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Original research article

An analysis of how smoking affects blood tests and lung function tests

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Abstract

Background: In both high-income and low-income nations, cigarette smoking is the major cause of premature illness and death. It causes coronary heart disease, chronic obstructive pulmonary disease (in 90% of cases), and lung cancer. In order to encourage adults to quit smoking and reduce their negative impact on their hematological parameters and lung functions, this study is being conducted.

Methods: One hundred seemingly healthy adults (50 smokers) between the ages of 30 and 50 were chosen from Mamata hospital outpatients between January 2022 to December 2022. Non-instructional staff members of Mamata Medical College constituted the control group (50 non-smokers). They were asked to give their informed permission.

Results: When compared to non-smokers, smokers have impaired lung function and aberrant hematological parameters, including increased Hb, RBC count, TLC, platelet, and ESR levels. PEFR, FVC, FEV1, and the ratio of FEV1/FVC all declined at a much faster rate among smokers.

Conclusion: According to our findings, cigarette smokers had abnormalities in their hematological and respiratory tests. The length of time a person has smoked has a substantial impact on their Hb concentration and other metrics. There were alterations in pulmonary functions indicative of peripheral airway narrowing among smokers, and these deteriorated with continuing smoking.

Keywords: Spirometry, pulmonary functions, and hematological parameters.

Introduction

It has long been agreed that cigarette smoking is responsible for more premature deaths and illnesses than any other factor combined. Some people light up to relieve stress, while others light up for pleasure or social reinforcement. Many young people start smoking for emotional reasons, such curiosity, rebellion, or a need to prove their independence from their parents. Nicotine's pharmacological effects play a significant role in the maintenance of the habit after it has established itself [1-3].

One-third of the world's 1.1 billion smokers are young adults, according to the World Health Organization (WHO). The vast majority of these smokers are men living in underdeveloped nations. If the current rate of increase in tobacco-related deaths holds, that amount will triple to 10 million yearly by 2020. Smoking tobacco is widely acceptable by Indian culture. There has been a significant shift in the types and practices of tobacco smoking in contemporary Indian society. The World Health Organization estimates that 194 million men and 45 million women in India regularly use tobacco products [4-6].

It is the single most major preventable cause of heart disease, COPD, high blood pressure, and cancers of the nasopharynx, bronchus, and other respiratory tract. It has been calculated that the average smoker loses 7 minutes of life for every cigarette they light up. Smoking reduces life expectancy by an average of 8 years for someone who starts when they're 15 years old, and by an average of 4 years for someone who starts when they're 25 years old. 1. The majority of the increased mortality caused by cigarette smoking can be attributed to coronary heart disease, cancer, and other respiratory disorders [7, 8]. Smokers have an average 16 times higher chance of developing lung cancer, a 12 times higher risk of developing chronic obstructive pulmonary disease, and a 2 times higher risk of developing a myocardial infarction. Many studies conducted since the 1950s have established a causal relationship between cigarette smoking and a variety of adverse health outcomes, including peripheral vascular disease and stroke. Although researchers discovered a correlation between smoking and lung ailments as early as the 1870s, it wasn't until 1964 that the United States Surgeon General issued a report warning of the link between smoking and emphysema [8-10].

Airflow blockage is more common in smokers across all populations with relevant prevalence data. When accounting for the effects of age and beginning forced expiratory volume in one second, cigarette smoking is the only statistically significant predictor of air flow blockage in most multivariate analyses

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(FEV1) [9-11]. Cigarette smoking is responsible for between 80% and 90% of cases of chronic obstructive pulmonary disease in the United States. A patient's breathing pattern, the root of wheezing, and the diagnosis of lung illness can all be gleaned from an evaluation of their pulmonary function. There is a battery of diagnostic procedures known as pulmonary function tests that have been employed to evaluate lung capacity. Ventilation, the connection between ventilation and perfusion, diffusion/gas exchange, and pulmonary circulation are all studied using different types of PFTs [12-14].

The most common test for assessing lung function is spirometry. The diagnostic procedure is equally applicable to the outpatient, in-office, or hospital scenario. A spirogram, also known as a volume time tracing or a flow volume tracing, is a graphical representation of airflow. The values obtained from even the most basic spirogram can tell you a lot about the mechanical qualities of the lungs, such as air flow, and can help you figure out if you have an obstructive or restrictive pulmonary condition [13-15].

Materials and Methods

Among the outpatient population at Mamata Hospital, one hundred adults in apparent good health (fifty smokers) were selected. Members of Mamata Medical College's non-instructional staff served as the study's control group (50 non-smokers). Their consent, after being fully informed, was sought from January 2022 to December 2022.

Cigarette smoking was investigated for its impact on hematological and pulmonary function tests in men aged 30 to 50 years old. The amount of cigarettes smoked daily and the number of years of smoking are also taken into account to determine the dosage response relationship.

Inclusion criteria

1. 30 to 50 years old
2. Men's gender
3. Smoking duration: 10 years and longer
4. Smoking more than 10 cigarettes per day

Exclusion criteria

1. Age: below 30 and above 50 years
2. A female.
3. Smoking history: 10 years or less
4. Smoking frequency: 10 cigarettes or less each day
5. High Blood Pressure
6. H/O Type 2 Diabetes

Spirometry

The first study a doctor should consider using is this one unless there are special circumstances. When instructed by a medical professional, the vast majority of patients find spirometry to be a simple task. The diagnostic procedure is equally applicable to the outpatient, in-office, or hospital scenario. Spirometry can be used for a wide variety of reasons. It has use in epidemiological and other types of research, as well as in the diagnosis and monitoring of respiratory symptoms and disease.

Statistical Analysis

Standard error numbers and the mean standard deviation were provided. Student’s t-test was used to compare two groups, and one-way ANOVA was used to compare more than two groups.

Results

The study involved 100 people, 50 smokers and 50 non-smokers. Fifty male smokers were subjected to hematological and pulmonary function tests. The outcomes depend on the analysis of these. The data was summarized using a Mean + SD format.

Table 1: Comparison of Smokers vs Nonsmokers by Age

Status of smoking	Number	Mean (completed years)	Std. Deviation	Std. Error	P value
Smokers	50	43.62	9.725	1.375	0.613
Non-smokers	50	42.78	6.544	0.926	0.613

The average age of the 50 smokers in the study was 43.62 9.725 (see Table 1), whereas the average age of the 50 nonsmokers was 42.78 6.544 (also see Table 1), which was not statistically different.

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Table 2: Smokers and non-smokers Hb levels

Status of smoking	Number	Mean (gms/dl)	Std. Deviation	Std. Error	P value
Smokers	50	14.508	1.5592	0.2205	0.000
Non-smokers	50	12.134	0.7041	0.0996	0.000

Hemoglobin levels were 14.508 1.5592 in smokers and 12.134 0.7041 in nonsmokers, as shown in Table 2. Smokers' Hb levels were much higher than those of nonsmokers.

Table 3: Red blood cell count in smokers versus nonsmokers

Status of smoking	Number	Mean (millions/mm^3)	Std. Deviation	Std. Error	P value
Smokers	50	5.255	0.6829	0.0938	0.000
Non smokers	50	4.634	0.5330	0.0754	0.000

The RBC count in smokers is 5.255 0.6629, while in nonsmokers it is 4.634 0.5530, as seen in Table 3. Smokers had a higher mean RBC count than nonsmokers by a statistically significant margin.

Table 4: Kind treatment for smokers and nonsmokers alike

Status of smoking	Number	Mean /mm^3	Std. Deviation	Std. Error	P value
Smokers	50	8050	1879.562	265.810	0.000
Non-smokers	50	6858	1245.414	176.128	0.000

TLC is estimated to be 8050 1879.562 in smokers and 6858 1245.414 in never-smokers, as shown in Table 4. When comparing smokers and nonsmokers, there was a statistically significant rise in TLC.

Table 5: Comparison between Smokers vs Nonsmokers for Platelet Count

Status of smoking	Number	Mean Lakhs/mm^3	Std. Deviation	Std. Error	P value
Smokers	50	255760.00	61835.054	8744.797	0.000
Non-smokers	50	216580.00	35575.209	5031.094	0.000

Platelet counts in smokers are 255760 61835.054 while in nonsmokers they are 216580 35575.209, as seen in Table 5. Platelet counts were significantly higher in smokers than in nonsmokers.

Table 6: Comparing ESR readings between smokers and nonsmokers

Status of smoking	Number	Mean (mm/hr)	Std. Deviation	Std. Error	P value
Smokers	50	11.74	10.778	1.524	0.006
Non- smokers	50	7.38	1.602	0.227	0.006

According to Table 6, the mean ESR for smokers is 11.74 10.778 and the mean for nonsmokers is 7.38 1.602. The ESR value was significantly higher in smokers than in nonsmokers.

Table 7: Length of smoking's impact on hemoglobin

Status of smoking	Number	Mean (gm/dl)	Std. Deviation	Std. Error	P value
>20 years	19	15.137	1.7179	0.3941	0.024
<20 years	31	14.123	1.3401	0.2407	0.024

Hemoglobin values ranged from 15.137 1.7179 in long-term smokers to 14.123 1.3401 in recent smokers, as seen in Table 7. Hb was found to rise in correlation with the number of cigarettes smoked each day. The Hb levels in the group of people older than 20 years old rose significantly (P 0.05).

Table 8: Length of smoking's impact on blood cell count

Status of smoking	Number	Mean (millions/mm^3)	Std. Deviation	Std. Error	P value
>20 years	19	5.310	0.6589	0.1512	0.650
<20 years	31	5.221	0.6740	0.1210	0.650

According to Table 8, the RBC count of long-term smokers was 5.310 0.6589, while that of recent smokers was 5.221 0.6740. It was shown that the red blood cell count rose in tandem with the number of years spent smoking. Yet, this finding failed to pass statistical muster.

Table 9: The Long-Term Consequences of Smoking

Status of smoking	Number	Mean/mm^3	Std. Deviation	Std. Error	P value
>20years	19	8131.58	1693.469	388.508	0.813
<20years	31	8000.00	2010.638	361.121	0.813

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In Table 9, we can see that the TLC was 8131.58 1693.469 in long-term smokers and 8000 2010.638 among recent smokers. TLC was shown to rise in tandem with cumulative smoking time. Yet, this finding failed to pass statistical muster.

Table 10: Platelet count and smoking duration

Status of smoking	Number	Mean (lakhs/mm^3)	Std. Deviation	Std. Error	P value
>20years	19	238210.53	73473.789	16856.043	0.117
<20years	31	266516.13	51879.907	9317.906	0.117

Platelet counts ranged from 238210.53 73473.789 in long-term smokers to 266516.13 51879.907 in recent smokers, as shown in Table-10. Platelet count was found to decline with increasing time spent smoking. Yet, this finding failed to pass statistical muster.

Discussion

Tobacco usage is a leading cause of preventable death in many countries. Deaths and long-term impairments are disproportionately caused by tobacco-related respiratory illnesses and cardiovascular diseases. Numerous studies have established a connection between cigarette smoking and a variety of adverse health outcomes, including peripheral vascular disease and stroke. A patient's breathing pattern, the root of breathlessness, and the presence of lung illness can all be ascertained using a battery of diagnostic procedures designed to evaluate pulmonary function. So, the purpose of our study entitled "Impact of cigarette smoking on haematological parameters and pulmonary function tests" was to investigate the impact that smoking has on a person's blood and lung functions [14-16].

Both the RBC count and the Hb concentration were found to be higher in smokers in our study. The carbon monoxide in cigarettes could be to blame for this. Smokers are more likely to have carboxy-hemoglobin, which prevents the body from properly utilizing oxygen. Tissue hypoxia is caused by smoking because oxygen delivery is decreased. Interstitial cells in the peritubular capillary bed of the kidney and perivenous hepatocytes of the liver respond strongly to this hypoxia, releasing erythropoietin. Erythropoietin stimulates the proliferation of erythropoietin-sensitive bone marrow stem cells, which thereafter differentiate into progenitor cells for mature erythrocytes. It also stimulates -amino Levulinic acid synthetase, which is necessary for the production of hemoglobin, and increases globin synthesis. As a result, both the RBC count and the Hb concentration rose [15-17].

Our findings are consistent with those of studies by Sagone A.L. Jr. *et al*., R.D. Forrest *et al*., D. Nordenberg *et al*., and Whitehead T.P. *et al*. on RBC count and Hb concentration. Our research shows that smokers have a significantly higher total leucocyte count (TLC). It is unclear how exactly cigarette smoking causes an increase in TLC. Many explanations have been proposed to explain this phenomenon in smokers. Tobacco smoke is toxic because it contains acrolein, nicotine, and acetaldehyde, a type of formaldehyde created by combustion. Cigarette smoke also alters the structure of the respiratory system. There is inflammation and fibrosis around the bronchioles, the mucosa becomes more permeable, mucociliary clearance is impaired, pathogen adherence is altered, and the respiratory epithelium is disrupted. Such alterations increase susceptibility to upper and lower respiratory tract infections, which in turn may enhance the inflammatory response of the lungs to cigarette smoke. Colony stimulating factors like granulocyte-monocyte colony stimulating factor help stimulate the bone marrow, which then releases leucocytes into the blood as part of the systemic inflammatory response (GM-CSF). This may cause smokers' total leucocyte counts to rise [17-19].

Smoke contains a number of chemicals, including free radicals and phenol-rich glycoproteins, that may stimulate the production of inflammatory cytokines such tumor necrosis factor alpha (TNF-), interleukin-1 (IL-1), and interleukin-6 (IL-6) in macrophages [20]. The overall number of leucocytes in the blood of smokers is higher because of the work of Van Eeden and coworkers, who discovered that the cytokine IL-6 strongly encourages bone marrow to release leucocytes. Anwar M. Al-Awadhi, Margit Frohlich *et al*., A. B. Bridges *et al*., YoshitoMomose *et al*., and our own study all found similar things about TLC [21, 22].

Nicotine increases leucocyte count by a roundabout mechanism - it causes the production of the stress hormones cortisol and catecholamines. Our research shows that smokers tend to have higher platelet counts. Some research suggests that smoking raises women's platelet counts, whereas other research finds no association between smoking and platelet counts. Platelets were released from the bone marrow after cigarette smoke produced inflammation in the lungs.

Chronic smoking appears to have a negative effect on platelet activity and survival. Prostacyclin is inhibited and thromboxane A2 production is increased due to smoking's acute and chronic suppression of Cyclo-oxygenase. Vasoconstrictor and platelet agonist thromboxane A2 is extremely powerful. Smokers may have a greater platelet count because of this. Our findings are consistent with those of a research on platelet count by G.S. Tell *et al*. [21-23].

Our results show that ESR levels are higher in smokers. This is because the ESR is mostly determined by

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the size or mass of the particles (rouleaux) that are falling. Plasma fibrinogen concentration is raised in smokers, which is a factor in rouleaux production and size. Red cells are negatively charged, thus they normally reject one another and stay at distinct locations. Fibrinogen prevents the modifications on the red cells from taking effect and makes the red cells sticky, which in turn promotes rouleaux formation and the ESR. Our results are consistent with those of Adekunle A. Famodu *et al*. and Mehrun Nisa *et al*., who also studied ESR. Our research shows that people who have smoked for more than 20 years have higher levels of hemoglobin, red blood cells, white blood cells, and eosinophil cationic protein (ESR). There is a discernible spike in hemoglobin levels. In our research, we found that people who have smoked for more than 20 years have a statistically negligible drop in platelet count. Smoking's influence on platelet count is controversially reported in the literature. Brummit *et al*. discovered no connection between smoking and platelet count in healthy participants. Similarly, Dotevall *et al*. and Suwan saksri *et al*. found no differences in platelet count between smokers and never-smokers for either gender [22-24].

We found evidence of a statistically significant drop in PEFR. The PEFR has also been proven to decline with both the length of time that a person smokes and the average amount of cigarettes they smoke daily. This is because smoking raises airway resistance and lowers elastic recoil pressure in the lungs, both of which are detrimental to healthy breathing. These results were consistent with those found by Prasad BK *et al*. and Nancy *et al*. [23-25].

When we compared smokers and nonsmokers, we found that smokers had significantly lower FVC. It has also been demonstrated that both the length of smoking and the average daily amount of cigarettes smoked contribute to a greater decline in FVC. As FVC reflects lung volume, its decline reflects a possible secondary cause in our study. Several studies, including those by Nancy N R *et al*., Danuser B *et al*., Miller A, *et al*., and Mhase VT *et al*., reported similar results [24-26].

We found that smokers' FEV1 levels were significantly lower than those of nonsmokers'. More time spent smoking and more cigarettes smoked daily were both associated with a steeper decline in FEV1. In our study, we found that the more time spent smoking and the more cigarettes consumed daily, the more significant the decline in lung function metrics was. Miller A. *et al*., Gold RD. *et al*., Apostol G. *et al*., and Isobel U. *et al*. all came to similar conclusions [27]. It was demonstrated that the effect was highly sensitive to the intensity of exposure, which included both the length of time and the total number of cigarettes smoked. Accumulation of inflammatory exudates, excessive mucus secretion, changes in surface tension qualities, and alterations in smooth muscle tone are all potential causes of the aforementioned impacts on the airways. Mediators released from cells already in or transported to the airway may also play a role in these alterations. These alterations are gradual with continuing smoking, suggesting that some percentage of current smokers may go on to acquire COPD. The human body has incredible reserves for dealing with adversity. Disability does not appear until a particular level of impairment has been reached. Many studies, including ones by Tashkin DP *et al*., Camilli AE *et al*., Dockery DW *et al*., and Gorecka *et al*., have demonstrated that giving up smoking can increase lung capacity. So, when people stop smoking, the inflammatory changes in their tiny airways often go away as well [26-28].

Conclusion

The results of this study show that compared to nonsmokers, both the RBC count and Hb concentration are much higher in smokers. Among smokers, there is a statistically significant rise in total leucocyte count. When compared to the general population, smokers have a significantly higher platelet count. Smokers show alterations in pulmonary function consistent with peripheral airway narrowing, and these effects intensify with continuous smoking, according to this study, which sheds light on the functional abnormalities of the lung caused by cigarette smoking even in the absence of overt disease. Thus, it is recommended to regularly check these indicators in smokers in order to detect changes at an earlier stage for the purpose of implementing preventative actions such as quitting smoking. Public spaces should be smoke-free, and efforts should be made to educate the people about the risks of smoking and pass laws prohibiting it.

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None

Conflict of Interest

None

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