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Original Article

Dentistry Section

Assessment of Haematological Parameters among Smoking and Smokeless Tobacco Users in Bengaluru: A Cross-sectional Study

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ABSTRACT

Introduction: Use of tobacco is one of the major causes of death worldwide, and it harms all the tissues and organs of the body, including blood. Tobacco is consumed worldwide despite its adverse effects. Numerous studies have been done on the haematological parameters and smoking or smokeless tobacco use, but limited studies exist on the combined form (smoking and smokeless tobacco use) and haematological parameters.

Aim: To investigate the effect of smoking and smokeless tobacco on the haematological parameters of tobacco users in Bengaluru, Karnataka, India.

Materials and Methods: This cross-sectional study was conducted in Rajarajeswari Dental College and Hospital, Bengaluru, Karnataka, India from September 2024 to February 2025, with a total of 80 participants (20 smokers, 20 smokeless tobacco users, 20 both smoking and smokeless tobacco users and 20 non tobacco users) in the age range of 20 to 85 years. They were selected on the basis of inclusion criteria of using five cigarettes/day for at least three years and three packets of chewable tobacco/day for at least three years among the participants attending a dental Institution in Bengaluru. Clinical examination was done for any mucosal lesions. A 5 mL of venous blood was collected, and complete blood count analysis was carried out. All the data were subjected to statistical analysis; the Kruskal-Wallis test and Analysis of Variance (ANOVA) test were used for intergroup comparisons, and post-hoc pairwise comparisons were performed where applicable. A p-value of less than 0.05 was considered statistically significant.

Results: RBCs and Haemoglobin (Hb) (g/dL) were decreased in smokers and smokeless tobacco users and increased in patients with both habits. Among tobacco users, WBCs were decreased in smokers and increased in smokeless tobacco users and in patients with both habits. Mean platelet counts were higher in the three tobacco-use groups than in controls; however, the between-group difference was not statistically significant. Red Blood Cells (RBC) (p=0.031), Packed Cell Volume (PCV) (p=0.026) and eosinophil count (p=0.049) were statistically significant between the groups.

Conclusion: The current study shows that there are variations in the haematological parameters due to the tobacco habit of all forms, and awareness of this among the public may prevent Oral Cancer, atherosclerosis, cardiovascular disease and other tobacco-related disorders.

Keywords: Haemoglobin, Leukoplakia, Oral mucosal lesions, White blood cells

INTRODUCTION

In the Indian subcontinent, oral cancer is one of the top three cancer forms and a contemporary epidemic of noncommunicable diseases; the cause of this is the use of Tobacco [1]. Tobacco is the dried, fermented, and processed leaves of the Nicotiana tabacum plant, which are used in chewed, sniffed or smoked forms [2]. According to WHO, nearly one-third of the global adult population are tobacco users. Global Adult Tobacco Survey-2 (GATS-2) reports that 28.6% of the population consumes tobacco in any form, 10.7% smoke and 21.4% use smokeless tobacco [3]. Around 6.4 million fatalities were attributed to tobacco use in 2015, and it is predicted that the number will climb to 8.3 million in 2030 and one billion in the twenty-first century [4]. Tobacco smoke contains 4000 compounds that have a more or less adverse effect on human health, among which high concentrations of oxidants and reactive species, nicotine, carbon monoxide and nitrogen are linked with increased risk of cancer [5,6].

Tobacco products act locally on keratinocyte stem cells; they are absorbed and produce DNA adducts which interfere with the accuracy of DNA replication and cause mutations [5]. Tobacco products significantly alter the metabolic pattern in a robust way and accelerate the risk of systemic inflammation, such as RBC morphology, influence leukocyte count and cardiovascular diseases [7]. Tobacco is associated with increased risk of many chronic diseases that shorten life and decrease quality of life [8].

Numerous studies have been carried out on haematological parameters and smoking or smokeless forms with varied results, and very few studies have been published on the combined use of both smoking and smokeless forms of tobacco and haematological parameters; therefore, the current study aimed to evaluate the effect of both smoking and smokeless tobacco on haematological parameters.

The null hypothesis was that there is no difference in haematological parameters between tobacco users and non users.

The alternative hypothesis was that haematological parameters differ among individuals using different forms of tobacco and non tobacco users.

MATERIALS AND METHODS

This cross-sectional observational study was conducted at Rajarajeswari Dental College in Bengaluru, India, from September 2024 to February 2025 after receiving permission from the Ethical Committee of the Institution RRDCH/IEC/2024/034. Written informed consent was obtained from all participants before enrollment.

Sample size calculation: The sample size for the present study was estimated using G Power software (latest ver. 3.1.9.7;

Heinrich-Heine-Universität Düsseldorf, Düsseldorf, Germany). The sample size estimation was performed at 5% alpha error (α=0.05), with an effect size of 0.40 {Based on the Cohen classification [9], considering a moderate effect size for the anticipated difference in mean RBC levels between 4 groups} and the power of the study at 80%, revealed that a minimum of 76 samples needed for the study, which was rounded off to 80 samples. The sample size was divided into 20 samples, each for four groups.

Inclusion criteria: Healthy individuals with a history of consumption of tobacco (smoking and smokeless form), smoking a minimum of five cigarettes per day for at least three years and three packs of chewable tobacco for at least three years, who were included as study groups, and 20 healthy individuals with no history of tobacco consumption in any form were selected as controls.

Exclusion criteria: Individuals with any systemic illness or immunocompromised patients, Individuals consuming alcohol and individuals not willing to participate in the study.

Study Procedure

So, a total of 80 patients aged 20 to 85 years who fulfilled the inclusion and exclusion criteria were selected and grouped as follows: 20 smokers (Group 1), 20 smokeless tobacco users (Group 2), 20 patients who used both smoked and smokeless tobacco (Group 3), and 20 non tobacco users (Group 4).

The data regarding demography, socioeconomic status and tobacco usage were recorded, and the patient was examined intraorally for any tobacco-related oral mucosal lesions. A 5 mL of venous blood was collected from all subjects using a routine venipuncture method and was stored in vials containing EDTA. Complete blood count {red blood cell count, Total Leukocyte Count (TLC), Differential Leukocyte Count (DLC), platelet count, PCV, Haemoglobin, Mean Corpuscular Volume (MCV), Mean Corpuscular Haemoglobin (MCH), Mean Corpuscular Haemoglobin Concentration (MCHC)} were performed manually by a single trained examiner, and calibration was done before the start of the study.

Complete blood count was performed manually using standard haematological techniques. Haemoglobin concentration was estimated by Sahli's acid hematin method. TLC and platelet count were determined using an improved Neubauer haemocytometer. DLC was assessed on Leishman-stained peripheral blood smears by counting 100 leukocytes under light microscopy. PCV was measured using the microhematocrit method. Erythrocyte indices including MCV, MCH, and MCHC were calculated using standard formulae. The reference ranges used were Hb: 12-16 g/dL (females) and 13-17 g/dL (males); TLC: 4,000-11,000 cells/mm^3; platelet count: 150,000-450,000/mm^3; PCV: 36-46% (females) and 41-53% (males); MCV: 80-100 fL; MCH: 27-33 pg; and MCHC: 32-36 g/dL. All procedures and reference values were based on standard methods described in Dacie and Lewis [10].

Before commencement of the study, the examiner underwent calibration training. Standard operating procedures were followed for specimen collection, transportation, storage, and analysis. All blood samples were collected under similar conditions using sterile disposable equipment and processed within the recommended time period. Reagents were checked for expiry, and duplicate measurements were performed randomly on 10% of samples to ensure consistency. Data entry was cross-verified to minimise transcription errors. All the data obtained were subjected to statistical analysis

To calculate intraobserver calibration, the single trained examiner re-examined a random sample of duplicate measurements for the above measurements. The reliability was calculated using the coefficient of variation (continuous haematological parameters), which was 0.88%, indicating low measurement variability and good intraobserver reproducibility.

STATISTICAL ANALYSIS

The collected data were entered into Microsoft Excel and analysed using Statistical Package for Social Sciences (SPSS) statistics version 25.0 (IBM Corp., Armonk, NY, USA). Descriptive statistics were used to summarise the study variables. Continuous variables were expressed as Mean±Standard Deviation (SD) or median with Interquartile Range (IQR) based on data distribution, while categorical variables were presented as frequencies and percentages. Normality of the data was assessed using the Shapiro-Wilk test. Since the data were not normally distributed, the Kruskal-Wallis test was used to compare continuous variables among more than two independent groups. Post-hoc pairwise comparisons were performed where applicable. A p-value of less than 0.05 was considered statistically significant.

RESULTS

A total of 80 subjects with 20 smokers, 20 smokeless tobacco chewers, 20 both smokers and chewers and 20 non tobacco users (77 males and 3 females) were included in the study. Mean age of the non users (control/group 4) were 36.85 years; smokers (group 1) had a higher mean age of 42.45 years; chewers (group 2) were also at a similar mean age of 42.60 years; in contrast, individuals with both smoking and chewing (group 3) were the youngest, with mean age of 31.30 years, as shown in [Table/Fig-1,2].

Variable		Min-Max	Mean±SD	Median (IQR)	Test value	p-value
Age (in years)	Control	26-59	36.85±9.615	36 (12)	χ^2= 11.720	0.008*
	Smokers	22-62	42.45±11.834	45 (16.5)		
	Chewers	21-72	42.60±14.763	42.5 (20.75)		
	Both	20-48	31.30±7.665	31.5 (10.5)		

[Table/Fig-1]: Age distribution among the study groups.
Test done: Kruskal Wallis Test; IQR- Inter Quartile Range; p-value <0.05 statistically significant

		n (%)
Gender	Female	3 (3.75)
	Male	77 (96.25)

[Table/Fig-2]: Gender distribution among the study groups.

The various mucosal changes observed were homogenous leukoplakia in 6.3%, OSMF in 7.5%, smoker's melanosis in 3.8%, tobacco pouch keratosis in 3.8% chewers mucosa in 2.6%, chewers mucosa with OSMF in 1.3%, less frequent findings included angular cheilitis, lichenoid reaction, malignancy, xerostomia and smokers palate each observed in 1.3% of individuals as displayed in the [Table/Fig-3].

Comparison of Hb: Mean serum Hb was lower in smokers and chewers, whereas individuals with both habits had increased mean serum Hb when compared to the controls, with a Kruskal-Wallis value of 6.843 and p-value of 0.077, as shown in [Table/Fig-4].

Oral manifestation	n (%)
Nil	50 (62.5)
Angular chelitis	1 (1.3)
Chewers mucosa	2 (2.6)
Homogenous leukoplakia	5 (6.3)
Leukoedema	2 (2.5)
Lichenoid reaction	2 (2.5)
Malignant growth, Xerostomia	1 (1.3)
Oral submucous fibrosis	7 (8.75)
Oral submucous fibrosis with chewers mucosa	1 (1.3)
Smokers melanosis	3 (3.8)
Smokers palate	1 (1.3)
Speckled leukoplakia	1 (1.3)

Tobacco pouch keratosis	4 (5.0)

[Table/Fig-3]: Distribution of oral manifestations of tobacco associated lesions among the study group.
Test done: Kruskal Wallis Test; IQR: Inter Quartile Range; χ^2 - Kruskal Wallis Test value; p-value <0.05 statistically significant

Comparison of Hb		Min-Max	Mean±SD	Median (IQR)	Test value	p-value
Hb (g/dL)	Control	13.0-16.0	15.450±.7480	15.8 (1)	χ^2 =6.843	0.077
	Smokers	12.0-16.0	15.310±.9635	15.8 (1)		
	Chewers	11.0-16.2	14.940±1.3964	15.4 (0.8)		
	Both	14.0-16.2	15.720±.5444	16 (0.2)		

[Table/Fig-4]: Comparison of Hb g/dL among controls, smokers, smokeless tobacco users and both smokers and smokeless tobacco users.
Test done: Kruskal Wallis Test; IQR: Inter Quartile Range; χ^2 - Kruskal Wallis Test value; p-value <0.05 statistically significant

Comparison of Total RBCs (TRBCs): Mean serum TRBCs were slightly lower in smokers, with wider variability; lowest in chewers and the mean serum TRBCs were increased in subjects with both habits. The significant p-value of 0.031 and Kruskal-Wallis value of 8.873 strengthen the evidence for habit-related haematological alterations, as shown in [Table/Fig-5].

Comparison of RBC		Min-Max	Mean±SD	Median (IQR)	Test value	p-value
RBC in cells/mm^3	Control	4.6-5.2	5.065±.1599	5.05 (0.2)	χ^2= 8.873	0.031*
	Smokers	4.5-5.2	5.005±.1905	5 (0.25)		
	Chewers	4.2-5.2	4.890±.3354	5 (0.75)		
	Both	4.5-5.2	5.100±.1686	5.2 (0.2)		

[Table/Fig-5]: Comparison of RBC among controls, smokers, smokeless tobacco users and both smokers and smokeless tobacco users.
Test done: Kruskal Wallis Test; IQR: Inter Quartile Range; χ^2 - Kruskal Wallis Test value; p-value <0.05 statistically significant

Comparison of PCV: Mean PCV was decreased in smokers and chewers, while it was increased in individuals with both habits, which was statistically significant with a Kruskal-Wallis value of 9.260 and p-value of 0.026 [Table/Fig-6].

Comparison of MCV, MCH and MCHC: There was no significant difference in the values of MCV with Kruskal Wallis value of 0.122 and p-value of 0.989, MCH with Kruskal Wallis value of 2.093 and p-value of 0.553 and MCHC with Kruskal Wallis value of 2.433 and p-value of 0.488 among the different groups [Table/Fig-6].

Comparison of Total Leukocyte Count (TLC): Among tobacco users, mean serum TLC was lower in smokers and also in individuals with both smoking and chewing habits, but the mean serum TLC was increased in chewers, which was not statistically significant, with a Kruskal-Wallis value of 1.716 and p-value of 0.633, as shown in [Table/Fig-7].

Comparison of Differential Leukocyte Count (DLC): The neutrophil count was slightly increased in smokers when compared to the controls, whereas there was no change in chewers and in individuals with both habits, which was not statistically significant with a p-value of 0.231, as shown in [Table/Fig-8].

The lymphocyte count was slightly decreased in smokers, chewers and individuals with both habits when compared to controls, which was non significant with a p-value of 0.704, as shown in [Table/Fig-8].

The eosinophil count was slightly decreased in smokers, increased in chewers and individuals with both habits also showed an increased eosinophil count when compared to controls and showed a borderline statistically significant overall difference (p=0.049), which should be interpreted cautiously in view of multiple comparisons with a Kruskal-Wallis value of 7.853. Monocytes and basophils showed no change among the different groups [Table/Fig-8].

Comparison of platelet count: The mean platelet count was increased in smokers, chewers and in individuals with both habits, which was not statistically significant, with a Kruskal-Wallis value of 4.267 and a p-value of 0.234 [Table/Fig-9].

Post-hoc pairwise comparisons were performed, where a p-value<0.05 was considered statistically significant in RBC, Eosinophil and PCV to show the significant difference in the particular groups, as shown in [Table/Fig-10-12], respectively.

DISCUSSION

Tobacco is the dried and processed leaves of the plant Nicotiana Tabacum that is widely cultivated and commercially grown in many countries of the world. Tobacco is mostly consumed in the form of smoking, chewing, snuffing or dipping tobacco [11]. Nicotine is a psychoactive ingredient metabolically inactivated by CYP2A6 to cotinine, metabolised by the liver and detoxified [3].

Thousands of compounds are found in both forms of tobacco. The smoking form contains over 4000 different chemicals, and at least 50 are known carcinogens. Smokeless tobacco contains 28 carcinogens; tobacco-specific nitrosamines are present in higher concentrations, and nicotine content is lower in smokeless tobacco compared to the smoking form of tobacco [12].

This may be the reason for the mucosal permeability and easy penetration of the carcinogens, which can be clinically appreciated as smokers' palate, chewers' mucosa, leukoplakia, erythroplakia, OSMF etc., which later may undergo malignant transformation. Most of the oral mucosal changes were observed in both habits in the present study [3,12].

In the present study, RBCs were decreased in both smokers and chewers but increased in individuals with both habits. This was in

Comparison		Min-Max	Mean±SD	Median (IQR)	Test value	p-value
PCV (%)	Control	39-48	46.80±2.262	48 (1.5)	χ^2=9.260	0.026*
	Smokers	36-48	45.85±2.852	47 (3)		
	Chewers	33-49	44.70±4.169	46 (2)		
	Both	42-49	47.10±1.651	48 (1)		
MCV (cumm)	Control	85-96	92.25±2.593	92 (0.5)	χ^2=0.122	0.989
	Smokers	80-94	91.50±3.236	92 (4)		
	Chewers	79-94	91.25±3.782	92 (4)		
	Both	90-94	92.15±1.226	92 (0.25)		
MCH (pg)	Control	28-32	30.60±.821	31 (1)	χ^2=2.093	0.553
	Smokers	27-32	30.70±1.174	31 (1)		
	Chewers	26-32	30.60±1.465	31 (1.25)		
	Both	30-32	31.00±.562	31 (0)		

MCHC (%)	Control	31-34	32.85±.875	33 (0)	χ^2=2.433	0.488
	Smokers	33-34	33.20±.410	33 (0)		
	Chewers	33-34	33.20±.410	33 (0)		
	Both	33-34	33.20±.410	33 (0)		

[Table/Fig-6]: Comparison of PCV, MCV, MCH and MCHC among controls, smokers, smokeless tobacco users and both smokers and smokeless tobacco users.
Test done: Kruskal Wallis Test; IQRL Inter Quartile Range; χ^2 Kruskal Wallis Test value; p-value <0.05 statistically significant

Comparison of TLC		Min-Max	Mean±SD	Median (IQR)	Test value	p-value
TLC (cells/mm^3)	Control	6800-9800	7972.50±1013.134	7925 (2050)	χ^2=1.716	0.633
	Smokers	3500-9200	7695.00±1343.003	8100 (1587.5)		
	Chewers	7000-9000	7870.00±712.002	7925 (1500)		
	Both	6800-9500	7639.00±893.396	7375 (1475)		

[Table/Fig-7]: Comparison of TLC among controls, smokers, smokeless tobacco users and both smokers and smokeless tobacco users.
Test done: Kruskal-Wallis Test; IQR- Inter Quartile Range; χ^2 - Kruskal Wallis Test value; p-value <0.05 statistically significant

Comparison of differential WBC count		Min-Max	Mean±SD	Median (IQR)	Test value	p-value
Neutrophil (%)	Control	58-68	62.45±2.645	62.5 (4.25)	χ^2=1.414	0.231
	Smokers	60-70	63.75±2.731	64 (4)		
	Chewers	58-68	62.25±3.160	62 (5)		
	Both	58-68	62.15±2.621	62 (4)		
Lymphocyte (%)	Control	30-47	35.90±3.972	34.5 (4.5)	χ^2=0.955	0.704
	Smokers	30-40	34.40±2.798	34		
	Chewers	27-39	34.25±3.582	35 (4.5)		
	Both	28-40	34.90±3.177	34 (5.25)		
Eosinophil (%)	Control	0-6	2.30±1.750	2 (1.75)	χ^2=7.853	0.049*
	Smokers	0-6	1.85±1.496	2 (1.25)		
	Chewers	1-10	3.50±2.259	2.5 (2)		
	Both	0-6	2.95±1.820	3 (2)		
Monocytes (%)	Control	0	0	0	χ^2=0.000	NA
	Smokers	0	0	0		
	Chewers	0	0	0		
	Both	0	0	0		
Basophil (%)	Control	0	0	0	χ^2=0.000	NA
	Smokers	0	0	0		
	Chewers	0	0	0		
	Both	0	0	0		

[Table/Fig-8]: Comparison of differential WBC count among controls, smokers, smokeless tobacco users and both smokers and smokeless tobacco users.
Test done: Kruskal Wallis Test; IQR: Inter Quartile Range; χ^2 - Kruskal Wallis Test value; p-value <0.05 statistically significant

Comparison of platelet count		Min-Max	Mean±SD	Median (IQR)	Test value	p-value
PC (lakhs/cu.mm)	Control	1.8-3.0	2.225±.2936	2.2 (0.4)	χ^2= 4.267	0.234
	Smokers	2.0-3.0	2.370±.3389	2.3 (0.65)		
	Chewers	2.0-3.0	2.420±.3172	2.5 (0.4)		
	Both	2.0-3.0	2.340±.3185	2.4 (0.6)		

[Table/Fig-9]: Comparison of platelet count among controls, smokers, smokeless tobacco users and both smokers and smokeless tobacco users.
Test done: Kruskal Wallis Test; IQR: Inter Quartile Range; χ^2 - Kruskal Wallis Test value; p-value <0.05 statistically significant

RBC	Mean difference	95% CI	Test value z-score	p-value
Control vs Smokers	0.060	-0.144 to 0.264	-1.087	0.277
Control vs Chewers	0.175	-0.029 to 0.379	-1.909	0.056
Control vs Both	-0.035	-0.239 to 0.169	-0.975	0.329
Smokers vs Chewers	0.115	-0.089 to 0.319	-0.790	0.429
Smokers vs Both	-0.095	-0.299 to 0.109	-1.923	0.055
Chewers vs Both	-0.210*	-0.414 to -0.006	-2.752	0.006*

[Table/Fig-10]: Post-hoc analysis for significant result of RBC.
Test applied: Mann Whitney u; CI: Class Interval; Z-score - Mann Whitney u test score; p-value <0.05 statistically significant

Eosinophil	Mean difference	95% CI	Test value z score	p-value
Control vs Smokers	0.450	-1.22 to 2.12	-0.981	0.327
Control vs Chewers	-1.200	-2.87 to 0.47	-1.507	0.132
Control vs Both	-0.650	-2.32 to 1.02	-1.120	0.263
Smokers vs Chewers	-1.650	-3.32 to 0.02	-2.645	0.008*
Smokers vs Both	-1.100	-2.77 to 0.57	-2.045	0.041*
Chewers vs Both	0.550	-1.12 to 2.22	-0.328	0.743

[Table/Fig-11]: Post-hoc Analysis for significant result of Eosinophil.
Test applied : Mann Whitney u; CI: Class Interval; Z score - Mann Whitney u test score; p-value <0.05 statistically significant

PCV	Mean difference	95% CI	Test value z score	p-value
Control vs Smokers	0.950	-1.66 to 3.56	-1.637	0.102
Control vs Chewers	2.100	-0.51 to 4.71	-2.303	0.021*
Control vs Both	-0.300	-2.91 to 2.31	-0.123	0.902
Smokers vs Chewers	1.150	-1.46 to 3.76	-0.797	0.425

Smokers vs Both	-1.250	-3.86 to 1.36	-1.825	0.068
Chewers vs Both	-2.400	-5.01 to 0.21	-2.496	0.013*

[Table/Fig-12]: Post-hoc analysis for significant result of PCV.
Test applied : Mann Whitney u; CI: Class Interval; Z score - Mann Whitney u test score; p-value <0.05 statistically significant

accordance with the study done by Shukla AK et al., [3] in chewers. Whereas most of the studies showed an increase in the RBC count [2,13]. In the present study, the individuals with both smoking and chewing habits showed an increase in RBC count. This combined group result could not be compared, as there are no studies in the literature.

The Hb (g/dL) was slightly decreased in the smokers and chewers but increased in both habits, which was in accordance with the study done by Shukla AK et al., [3] in chewers and the study done by Lakshmi SV [14] in smokers. There are no studies done to compare both habits.

In the present study, PCV was decreased in smokers and in chewers and increased in patients with both the habit, study done by Shukla AK et al [3], Metin K et al., on chewers also showed the same result as in the present study [15]. This was in contrast with the study by Mukherjee R and Chatterjee A [16].

In the present study, there was no difference in MCV, MCH and MCHC in all the groups, whereas the previous studies show either an increase, a decrease or no difference in these parameters [3-5,7,13,17].

The total WBC count was decreased in the smokers in present study which was similar to the study done by Bashir BA et al., [17] and was in contrast to the study done by Mufarah MA et al., [4] and Maja Malenica 1 et al., [5]. In the present study, the total WBC count was increased in the chewers, which was in accordance with the study done by Shukla AK et al., Kumar D et al., Memon SM et al., and Jaganmohan P et al., In patients with both habits, there was an increase in the total WBC count [2,3,18-20].

According to some experts, the release of catecholamines and steroid hormones from the adrenal gland's core caused by nicotine may also result in an increase in leukocytes. Leukocyte counts are known to rise in response to increases in the levels of some endogenous hormones, including cortisol and adrenaline [21]. Leukocyte counts can also increase as a result of the irritating effects of tobacco smoke on the respiratory system. Inflammatory stimulation of the respiratory system has been shown to enhance circulating inflammatory markers, particularly cytokines, which impact leukocyte counts [5].

In the present study, the neutrophil count was higher in smokers and showed no change in chewers and in patients with both habits which was similar to the study done by Ahmed A et al., Lakshmi SV Ahmed OA et al., [7,14,22]. A study conducted by Kumar D et al., showed increased neutrophil count in chewers, which was in contrast with the present study; some studies also showed no significant difference in neutrophil count [2,5].

The increased neutrophil count may be due to the ongoing inflammation. Neutrophils produce cytotoxic substances that affect lung function [11].

In the present study, the lymphocyte count was lower in the three tobacco-use groups than in the control group, in accordance with previous studies [2,13,14,16,21]. The sympathetic nervous system is more active when there is systemic stress. Consequently, this increases cortisol output, which is linked to a drop in the percentage of blood lymphocytes [11]. Eosinophils were decreased in smokers and increased in chewers and both habit patients in our study, which was similar to the study done by Badea M et al., but in contrast with the study conducted by Lakshmi SV [8,14]. There was no change in the Basophil and monocyte counts in this study, which was in line with the study done by Badea M et al., [8]. In the present study, platelet count was increased in all smokers, chewers and individuals having both habits, which was in accordance with the study done by Shakiba E et al., and Ahmed OA et al., but in contrast with the study conducted by Kumar D et al., in chewers [2,13,22].

The null hypothesis was rejected for selected haematological parameters showing statistically significant between-group differences; however, the cross-sectional design precludes causal inference. In summary, the current study revealed that group 3 (individuals with both smoking and chewing tobacco habit) had increased RBC and Hb% when compared to a single form of tobacco, which suggests that participants reporting both forms of tobacco use showed higher values for selected haematological parameters in the present sample. Some of the parameters, such as Neutrophils, lymphocytes and monocytes, were not significant, but the marginal value close to significance implies that habit-related haematological variations cannot be ruled out. The significant p-value in RBC and eosinophils strengthens the evidence for habit-related haematological alteration.

Further studies on dosage of consumption as a contributing factor for malignancy can fill the gap in knowledge. Adequate dissemination of knowledge and adverse effects of tobacco, with early intervention for cessation of tobacco, is required to reverse these pathways of causing serious health hazards.

Limitation(s)

The present study was a single-centred, cross-sectional design with small sample size. Self-reported bias on tobacco usage may occur along with gender and age imbalance, which can be confounders for the study.

CONCLUSION(S)

In this cross-sectional study, participants reporting both smoking and smokeless tobacco use showed higher descriptive values for selected haematological parameters, including RBC count, haemoglobin, PCV, eosinophil and platelet counts. However, statistically significant differences were observed only for selected parameters, and the findings should be interpreted cautiously because of potential confounding, the predominantly male sample and the cross-sectional design. Larger, adequately powered longitudinal studies with standardised tobacco-exposure assessment and adjustment for relevant confounders are warranted.

REFERENCES

[1] Coelho KR. Challenges of the oral cancer burden in India. J Cancer Epidemiol. 2012;2012:701932.
[2] Kumar D, Binawara BK, Beniwal P, Sharma P. Effect of chewing tobacco on hematological parameters in Bikaner city population. J Med Sci Clin Res 2017;5(2):17721-27.
[3] Shukla AK, Khaitan T, Gupta P, Naik SR. Smokeless tobacco and its adverse effects on hematological parameters: A cross-sectional study. Adv Prev Med. 2019;2019:3182946.
[4] Mufarah MA, Alharbi NO, Aqeeli AY, Bokhedher RF, Mohsen ZA. Consequence of smoking on haematological parameters in apparently healthy medical students. I P Int J Med Microbiol and Trop Dis. 2022;8(4):288-93.
[5] 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):132-36.
[6] Nargish S, Kabir MH, Akhter K, Nahid KA, Hossain MM, Karmakar P, et al. Effect of Smoking on the Red Blood Cell Count, Haemoglobin Concentration, Hematocrit and Red Cell Indices in Adult Male Smokers. Eastern Med Coll J. 2022;7(1):01-05.
[7] Ahmed IA, Mohammed MA, Hassan HM, Ali IA. Relationship between tobacco smoking and haematological indices among Sudanese smokers. J Health Popul Nutr. 2024;43(1):5.
[8] Badea M, Gaman L, Delia C, Ilea A, Leas F, Henríquez-Hernández LA. Trends of lipophilic, antioxidant and haematological parameters associated with conventional and electronic smoking habits in middle-age Romanian's. J Clin Med. 2019;8:665.
[9] Kim HY. Statistical notes for clinical researchers: Sample size calculation 3. Comparison of several means using oneway ANOVA. Restor Dent Endod 2016;41(3):231-34.
[10] Bain BJ, Bates I, Laffan MA. Dacie and Lewis Practical Haematology. 12th ed. Philadelphia: Elsevier; 2017.

[11] Okpokam DC, Akwiwu EC, Egong SM, Ofem PE. Outcome of red and white blood cell indices of smokeless tobacco (snuff) consumers in Nigeria. J Med Sci. 2022;22:196-202.
[12] Chandra P, Govindraju P. Prevalence of oral mucosal lesions among tobacco users. Oral Health Prev Dent. 2012;10:149-53.
[13] Shakiba E, Moradinazar M, Rahimi Z, Najafi F, Pasdar Y Kohsari M. Tobacco smoking and blood parameters in the Kurdish population of Iran. BMC Cardiovasc Disord. 2023;23:401.
[14] Lakshmi SV. Comparative study of haematological profile among smokers and non-smokers in rural part of South India. Int Arch Integr Med. 2018;5(9):34-38.
[15] Erdogar MK, Lihami O, Fatma Y, Ergul I, BK. The investigation of the effect of Maras's powder (smokeless tobacco) on haematological parameters. Turk J Hematol. 2004;21(3):131-36.
[16] Mukherjee R, Chatterjee A. Assessment of the effects of smoking and consuming gutka (smokeless tobacco) on selected haematological and biochemical parameters: A study on healthy adult males of Hazaribag, Jharkhand. Int J Pharm Chem Biol Sci. 2013;3(4):1172-78.
[17] Bashir BA, Gibreel MO, Abdalatif HM, Mohamed MA, Ahmed EA, Mohamed MS, et al. Impact of tobacco cigarette smoking on hematologic parameters among male subjects in port Sudan Ahlia College, Sudan.Sch J App Med Sci. 2016;4(4A):1124-28.
[18] Kumar D, Jaiprakash, Dubey P, Sharma M. Impact of smokeless tobacco on red cell indices in southern Rajasthan population. Int J Acad Med Pharm. 2024;6(2):921-25.
[19] Memon SM, Kumar N, Atta-Ur-Rahman A, Syed BM. Evaluation of C-reactive protein and haematological parameters in smokeless tobacco users: A comparative cross-sectional study. Pak J Med Sci. 2021;37(4):983-87.
[20] Jaganmohan P, Sarma AP. Studies on changes in haematological and biochemical parameters in smokeless tobacco (Gutka) chewing auto drivers in Nellore district of Andhra Pradesh, India. J Appl Nat Sci. 2011;3(1):106-07.
[21] Kapoor D, Jones TH. Smoking and hormones in health and endocrine disorders. Eur J Endocrinol. 2005;152(4):491-99.
[22] Ahmed OA, Ameen BMM, Mawlood RZ. Assessment of hematological parameters of young male with hookah smoking in Rania City. Mosul J Nurs. 2016;4(1):49-55.

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