

# Cigarette smoking and its effect on coagulation profile, hematological parameters, and oxygen saturation in healthy blood donor



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## ABSTRACT

**Background:** Cigarette smoking causes millions of deaths all around the world each year as it is one of the leading causes of cancer and cardiovascular diseases. **Aims and Objectives:** The study aimed to study the effect of cigarette smoking on coagulation profile, hematological parameters, and oxygen saturation in healthy blood donors. **Materials and Methods:** A case-control study was carried out at the Department of Pathology, Era's Lucknow Medical College which is a tertiary care center, and took place between September 2018 and September 2020 (24 months). **Results:** Cigarette smoking has adverse effects on hematological parameters (red blood cell count, hemoglobin, hematocrit, mean corpuscular hemoglobin, red cell distribution width, and total leukocyte count) and coagulation profile (prothrombin time, activated partial thromboplastin time, and international normalized ratio). In our study, no adverse effects are seen on oxygen saturation though. **Conclusion:** Our study shows that hematological parameters and coagulation profile were altered in donors who were smokers and thus, it should be considered during blood donation. Although oxygen saturation was not adversely affected, this study strongly recommends the need for further studies to be done on this parameter of donated blood.

**Key words:** Blood donation; Coagulation profile; Hematological parameters; SpO<sub>2</sub> saturation

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## INTRODUCTION

The WHO has estimated that there are around thirteen hundred million smokers out of which nearly 75% are in developing countries. It has been prognosticated that if the same scenario of smoking continues, there might be ten million tobacco-related deaths globally per year and around seven million will take place in developing countries after year 2020. About 4000 substances in tobacco are identified and all of them have toxic effects of a certain degree.<sup>1</sup>

Several studies have shown that smoking has adverse effects on the health of humans and has proved to be a cause for the development of various pathologies and diseases like chronic

obstructive pulmonary disease,<sup>2</sup> various cancers,<sup>3</sup> pancreatitis,<sup>4</sup> gastrointestinal disorders,<sup>5</sup> periodontal disease,<sup>6</sup> metabolic syndrome<sup>7</sup>, and also some autoimmune diseases.<sup>8</sup> Cigarette smoking is also associated with an increased risk for cardiac diseases such as coronary artery disease,<sup>9</sup> ischemic heart disease,<sup>10</sup> myocardial infarction, atherosclerosis<sup>11</sup>, and stroke.<sup>12</sup>

An extremely complex interaction of cellular events in arterial walls, mainly endothelial cells, and platelets (PLTs) is involved that balance between clotting and dissolution of clots.<sup>13</sup>

Cigarette smoke hinders hemostasis by various pathways by altering the functions of PLTs, endothelial cells, and coagulation factors.<sup>14</sup>

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Predictors such as age, number of cigarette sticks smoked per day, and duration of smoking are well-established factors for analyzing the risk of developing complications that are smoke-related in chronic smokers.<sup>15</sup> Various studies have shown that smokers have higher total leukocyte counts (TLCs) than non-smokers.<sup>16-18</sup>

The exact mechanisms of occurrence of these disorders in smokers are not known, but it is presumed that these effects are caused by abnormalities in the blood rheology, infection and inflammation, oxidative stress, and alterations of the antithrombotic and fibrinolysis system.<sup>19</sup> The damage caused by smoking cigarettes is affected by the number of cigarettes that are smoked.<sup>20</sup>

Some studies have found that TLC increases as a number of sticks of cigarettes smoked daily increases and decreases after the stoppage of smoking.<sup>21</sup>

Cigarette smokers show an increase in many hematological variables, including hemoglobin (Hb) and blood cell indices. It also increases the TLC, hematocrit (HCT), plasma viscosity (PV), and whole blood viscosity (WBV). It decreases the international normalized ratio (INR). This decreased INR was related to increased PV, WBV, and HCT.<sup>22</sup>

Cigarette smoking has an adverse effect on coagulation profiles which leads to abnormalities in circulation, that is, the ability of blood to coagulate increases which can lead to obstructive clotting called thrombosis.<sup>23</sup>

Cigarette smoke contains various harmful gases such as carbon monoxide which reduces SpO<sub>2</sub> as it decreases the ability of hemoglobin to deliver oxygen to the tissues since carbon monoxide has a high affinity toward red blood cell (RBC) when it reaches lungs (250 times more compared to oxygen). SpO<sub>2</sub> must be maintained at >95%. When it decreases, it may lead to changes in decision-making ability, attention, and memory.<sup>24</sup>

Smoking accelerates the rate of decline of lung function in adulthood. Any compromise of lung function will reduce oxygen saturation which is easily measured by pulse oximetry.<sup>25</sup>

In these days, pulse oximetry is a reliable, simple technique that is used to analyze oxygen saturation. If values are <93% it means that O<sub>2</sub> therapy might be necessary and may require closer monitoring of that person.<sup>26</sup>

The first blood bank in the world was set up in Madrid in 1936, during the Spanish Civil War. In India, the first blood bank was set up in 1942 in Kolkata to meet war needs. It was later shifted to Kolkata Medical College in

1945, where it started functioning as the first civilian blood bank in the country.<sup>27</sup>

Until the early 1980s blood transfusion in India was largely whole blood, but the introduction of plastic blood bags in the country began to transform the blood processing techniques.<sup>28</sup> Although various methodological approaches to analyze the effect of donor history questionnaires can be done but many questions of donor history have been implemented as precautionary measures which has no evidence of effectiveness.<sup>29</sup>

Blood donations are routinely tested for viral markers, that is, (human immunodeficiency virus/hepatitis B surface antigen/hepatitis C virus), malaria, and syphilis. However, the only routinely measured hematological parameter is the hemoglobin level. The effectiveness of transfusion therapy depends on the quality of blood and blood products such as red cells, granulocytes, or PLT concentrates, which in turn depend on the values of all hematological parameters. The effect of cigarette smoking on the reduction of coagulation factors from whole blood may increase prothrombin time (PT), activated partial thromboplastin time (APTT), and ultimately affect the quality of fresh frozen plasma that is produced from the donor's blood.<sup>30</sup>

Blood donors are groups of individuals who differ demographically and genetically. This difference in the characteristics of the donor may alter recipient outcomes related to transfused blood components.<sup>31</sup>

Since blood donors are part of the general population, from young to old subjects, most of them will be inevitably smokers. Till now there is very little information regarding how the smoking habit of blood donors could affect blood transfusion products.<sup>32</sup>

Behaviors such as cigarette smoking and alcohol use, other legal drug use, or illicit drugs are not assessed by laboratory testing or history. Although regulatory standards are developing to reduce risks of blood transfusion by infection but the potential effect of donor habit, behaviors, and drug use on the quality of blood components donated has not been previously investigated.<sup>33</sup>

### Aims and objectives

The study aimed to study the effect of cigarette smoking on coagulation profile, hematological parameters, and oxygen saturation in healthy blood donors.

## MATERIALS AND METHODS

### Study design

This was a case-control study.

**Table 1: Distribution of mean of hematological profile**

Attributes	Smoker				Non-smoker			
	Mean	SD	Min.	Max.	Mean	SD	Min.	Max.
Age	29.02	7.924	18	55	29.57	8.428	18	56
Hb (g/dL)	16.13	0.96	14.20	18.60	13.56	0.68	12.10	16.40
TLC (10 <sup>3</sup> /μL)	8.80	2.23	4.01	15.87	6.63	2.01	3.04	12.91
RBC (10 <sup>6</sup> /μL)	5.07	0.56	3.70	6.88	4.17	0.56	3.05	5.80
HCT (%)	45.03	3.42	33.80	54.90	37.05	3.78	28.60	47.10
MCV (fL)	89.38	7.15	66.03	114.05	89.62	9.12	69.45	128.52
MCH (pg)	32.08	2.95	25.16	44.86	32.99	3.85	24.69	45.25
MCHC (g/dL)	35.90	1.76	32.23	47.04	36.88	3.07	31.96	46.53
RDW (%)	15.26	1.29	12.20	18.90	13.65	0.89	11.20	18.90
PLT (10 <sup>3</sup> /μL)	228.19	66.56	145.00	442.00	230.61	73.85	120.00	472.00

Hb: Hemoglobin, TLC: Total leukocyte count, RBC: Red blood cell, HCT: Hematocrit, MCV: Mean corpuscular volume, MCH: Mean corpuscular hemoglobin, MCHC: Mean corpuscular hemoglobin concentration, RDW: Red cell distribution width, PLT: Platelet

**Table 2: Distribution of mean of peripheral capillary oxygen saturation**

Attributes	Smoker				Non-Smoker			
	Mean	SD	Min.	Max.	Mean	SD	Min.	Max.
SpO <sub>2</sub> (%)	96.97	0.99	95.00	99.00	97.16	1.07	95.00	100.00

**Table 3: Significance of hematological parameters and coagulation profile by t-test of smokers and non-smokers**

Attributes	Status	n	Mean	Standard Deviation	t-test for equality of means	Significance (P-value) (2-tailed)
TLC (10 <sup>3</sup> /μL)	Smoker	150	8.8043	2.22881	8.867	0.000
	Non-smoker	150	6.6300	2.01292		
	Total	300				
RBC (10 <sup>6</sup> /μL)	Smoker	150	5.0689	0.55569	13.950	0.000
	Non-smoker	150	4.1690	0.56160		
	Total	300				
HCT (%)	Smoker	150	45.025	3.4173	19.174	0.000
	Non-smoker	150	37.045	3.7819		
	Total	300				
MCH (pg)	Smoker	150	32.99	3.846	-2.303	0.022
	Non-smoker	150	32.018	2.949		
	Total	300				
APTT (s)	Smoker	150	27.019	3.3850	5.354	0.000
	Non-smoker	150	25.128	2.6944		
	Total	300				

TLC: Total leukocyte count, RBC: Red blood cell, HCT: Hematocrit, MCH: Mean corpuscular hemoglobin, APTT: Activated partial thromboplastin time

The study was carried out at the Department of Pathology, Era’s Lucknow Medical College which is a tertiary care center with state of art infrastructure catering primarily to socioeconomically underprivileged suburban and rural populations of Lucknow and nearby areas. The study took place between September 2018 and September 2020 (24 months).

**Sampling**

After obtaining approval and clearance from the Institution Ethical Committee, donors were included in the study.

a. Sampling population: All the male blood donors who were eligible as per SBTC/American Association of Blood Bank (AABB) guideline donor screening protocol

b. First, we took a detailed history of the donor based on which donors were classified as

- Cases: Donors with a history of smoking (>1 pack year)
- Controls: Non-smoker donors (<1 pack year).

Number of pack-year = (number of cigarettes smoked per day/20) × number of years smoked (1 pack has 20 cigarettes).<sup>21</sup>

**Inclusion criteria**

All male blood donors who are eligible as per National and State Blood Transfusion Council/AABB guideline donor screening protocol were included in the study.

**Table 4: Significance of hematological parameters by Mann–Whitney U test of smokers and non-smokers**

Attributes	Type of College	n	Mean Rank	Sum of Ranks	Mann-Whitney U	Asymptotic significance (2-tailed)
Hb (g/dL)	Smoker	150	224.23	33635.00	190.000	0.000
	Non-smoker	150	76.77	11515.00		
	Total	300				
MCV (fL)	Smoker	150	151.43	22714.00	11111.000	0.853
	Non-smoker	150	149.57	22436.00		
	Total	300				
MCHC (g/dL)	Smoker	150	141.30	21195.00	9870.000	0.066
	Non-smoker	150	159.70	23955.00		
	Total	300				
RDW (%)	Smoker	150	207.22	31082.50	2742.500	0.000
	Non-smoker	150	93.78	14067.50		
	Total	300				
PLT (10 <sup>3</sup> /μL)	Smoker	150	151.28	22692.50	11132.500	0.876
	Non-smoker	150	149.72	22457.50		
	Total	300				
PT (s)	Smoker	150	216.22	32433.00	1392.000	0.000
	Non-smoker	150	84.78	12717.00		
	Total	300				
INR	Smoker	150	215.70	32355.00	1470.000	0.000
	Non-smoker	150	85.30	12795.00		
	Total	300				
SpO <sub>2</sub> (%)	Smoker	150	142.20	21330.50	10005.500	0.084
	Non-smoker	150	158.80	23819.50		
	Total	300				

Hb: Hemoglobin, MCV: Mean corpuscular volume, MCHC: Mean corpuscular hemoglobin concentration, RDW: Red cell distribution width, PLT: Platelet, PT: Prothrombin time, INR: International normalized ratio

### Exclusion criteria

Donors who smoked cigars, shisha (hookah), bidi, or any other form of tobacco except cigarettes were excluded from the study group.

### Randomization

Our study does not require randomization.

### Study procedure

All participants were informed about the aim of the research and contents of that. The subjects gave written consent for participating in the study.

About 4 mL of whole blood was collected out of which 2.0 mL was dispensed into an EDTA vacutainer for hematological parameters and 1.8 mL was added to 0.2 mL of 3.2% trisodium citrate vacutainer giving blood to an anticoagulant ratio of 9:1 for coagulation studies.

The following parameters were considered – Hb and HCT, total RBC count, TLC and PLT count along with RBC indices – mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), MCH concentration (MCHC), and red cell distribution width (RDW).

### SpO<sub>2</sub> values measurement

Peripheral capillary oxygen saturation measurement was done with a pulse oximeter. After the subjects sat for

30 min, oxygen saturation was measured from the hand, and the pulse source of pulse oximetry was placed on the fingertip of the hand.

## RESULTS

The present study was conducted to study the effect of cigarette smoking on the coagulation profile, hematological parameters, and peripheral capillary oxygen saturation of healthy blood donors.

The age distribution of smoker and non-smoker donors has been shown in the following two figures through pie chart (Figures 1 and 2).

The mean value of Hb, TLC, RBC, HCT, MCHC, and RDW was higher in smokers who were donors compared to non-smoker donors (Table 1 and Figures 3-7).

The mean value of PT, APTT, and INR was higher in smokers who were donors compared to non-smoker donors (Figures 8-10).

The mean value of SpO<sub>2</sub> was higher in smokers who were donors compared to non-smoker donors (Table 2).

Significant P-value was observed in the case of TLC

**Table 5: Effect of smoking on red blood cell parameters in contemporary studies from various countries and its comparison with the present study**

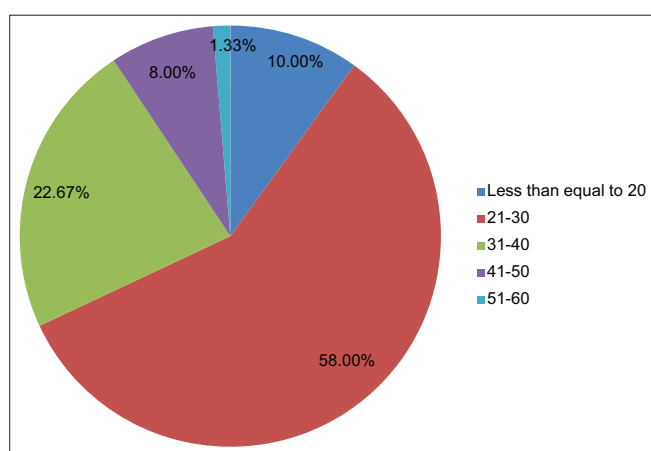
S. No.	Author (year), Place	Sample size	Mean Hb (g/dL)		Mean RBC (10 <sup>6</sup> /µl)		Mean HCT (%)		Mean MCV (fL)		Mean MCH (pg)		Mean MCHC (g/dl)		Mean RDW (%)	
			S	NS	S	NS	S	NS	S	NS	S	NS	S	NS	S	NS
1	Asif et al. <sup>34</sup> (2013), Pakistan	142 (S=71, NS=71)	16.01	14.71	5.38	5.06	49.67	45.34	90.26	88.61	28.19	28.84	31.50	32.56	13.28	12.83
2	Almarshad and Hassan22 (2014), Saudi Arabia	321 (S=195, NS=126)	15.23	14.0	5.37	5.35	52.13	50.5	92.23	91.8	26.93	26.3	29.93	29.1	14.2	14.35
3	Khan et al. <sup>35</sup> (2014), Pakistan	100 (S=50, NS=50)	16.11	14.57	5.40	5.09	48.0	44.28	88.93	87.18	30	28.87	33.57	33.83	NA	NA
4	Bilto39 (2015), Jordan	606 (S=302, NS=304)	15.01	14.21	4.65	4.62	41.29	39.68	88.87	85.93	32.24	30.76	36.32	35.70	13.34	13.39
5	Okafor and Okoroiwu36 (2017), Southern Nigeria	260 (S=150, NS=110)	15.30	13.88	5.20	4.99	44.53	44.55	86.06	88.53	29.52	28.16	33.91	31.83	14.81	14.58
6	Malenica et al. <sup>19</sup> (2017), Bosnia and Herzegovina	156 (S=56, NS=100)	14.7	13.9	4.88	4.88	41.65	40.63	88.50	84.00	29.82	28.70	33.39	33.71	14.10	14.00
7	Present study (2020), Lucknow	300 (S=150, NS=150)	16.13	13.56	5.07	4.17	45.03	37.05	89.38	89.62	32.08	32.99	35.90	36.88	15.26	13.65

Hb: Hemoglobin, RBC: Red blood cell, HCT: Hematocrit, MCV: Mean corpuscular volume, MCH: Mean corpuscular hemoglobin, MCHC: Mean corpuscular hemoglobin concentration, RDW: Red cell distribution width

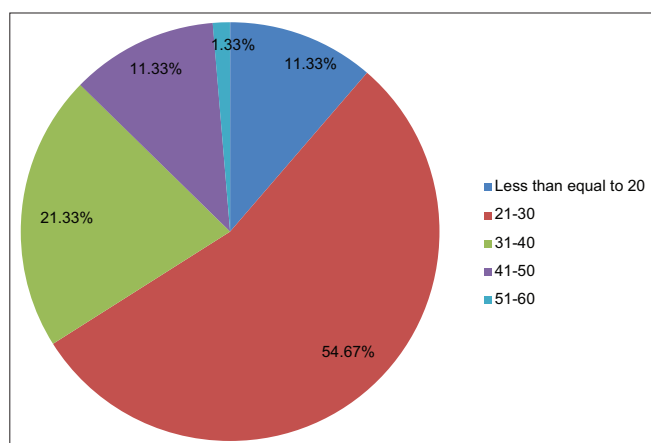
**Table 6: Effect of smoking on coagulation profile in contemporary study from various countries and its comparison with the present study**

S. No.	Author (year), Place	Sample size	Mean PT (s)		Mean APTT (s)		Mean INR	
			S	NS	S	NS	S	NS
1.	Almarshad and Hassan <sup>22</sup> (2014), Saudi Arabia	321 (S=195, NS=126)	13.53	14.05	31.68	31.20	1.14	1.37
2.	Soronnadi et al. <sup>23</sup> (2015), South-East Nigeria	200 (S=100, NS=100)	9.8	12.7	27.0	33.04	NA	NA
3.	Sivagangailakshmi and Rajkumar <sup>1</sup> (2017), Tamil Nadu, India	100 (S=50, NS=50)	23.81	18.67	45.62	39.53	NA	NA
4.	Elkhalifa <sup>40</sup> (2018), Sudan	200 (S=100, NS=100)	12.9	13.7	30.5	37.9	0.95	1.01
5.	Present study, (2020), Lucknow, India	300 (S=150, NS=150)	12.10	10.65	27.2	25.13	1.06	0.93

PT: Prothrombin time, APTT: Activated partial thromboplastin time, INR: International normalized ratio



**Figure 1:** Pie- chart showing distribution of age of donors with history of smoking



**Figure 2:** Pie- chart showing distribution of age of non-smoker donors

(0.001), RBC (0.002), HCT (0.004), MCH (0.022), and APTT (0.001), as  $P < 0.05$ . Therefore, we can conclude, that for these attributes, smokers and non-smokers show a significant difference (Table 3).

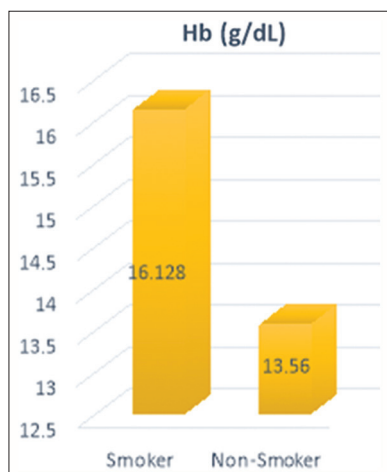
Significant P-value was observed in the case of Hb (g/dL) (0.001), RDW (%) (0.003), PT (s) (0.001), and INR (0.002),

as  $P < 0.05$ . Therefore, we can conclude that for these attributes, smokers and non-smokers show a significant difference (Table 4).

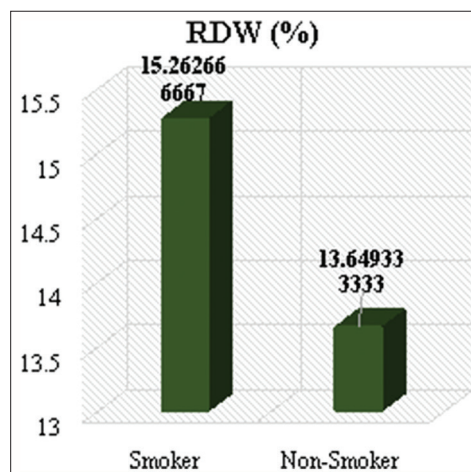
## DISCUSSION

Cigarette smoking has been related to induce various morphological and biochemical problems in individuals. In our study, we investigated the effect of smoking by analyzing the hematological profile, coagulation profile, and peripheral capillary oxygen saturation of healthy blood donors. In our study, RBC count showed a significant increase in smokers who were donors as compared to non-smoker donors ( $5.07 \times 10^3/\mu\text{L}$  among smokers vs.  $4.17 \times 10^3/\mu\text{L}$  among non-smokers). The findings of our study were consistent with Asif et al.,<sup>34</sup> who conducted a similar study on 142 male subjects, comparing the effect of cigarette smoking on hematological parameters in male smokers and non-smokers where the RBC count showed a significant increase in smokers when compared with non-smokers ( $5.3 \times 10^3/\mu\text{L}$  vs.  $5.06 \times 10^3/\mu\text{L}$ ). Khan et al.,<sup>35</sup> in a cross-sectional study, investigated the effect of smoking on RBC count, hemoglobin concentration, and red cell indices in 100 age-matched male donors (50 smokers and 50 non-smokers). They found that the RBC count ( $5.40 \times 10^6/\mu\text{L}$  among smokers vs.  $5.09 \times 10^6/\mu\text{L}$  among non-smokers); showed a significant increase in the group of smokers than that of non-smokers. Both of these studies depicted data similar to ours. Contrary to our results, Okafor and Okoroiwu,<sup>36</sup> in their study on the effects of cigarette smoking on hematological parameters of male cigarette smokers stated that the RBC count of smokers and non-smokers ( $5.20 \times 10^6/\mu\text{L}$  vs.  $4.99 \times 10^6/\mu\text{L}$ ) did not show any significant difference (Table 5).

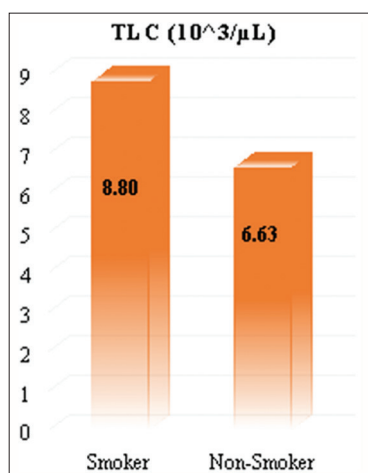
It has been noted that increased blood viscosity and clotting in smokers have been associated with increased levels of RBC. Increased RBC mass slows down the blood velocity increases the risk of clotting intravascularly and increases



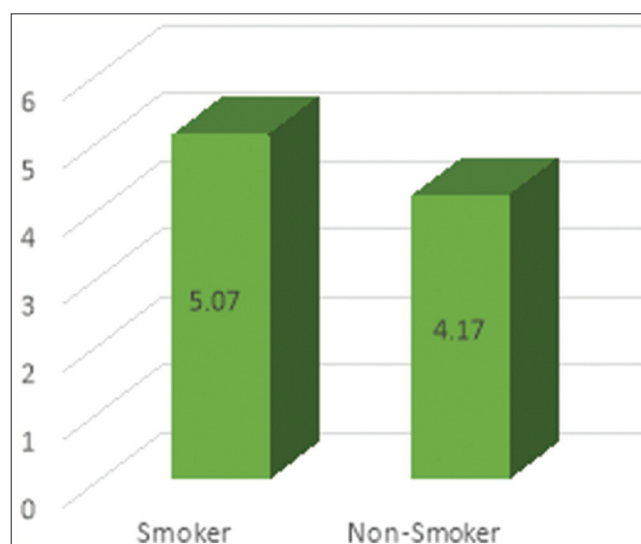
**Figure 3:** Mean of smokers (16.12 g/dL) and non-smokers (13.5 g/dL) in case of hemoglobin (Hb) smokers have a higher Hb than non-smokers



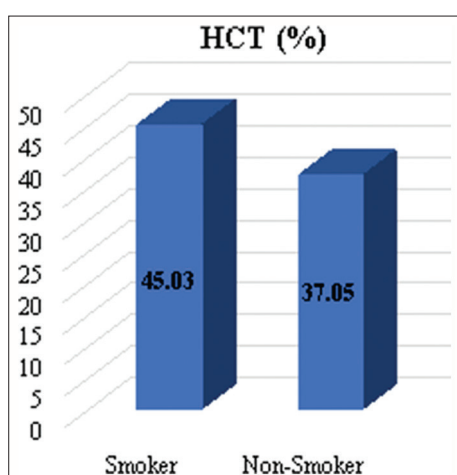
**Figure 6:** Mean of smokers (15.2) and non-smokers (13.6) in case of red cell distribution width (RDW), smokers have a higher RDW than non-smokers



**Figure 4:** Mean of smokers (8.8043) and non-smokers (6.6300) for total leukocyte count (TLC), TLC is higher in the case of smokers



**Figure 7:** Mean of smokers (5.07) and non-smokers (4.17) for red blood cell (RBC), RBC count is higher in case of smokers

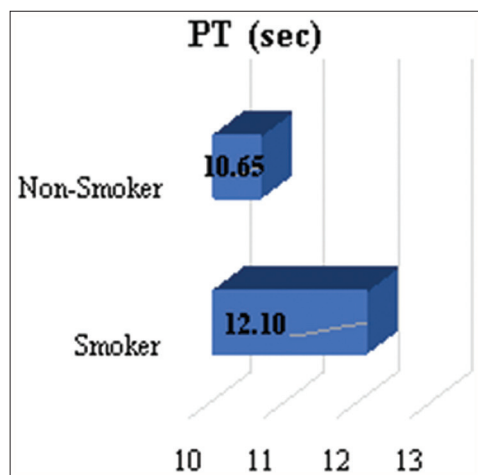


**Figure 5:** Mean of smokers (45.03) and non-smokers (37.05) for hematocrit count is higher in the case of smokers

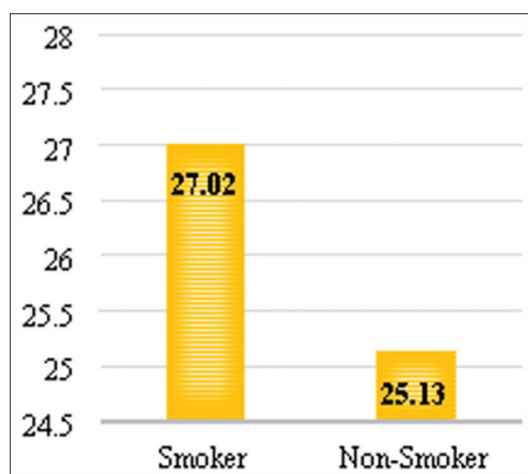
The Hb values recorded in our study were significantly high in smokers 16.13 g/dL as compared with non-smokers 13.56 g/dL. This finding is in consonance with previous findings of a few other studies. Asif et al.<sup>34</sup> also found in their study that Hb values were significantly higher in smokers 16.01 g/dL as compared with non-smokers 14.71 g/dL. Our study is also comparable with Khan et al.,<sup>35</sup> which demonstrated that mean hemoglobin level (16.11 g/dL smokers vs. 14.57 g/dL non-smokers) showed a significant increase in smokers than non-smokers. Okafor and Okoroiwu,<sup>36</sup> also found that tobacco cigarette smokers had significantly raised Hb values (15.30 g/dL smokers vs. 13.88 g/dL non-smokers) in smokers (Table 5).

The elevated Hb level is a compensatory reaction to carbon monoxide exposure. This inhaled carbon monoxide from cigarette smoke has a higher affinity for hemoglobin in

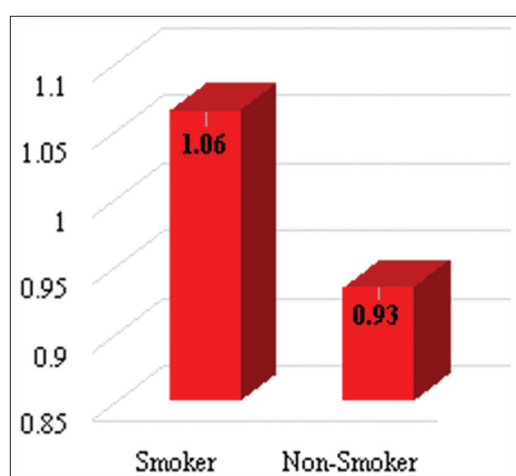
coronary vascular resistance along with decreased coronary blood flow and predisposes to thrombosis.



**Figure 8:** Mean of smokers (12.10 s) and non-smokers (10.65 s) in case of prothrombin time (PT), smokers have a higher PT than non-smokers



**Figure 9:** Mean of smokers (27.02 s) and non-smokers (25.13 s) for activated partial thromboplastin time (APTT), APTT count is higher in the case of smokers



**Figure 10:** Mean of smokers (1.06 s) and non-smokers (0.93 s) for international normalized ratio (INR), INR is higher in the case of smokers

comparison to oxygen. It displaces oxygen from hemoglobin in RBCs to produce carboxy-hemoglobin which leads to reduced release of oxygen in tissues creating hypoxia in chronic form.<sup>37</sup>

We also found significantly higher mean HCT 45.03% among smokers than non-smokers 37.05%. Higher values of HCT in smokers, in relation to non-smokers were confirmed by other studies as well. Asif et al.<sup>34</sup> also concluded that smokers had significantly higher mean HCT 49.67% than non-smokers 45.34%. Another study by Khan et al.<sup>35</sup> found that values of HCT (48% smoker vs. non-smokers 44.28%) show a significant increase in the same age group of smokers compared with non-smokers. While Okafor and Okoroiwu<sup>36</sup> found mean HCT (44.53% smokers vs. non-smokers 44.55%) which did not show any significant difference contrary to our study (Table 5).

In this study, the red cell indices which included MCH, MCV, MCHC, and RDW were analyzed. We found that the value of mean MCH was 32.99 (pg) in smokers as compared to non-smokers 32.01 (pg) and was found to be significant. The mean value of MCV in our study was 89.38 (fl) in smokers as compared to non-smokers 89.62 (fl) which was found to be insignificant. The mean value of MCHC came out to be 35.90 (g/dL) in smokers as compared to non-smokers 36.88 (g/dL) which was found to be insignificant. The mean RDW value in our study was 15.26% in smokers in comparison with non-smokers 13.65% and was found to be significant (Table 5).

Asif et al.<sup>34</sup> found that the mean MCHC 31.50 (g/dL) was significantly low in smokers compared with non-smokers 32.56 (g/dL) and MCV – 90.26 (fl), MCH 28.19 (pg), and RDW 13.28% in smoker did not show any significant difference as compared with non-smokers MCV – 88.61 (fl), MCH 28.84 (pg), and RDW 12.83%. Khan et al.<sup>35</sup> found that the values of MCH (30 pg – smoker and 28.87 pg – non-smoker) exhibited a significant increase in smokers as compared to non-smokers of the same age groups. While the increase in values of MCV (88.93fl smoker vs. non-smokers 87.18 fl) and MCHC (33.57 g/dL vs. smokers and 33.83 g/dL non-smokers) is found less marked and non-significant. Likewise, Okafor and Okoroiwu,<sup>36</sup> found that tobacco cigarette smokers had significantly higher values of MCH (29.52 pg among smokers and 28.16 pg among non-smokers) and MCHC (33.91 g/dL among smokers and 31.83 g/dL among non-smokers).

In our study, the mean TLC value was observed to be  $8.80 \times 10^3/\mu\text{L}$  in smokers compared to non-smokers  $6.63 \times 10^3/\mu\text{L}$  which was statistically significant. Higher values of TLC in smokers, in relation to non-smokers, were confirmed by other studies as well. Sandhya et al.<sup>38</sup> analyzed the impact of chronic cigarette smoking on PLT



aggregation and coagulation profile in apparently 60 healthy non-smoker males and compared them with 60 smokers. A highly significant difference was found in TLC ( $8.3 \times 10^3/\mu\text{L}$  smoker vs. non-smokers  $6.4 \times 10^3/\mu\text{L}$ ), thereby concluding that chronic smokers should be investigated for hemostatic dysfunctions.

TLC is the most useful, simple, and inexpensive biomarker for endothelial damage. We found that regular smokers had significantly higher TLC in comparison with non-smokers. Leukocytosis may be a marker of smoking-induced tissue damage. This high count can promote cardiac diseases through different mechanisms that cause inflammation, block the microvasculature, induce a hypercoagulable state, and promote infarct expansion.

The PLT count in this study was  $228.19 \times 10^3/\mu\text{L}$  for smokers and  $230.61 \times 10^3/\mu\text{L}$  for non-smokers which was statistically insignificant. There are only few studies addressing the effect of smoking on PLTs. A similar result was shown by Bילו,<sup>39</sup> who conducted a study on the effects of cigarette smoking on blood rheology and biochemistry. Among 606 subjects comprising 302 smokers and 304 control non-smokers were taken. There was no difference between smokers  $242.74 \times 10^3/\mu\text{L}$  and non-smokers  $245.74 \times 10^3/\mu\text{L}$  in regard to PLT count. Another study performed by Sandhya et al.<sup>38</sup> demonstrated a significant difference in PLT count ( $455 \times 10^3/\mu\text{L}$  -smoker vs. non-smokers  $480 \times 10^3/\mu\text{L}$ ) concluding that chronic smokers must be investigated for dysfunction in hemostasis.

We also investigated the effect of smoking on coagulation profile. The mean value of PT showed a significant increase in smoker donors 12.10 s than non-smoker donors 10.65 s. Few other studies are consistent with our study with slight variations of change in some parameters. Sivagangailakshmi and Rajkumar,<sup>1</sup> performed a case-control study on the effects of cigarette smoking on coagulation profile. Fifty smokers (subjects) and 50 non-smokers (controls) aged 20–50 were included in the study. Coagulation profile markers such as PT and APTT were estimated. The mean value of PT in smokers was 23.81 s in comparison with non-smokers 18.67 s and the mean value of APTT in smokers was 45.62 s in comparison with non-smokers 39.53 s and significantly prolonged in smokers compared to non-smokers; it was concluded that cigarette smoking alters PT and APTT values. While Elkhalfa<sup>40</sup> conducted a case-control study on the effects of cigarette smoking on coagulation screening tests and PLT counts in a Sudanese male adult population. One hundred adult cigarette smokers were selected and another 100 non-smokers were selected as healthy controls. The mean PT was significantly lower in smokers (12.9 s) compared with non-smokers (13.7 s). Cigarette smokers tend to have lower PT values, compared to non-smokers (Table 6).

We also investigated APTT which showed a significant increase in smokers 27.02 s than non-smokers 25.13 s. Sivagangailakshmi and Rajkumar,<sup>1</sup> found that the mean value of APTT in smokers was 45.62 s in comparison with non-smokers 39.53 s and was significantly prolonged in smokers compared to non-smokers. While Elkhalfa<sup>40</sup> in their study found that APTT had no significant variation in smokers (30.5 s) and non-smokers (37.9 s) (Table 6).

We also investigated the mean INR of smokers 1.06 smokers compared to non-smokers 0.93 which was statistically significant. Almarshad and Hassan<sup>22</sup> found that smoking alters some hematology parameters leading to significant deterioration in blood flow properties and decreased INR. The mean INR of smokers was 1.14 compared with non-smoker 1.37. In another study conducted by Elkhalfa,<sup>40</sup> the results showed that the mean INR was significantly lower in smokers (0.95 vs. 1.01) Cigarette smokers tend to have shorter INR values, compared to non-smokers. Therefore, smoking might be associated with bleeding disorders (Table 6).

We also analyzed the peripheral capillary oxygen saturation by measuring the SpO<sub>2</sub>. The mean value of smokers was 96.97% compared to non-smokers 97.16%. Jeon et al.,<sup>24</sup> studied the effect of smoking cigarettes on SpO<sub>2</sub>. Thirty-eight non-smokers and 29 smokers were included in the study. The mean SpO<sub>2</sub> of the non-smoking group was 96% in the pre-test and 97% in the post-test, that is, both groups did not show significant differences. This finding is similar to our study. Özdal et al.<sup>26</sup> studied the effect of smoking on oxygen saturation. A total of 406 individuals who were healthy smokers (n=189) and non-smokers (n=217) were included in this study. Pulse oximetry was utilized for the determination of SpO<sub>2</sub>. Non-smokers had significantly higher SpO<sub>2</sub> than smokers. SpO<sub>2</sub> values of smokers were 97.28%, whereas the percentage of SpO<sub>2</sub> values of non-smokers was 97.92%. Statistical analysis between smokers and non-smokers showed a significance level in favor of non-smokers in terms of SpO<sub>2</sub>.

#### Limitations of the study

Oxygen saturation has been decreased in many studies but in our study we didn't get any significant difference.

## CONCLUSION

Cigarette smoking has adverse effects on hematological parameters (RBC count, Hb, HCT, MCH, RDW, and TLC) and coagulation profiles (PT, APTT, and INR). In our result RBC, Hb, HCT, MCH, and RDW are significantly high in smokers all of which contribute to WBV, indicating higher blood viscosity in smokers. This is a marker for adverse blood rheology and thrombosis in smokers.

It has been seen that increases in RBC levels are associated with increased viscosity of blood and clotting in cigarette smokers. The elevated Hb level is a compensatory reaction to carbon monoxide exposure. The carbon monoxide from smoke has an increased affinity for Hb as compared with oxygen. It displaces oxygen from Hb in erythrocytes and forms carboxyhemoglobin. This carboxyhemoglobin decreases the release of oxygen to tissues and causes hypoxia in chronic form.

Increased levels of hematocrit have been observed in smokers. This increase is due to compensation for exposure to carbon monoxide. An increase in hematocrit is observed in smokers which may lead to a hypercoagulable state as can be seen from INR which directly affects the cardiac health. The increase in MCH and RDW could be due to the inhaled carbon monoxide gas (CO), which is one of the inhaled components of cigarette smoke.

TLC is a very useful, affordable, and simple biomarker for damage and inflammation in endothelium. We found that smokers had significantly higher TLC when compared to non-smokers. A high count can cause cardiac diseases through various mechanisms that cause inflammation, block the microvasculature, lead to a hypercoagulable state, and promote the expansion of infarct.

Oxidative stress caused by cigarettes alters the functions of the liver which alters the production of coagulation factors. This might cause prolongation of PT and APTT.

Our study also analyzed the effects of smoking on SpO<sub>2</sub>. Although there was no significant difference in SpO<sub>2</sub> among smokers and non-smokers in our study, we strongly recommend that further studies be done on the oxygen saturation of blood donors which might put light on this very important factor regarding the quality assessment of the donated blood.

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## REFERENCES

1. Sivagangailakshmi V and Rajkumar D. Effects of cigarette smoking on coagulation profile among smokers. *Int Arch Integr Med.* 2017;4(8):116-120.
2. Buist AS, Vollmer WM and McBurnie MA. Worldwide burden of COPD in high- and low-income countries. Part I. The Burden of Obstructive Lung Disease (BOLD) Initiative. *Int J Tuberc Lung Dis.* 2008;12(7):703-708.
3. Vineis P, Alavanja M, Buffler P, Fonham E, Franceschi S, Gao YT, et al. Tobacco and cancer: Recent epidemiological evidence. *J Natl Cancer Inst.* 2004;96(2):99-106.
4. Barreto SG. How does cigarette smoking cause acute pancreatitis? *Pancreatol.* 2016;16(2):157-163. <https://doi.org/10.1093/jnci/djh014>
5. Li LF, Chan RL, Lu L, Shen J, Zhang L, Wu WK, et al. Cigarette smoking and gastrointestinal diseases: The causal relationship and underlying molecular mechanisms (review). *Int J Mol Med.* 2014;34(2):372-380. <https://doi.org/10.1016/j.pan.2015.09.002>
6. Gautam DK, Jindal V, Gupta SC, Tuli A, Kotwal B and Thakur R. Effect of cigarette smoking on the periodontal health status: A comparative, cross sectional study. *J Indian Soc Periodontol.* 2011;15(4):383-387. <https://doi.org/10.4103/0972-124X.92575>
7. Slagter SN, van Vliet-Ostaptchouk JV, Vonk JM, Boezen HM, Dullaart RP, Kobold AC, et al. Associations between smoking, components of metabolic syndrome and lipoprotein particle size. *BMC Med.* 2013;11:195. <https://doi.org/10.1186/1741-7015-11-195>
8. Harel-Meir M, Sherer Y and Shoenfeld Y. Tobacco smoking and autoimmune rheumatic diseases. *Nat Clin Pract Rheumatol.* 2007;3(12):707-715. <https://doi.org/10.1038/ncprheum0655>
9. Lu JT and Creager MA. The relationship of cigarette smoking to peripheral arterial disease. *Rev Cardiovasc Med.* 2004;5(4):189-193.
10. Law MR, Morris JK and Wald NJ. Environmental tobacco smoke exposure and ischaemic heart disease: An evaluation of the evidence. *BMJ.* 1997;315(7114):973-980. <https://doi.org/10.1136/bmj.315.7114.973>
11. Messner B and Bernhard D. Smoking and cardiovascular disease: Mechanisms of endothelial dysfunction and early atherogenesis. *Arterioscler Thromb Vasc Biol.* 2014;34(3):509-515. <https://doi.org/10.1161/ATVBAHA.113.300156>
12. Shah RS and Cole JW. Smoking and stroke: The more you smoke the more you stroke. *Expert Rev Cardiovasc Ther.* 2010;8(7):917-932. <https://doi.org/10.1586/erc.10.56>
13. Calapai G, Caputi AP, Mannucci C, Russo GA, Gregg E, Puntoni R, et al. Cardiovascular biomarkers in groups of established smokers after a decade of smoking. *Basic Clin Pharmacol Toxicol.* 2009;104(4):322-328. <https://doi.org/10.1111/j.1742-7843.2008.00361.x>
14. Barua RS and Ambrose JA. Mechanisms of coronary thrombosis in cigarette smoke exposure. *Arterioscler Thromb Vasc Biol.* 2013;33(7):1460-1467. <https://doi.org/10.1161/ATVBAHA.112.300154>
15. Cheesbrough M. *District Laboratory Practice in Tropical Countries Part 2.* 2<sup>nd</sup> ed. Cambridge, UK: Cambridge University Press; 2006. p. 315.
16. Yarnell JW, Baker IA, Sweetnam PM, Bainton D, O'Brien JR, Whitehead PJ, et al. Fibrinogen, viscosity, and white blood cell count are major risk factors for ischemic heart disease. The Caerphilly and Speedwell collaborative heart disease studies. *Circulation.* 1991;83(3):836-844. <https://doi.org/10.1161/01.cir.83.3.836>
17. Wannamethee SG, Lowe GD, Shaper AG, Rumley A, Lennon L and Whincup PH. Associations between cigarette smoking, pipe/cigar smoking, and smoking cessation, and haemostatic and inflammatory markers for cardiovascular disease. *Eur Heart J.* 2005;26(17):1765-1773. <https://doi.org/10.1093/eurheartj/ehi183>
18. Van Tiel E, Peeters PH, Smit HA, Nagelderke NJ, Van

- Loon AJ, Grobbee DE, et al. Quitting smoking may restore hematological characteristics within five years. *Ann Epidemiol*. 2002;12(6):378-388.  
[https://doi.org/10.1016/s1047-2797\(01\)00282-4](https://doi.org/10.1016/s1047-2797(01)00282-4)
19. Malenica M, Prnjavorac B, Bego T, Dujic T, Semiz S, Skrbo S, et al. Effect of cigarette smoking on haematological parameters in healthy population. *Med Arch*. 2017;71(2):132-136.  
<https://doi.org/10.5455/medarh.2017.71.132-136>
  20. Elgari MM. Hematological changes induced by heavy cigarette smoking. *Glob Adv Res J Med Med Sci*. 2017;6(12):327-329.
  21. Anandha Lakshmi S, Lakshmanan A, Ganesh Kumar P and Saravanan A. Effect of intensity of cigarette smoking on haematological and lipid parameters. *J Clin Diagn Res*. 2014;8(7):BC11-BC13.  
<https://doi.org/10.7860/JCDR/2014/9545.4612>
  22. Almarshad HA and Hassan FM. Alterations in blood coagulation and viscosity among young male cigarette smokers of Al-Jouf region in Saudi Arabia. *Clin Appl Thromb Hemost*. 2014;22(4):386-389.  
<https://doi.org/10.1177/1076029614561319>
  23. Soronadi CN, Ugwene FO, Odurukwe O, Odetunde OI and Nweke LM. The impact of chronic smoking on the intrinsic and extrinsic coagulation pathways of smokers in Enugu, South-East Nigeria. *Br J Med Med Res*. 2015;12(9):1-5.  
<https://doi.org/10.9734/BJMMR/2016/22597>
  24. Jeon M, Jeong H, Lee K and Yim J. The acute effect of smoking a single cigarette on vascular status, SpO<sub>2</sub>, and stress level. *Med Sci Monit*. 2014;20:601-607.  
<https://doi.org/10.12659/MSM.890367>
  25. Chandra A, Swami S, Verma AK, Nair G, Feroz A, Prakash V, et al. Study of pulse oximetric and spirometric parameters in smokers. *Int J Biomed Res*. 2016;7(2):51-54.  
<https://doi.org/10.7439/IJBR.V7I2.2975>
  26. Özdal M, Bilgiç M, Pancar Z and Çinar V. Effect of smoking on oxygen saturation in healthy sedentary men and women. *EC Pulmonol Respir Med*. 2017;4(6):178-182.
  27. Marwaha N. Transfusion medicine in India: Expanding horizons. *Asian J Transfus Sci*. 2014;8(Suppl 1):S3-S5.  
<https://doi.org/10.4103/0973-6247.130948>
  28. Chatterjee K, Coshic P, Borgohain M and Agarwal N. Experience of buffy coat pooling of platelets as a supportive care in thrombocytopenic dengue patients: A prospective study. *Asian J Transfus Sci*. 2014;8(2):89-91.  
<https://doi.org/10.4103/0973-6247.137439>
  29. Salamat N. Effectiveness of blood donor questionnaire directed at risk factor for transfusion transmitted infections in Pakistani population. *Asian J Transfus Sci*. 2012;6(2):169-173.  
<https://doi.org/10.4103/0973-6247.98929>
  30. Ahmed SG, Kagu MB and Abjah UA. Haematological parameters of blood donors in North East Nigeria and the implication on quality of blood products. *Afr Sanguine*. 2010;13(1):1-8.
  31. Roubinian NH, Westlake M, St. Lezin EM, Edgren G, Brambilla DJ, Lee C, et al. Association of donor age, body mass index, hemoglobin, and smoking status with in-hospital mortality and length of stay among red blood cell transfused recipients. *Transfusion*. 2019;59(11):3362-3370.  
<https://doi.org/10.1111/trf.15541>
  32. Symvoulakis EK, Vardavas CI, Fountouli P, Stavroulaki A, Antoniou KM, Duijker G, et al. Time interval from cigarette smoke exposure to blood donation and markers of inflammation: Should a smoking cut-off be designated? *Xenobiotica*. 2010;40(9):613-620.  
<https://doi.org/10.3109/00498254.2010.500745>
  33. DeSimone RA, Hayden JA, Mazur CA, Vasovic LV, Sachais BS, Zhao Z, et al. Red blood cells donated by smokers: A pilot investigation of recipient transfusion outcomes. *Transfusion*. 2019;59(8):2537-2543.  
<https://doi.org/10.1111/trf.15339>
  34. Asif M, Karim S, Umar Z, Malik A, Ismail T, Chaudhary A, et al. Effect of cigarette smoking based on hematological parameters: Comparison between male smokers and non-smokers. *Turk J Biochem/Turk Biyokim Derg*. 2013;38(1):75-80.  
<https://doi.org/10.5505/tjb.2013.68077>
  35. Khan MI, Bukhari MH, Akhtar MS and Brar S. Effect of smoking on red blood cells count, hemoglobin concentration and red cell indices. *Pak J Med Health Sci*. 2014;8(2):361-364.
  36. Okafor IM and Okoroiwu HU. Effects of tobacco cigarette smoking on some hematological parameters of male cigarette smokers in Southern Nigeria. *Asian J Med Health*. 2017;5(3):1-6.  
<https://doi.org/10.9734/AJMAH/2017/33813>
  37. Cronenberger C, Mould RD, Roethig HJ and Sarkar M. Population pharmacokinetic analysis of carboxyhaemoglobin concentrations in adult cigarette smokers. *Br J Clin Pharmacol*. 2008;65(1):30-39.  
<https://doi.org/10.1111/j.1365-2125.2007.02974.x>
  38. Sandhya M, Satyanarayana U, Mohanty S and Basalingappa DR. 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.
  39. Bilto YY. Effects of cigarette smoking on blood rheology and biochemistry. *Int J Sci Res*. 2015;3(4):107-112.
  40. Elkhalifa AM. Effects of cigarette smoking on coagulation screening tests and platelet counts in a Sudanese male adults population. *Saudi Med J*. 2018;39(9):897-901.  
<https://doi.org/10.15537/smj.2018.9.22630>

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**AS**- Definition of intellectual content, literature survey, prepared first draft of manuscript, implementation of study protocol, data collection, data analysis, manuscript preparation, and submission of article; **KT**- Concept, design, clinical protocol, manuscript preparation, editing, and manuscript revision; **NZ**- Literature survey, and preparation of figures; **SI**- Statistical analysis, and interpretation; **SA**- Design of study, coordination, and manuscript revision; **MSR**- Concept, design, manuscript preparation, editing, and manuscript revision.

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