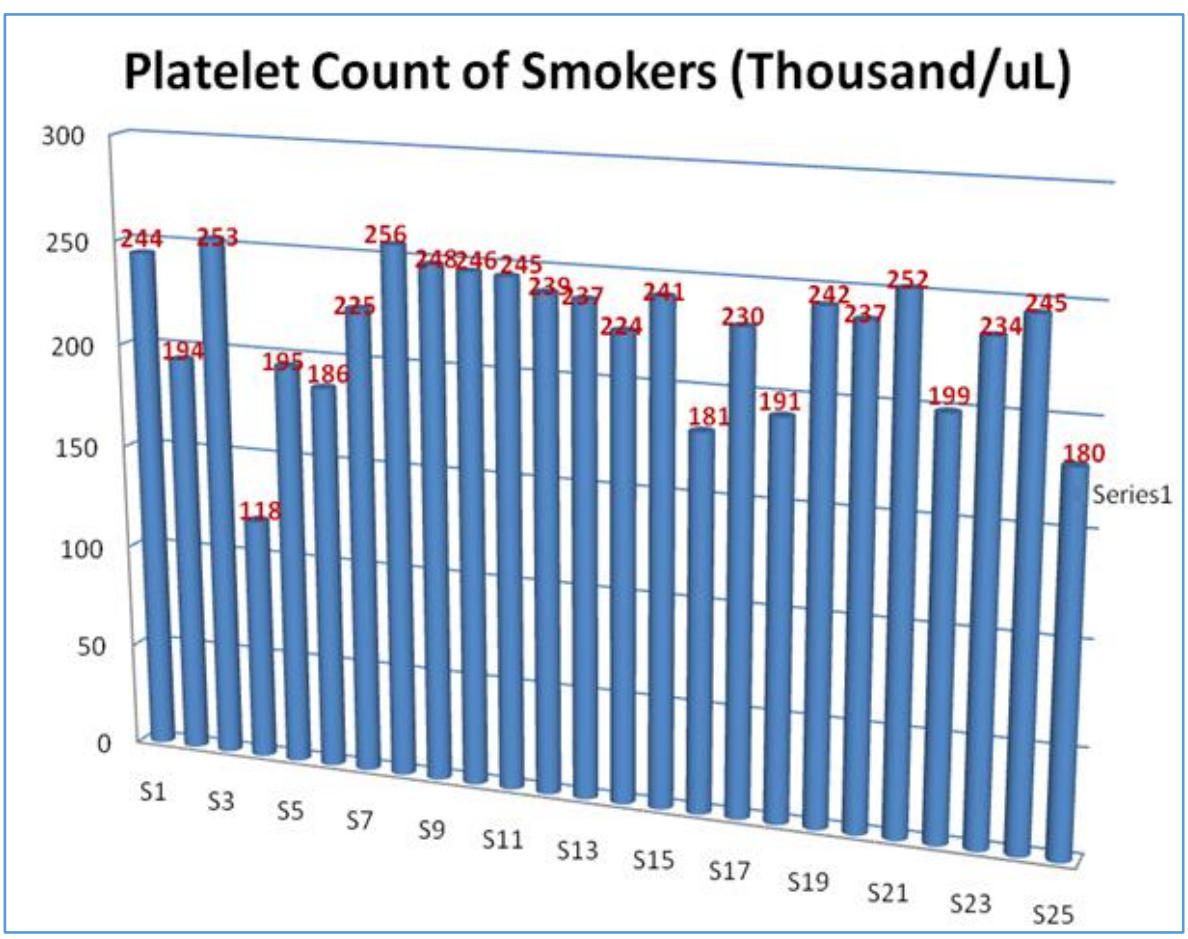

Figure 1. Graphical representation of Platelet Count of Smokers

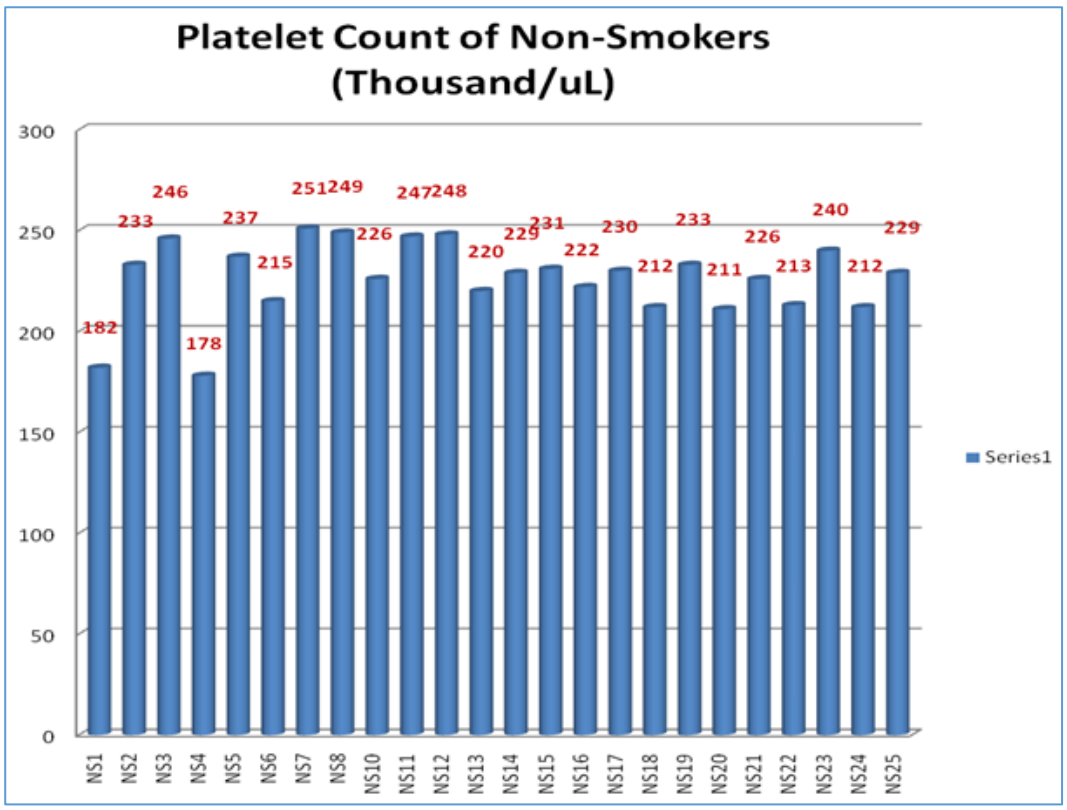

Figure 2. Graphical Representation of Platelet Count (thousand/uL) of Non-Smokers

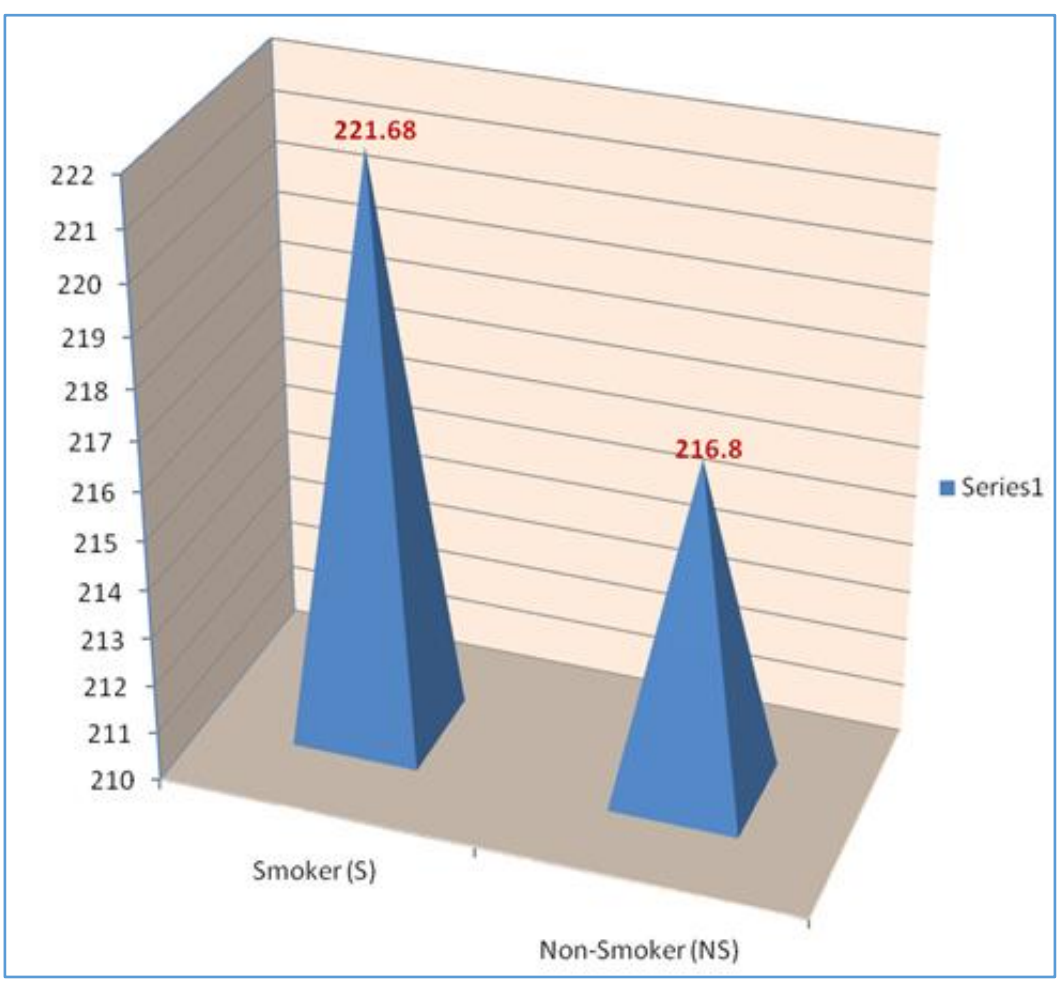

Figure 3. Graphical Representation of Mean Platelet Count (thousands/uL) of Smokers and Non-Smokers

The sample size taken for the study was of reasonable size, where N=50 with both NS (Nonsmokers) and S (Smokers) each being 25. The age group was in the range of 20 to 40. It was observed that there was no significant difference in the platelet count between the two types of subjects. However, for majority of the subjects who were smokers the platelet count was higher than that compared to non-smokers. From our theoretical knowledge as well as from a large series of studies it is indicated that undoubtedly smoke inhalation, either active (by smoking cigarettes directly) or passive (inhaling the smoke indirectly in the company of smokers) is a potential hazard for daily life and cause severe lesion to cardiovascular system.[3]

In the present study, the mean platelet count of smokers was higher than that calculated for non-smokers. The mean platelet count for smokers was 221.68 thousands/uL and for non-smokers 216.80 thousand/uL. This infers that smokers are more at risk of developing thromboembolic accidents than non-smokers.[4]

DISCUSSION

Platelets, also known as thrombocytes, are disc-shaped a-nucleate cells fragments that have shed from megakaryocytes in the bone marrow into the blood stream which circulates in the plasma of human blood under normal conditions. Platelets play a major role in haemostasis, which is the process of stopping bleeding after an injury and keeping the blood within the damaged blood vessel by the formation of a plug or blood clot and provides a surface that recruits and concentrates activated coagulation factors.

Endothelial injury allows the platelets to contact the underlying extracellular matrix. In smokers the endothelial lining is damaged which causes the platelets to adhere to the sub-endothelial collagen. Under normal conditions, when one cuts oneself, this is beneficial and prevents excessive bleeding. When activated after an injury, the platelets rush to the site of the cut and form finger-like projections which help the platelets stick together to form the plug or a scab. This helps to reduce bleeding through a process called aggregation. If injury occurs under the skin, the aggregation of platelets forms a blood clot and reduces bleeding from the blood vessel. The platelets are known to adhere at regions of endothelial damage. In smokers the endothelial lining is damaged and this causes platelets to adhere to the sub endothelial collagen therefore the number of platelets in circulation might vary. Earlier studies and research have reflected that endothelial dysfunction is an early event in atherogenesis that results in inflammation, vasoconstriction, and thrombosis.[5-6] It has been hypothesized that endothelial cell damage from inhaled cigarette smoke contributes to vascular injury, atherogenesis, and increased risk of cardiovascular diseases; smoking as few as 2 cigarettes/day doubles the number of nuclear-damaged endothelial cells.[7] Flow-mediated vasodilatation of the brachial artery is a noninvasive, validated measure that quantifies endothelial function and predicts future cardiovascular disease events.[8] In clinical and epidemiological studies, smoking-induced endothelial dysfunction seems to be dose related and may be reversible after smoking cessation.[9] However, the precise mechanisms by which smoking cessation decreases cardiovascular disease risk are not established. The impact on endothelial function due to long-term effects of continued smoking and smoking cessation have neither been established.

The objective of this study was to assess the platelet count of smokers versus non-smokers and also to stress counsel these subjects about the ills of smoking which has been rightly called the silent killer of today's adolescents.

A number of international, Asian and some Indian studies show effect of tobacco, relationship of white cell count, platelet count and haematocrit to cigarette smoking. These studies also observe the effects of smoking on cardiovascular diseases, have concluded that elevations in White Blood Cells and Platelet Count in adolescent smokers indicate that these blood components may have an early role in the pathogenesis of arteriosclerosis, whereas in some reports it was found that the plasma fibrinogen concentration and platelet count increased significantly in smokers.[7]

The effect of smoking on platelet count is still controversial. A study done to check the same was concluded that the platelet count and platelet parameters of the subjects were not significantly different between smokers and non-smokers.[10]

In another study it has also been concluded that acute smokers of 2 cigarettes in succession will activate leucocytes and cause endothelium damage but will not influence the platelet activity.[11]

In the Study of peripheral blood thrombocytosis in smokers, it has been found that the platelet count increased in smokers with history of smoking less than 5 years and platelet count was reduced in smokers of history of more than 5 years of smoking as compared to non-smokers.[12]

Study conducted in this area has shown that a majority of the individuals with idiopathic pulmonary fibrosis are smokers. Lung cancer is the most frequently diagnosed cancer in the world and the most common cause of mortality worldwide, about 29% to 87% of smokers have lung cancer. Research on Cigarette smoking and platelet aggregation has concluded that Habitual Smokers have a greater tendency to platelet aggregation than do non-habitual smokers. The long-term effects of smoking are probably more important than acute affect.[3]

This research was carried out to medically establish the ill effect of tobacco smoking to human body by studying the platelet count in smokers versus that in non-smokers. However, not much research/study has been done in this area and the threat of smoking is so great that it was a motivation to take up this study.

The primary objective of the research was to compare the blood platelet count of smokers versus non-smoker, the secondary objective was to educate the smokers in whatever little way it could be carried out in day to day life whether in hospital or in hostel and to facilitate in reducing this social evil the silent killer of today's adolescent. While conducting the research it was also realized that there is a great

hesitation amongst young smokers in getting their blood sample tested for the platelet count. A lot of convincing was required to get consent and they were informed about the test procedure and promised that their identity would not be disclosed and so on.

In the initial cause of arterial thrombosis, the blood platelets also play an important role. The difference in aggregation behaviour in platelets from smokers and non-smokers are also significant. It is well known that elevated platelet counts lead to blood clots developing in the blood vessels, which can lead to stroke, myocardial infarction. Reduced platelet count can lead to excessive bleeding. The importance of blood platelets is well established. Institutional Ethics Committee (IEC) clearance was taken prior to the beginning of the study, Informed Consent Form (ICF) of the subject was also obtained and privacy and confidentiality were strictly maintained for the subjects whose samples were taken.

Platelet Count was carried out both on smokers and non-smokers to correlate the implications of smoking. The known side effects of nicotine are multi-systemic like increase clotting tendency, bronchospasm, nausea, dyspepsia, increased gastric motility, increased heart rate arrhythmia and so on.

In the research done by Tell GS et al on Norwegian adolescent population the results were similar to our findings, that the mean platelet count for smokers was 300000 per mm^3 (300 thousand/uL) and for non-smokers 275000 per mm^3 (275 thousand/uL). Result of this study show that platelet count was increased in adolescent who started smoking relatively early. Elevation in platelet count in adolescent smokers indicate that these blood components may have an early role in the pathogenesis of arteriosclerosis.[13] Findings that subjects who smoke have elevated Platelet count are consistent with the hypothesis that inhalation of cigarette smoke causes inflammatory reactions. This has also been studied that chronic smoking causes alteration in platelet aggregation and even effects the morphological features of the platelets effecting the coagulation profile.[14-15] A plausible explanation for the higher platelet count among smokers may be due to the chemical constituents in cigarette smoke which simulates the bone marrow to increase the production of platelets.

This study revealed that there is an increase in the mean platelet count of smokers as compared to that in non-smokers. However, there is no significant difference observed in the total platelet count of smokers versus non-smokers probably because of the younger age group selected. It only confirms that to establish a concrete hypothesis further study/research should be taken up on other hematologic parameters like platelet activation, platelet-dependent thrombin generation, plasma fibrinogen and white blood cell count. To validate the results of the platelet count variation it is necessary to study large number of subjects.

In this study, subjects who had atleast three years of smoking history and who smoked about 5 to 6 cigarettes a day were also taken instead of subjects all being heavy smokers. Therefore, it established that initially smoking will not have much effect on the platelet count.

CONCLUSION

After the research which was carried out across three months on 50 subjects of different socio-economic parameters, it was concluded that mean platelet count of smokers was higher than that of non-smokers. These results are in concurrence with the other few similar studies. In the younger age group, and in the early years of smoking, there was no significant difference in the platelet count of smokers versus non-smokers. The study revealed that there was less difference in the platelet count of smokers versus non-smokers amongst subjects younger than 40 years of age. Hence it is important to promote the prevention from deleterious effects of tobacco smoking by motivating smokers to leave their habit, educating about the reversal of ill-effect after cessation of smoking. It only established that if proper counseling is organized for youth of this age group, emphasis given on yoga and such physical activities in colleges/institutes to combat the stress level, then many of the younger generation would not fall prey to the habit of smoking.

ACKNOWLEDGEMENTS

I am deeply grateful to my Indian Council of Medical Research for giving this opportunity to carry out the research project, to my guide Dr. Kusuma Venkatesh, Professor of Pathology, for her valuable guidance, suggestions and encouragement.

I am thankful to all the teachers and non-teaching staff of my medical college for providing me the best environment and my patients for their active co-operation and support towards successful completion of this research. Last but not the least I thank my family members, who have supported me throughout this study.

REFERENCES

[1] Leone A. Cardiovascular damage from smoking: a fact or belief? International Journal of Cardiology 1993;38(2):113-117.
[2] Thomas CB, Bateman JL, Lindberg EF. Observation on the individual effects of smoking on Blood pressure, platelet, stroke volume and cardiac output of healthy young adults. Ann Intern Med 1956;44(5):874-892.
[3] Glynn MF, Mustard JF, Buchanan MR, et al. Cigarette smoking and platelet aggregation. Can Med Assoc J 1966;95(11):549-553.
[4] Subratty AH, Beerbul M. Is fibrinogen a reliable haemostatic marker for monitoring possible risk of thromboembolic events in smokers? Science and Technology 1999;3:103-108.
[5] Puranik R, Celermajer DS. Smoking and endothelial function. Prog Cardiovasc Dis 2003;45(6):443-458.
[6] Deanfield JE, Halcox JP, Rabelink TJ. Endothelial function and dysfunction: testing and clinical relevance, Circulation 2007;115(10):1285-1295.

[7] Ambrose JA, Barua RS. The pathophysiology of cigarette smoking and cardiovascular disease: an update. J Am Coll Cardiol 2004;43(10):1731-1737.
[8] Corretti MC, Anderson TJ, Benjamin EJ, et al. Guidelines for the ultrasound assessment of endothelial-dependent flow-mediated vasodilation of the brachial artery: a report of the International Brachial Artery Reactivity Task Force. J Am Coll Cardiol 2002;39(2):257-265.
[9] Celermajer DS, Sorensen KE, Georgakopoulos D, et al. Cigarette smoking is associated with dose-related and potentially reversible impairment of endothelium-dependent dilation in healthy young adults. Circulation 1993;88(5 Pt 1):2149-2155.
[10] Suwansaksri J, Wiwanitkit V, Soogarun S. Effect of smoking on platelet count and platelet parameters: an observation. Clin Appl Thromb Hemost 2004;10(3):287-288.
[11] Blann AD, Kirkpatrick U, Devine C, et al. Influence of acute smoking on leucocytes, platelets and endothelium, Atherosclerosis 1998;141(1):133-139.
[12] German AK, Negreeva TA. Study of peripheral blood thrombocytosis in smokers. Lab Delo 1989;(10):23-25.
[13] Tell GS, Grimm RH, Vellar OD, et al. The relationship of white cell count, platelet count and hematocrit to cigarette smoking in adolescent: the Oslo Youth Study. Circulation 1985;72(5):971-874.
[14] Sandhya M, Satyanarayana U, Mohanty S, et al. Impact of chronic cigarette smoking on platelet aggregation and coagulation profile in apparently healthy male smokers. Int J Clin Exp Physiol 2015;2(2):128-133.
[15] Ghahremanfard F, Semnani V, Ghorbani R, et al. Effects of cigarette smoking on morphological features of platelets in healthy men. Saudi Med J 2015;36(7):847-850.