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ANALYSIS OF HEMATOLOGICAL PARAMETERS IN SMOKERS VS NON-SMOKERS IN KASHMIR VALLEY

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ABSTRACT

Tobacco use is a persistent global health challenge, yet the haematological effects of different smoking modalities in high-altitude, resource-limited settings remain underexplored. We conducted a cross-sectional study in Budgam, Kashmir (15 January–15 June 2025) enrolling 120 adults (62 smokers; 58 non-smokers) to compare complete blood count (CBC) indices and modality-specific patterns. Venous blood was analysed under stringent quality control; smokers were stratified as cigarette, hookah, or mixed users. Primary outcomes included haemoglobin (Hb), red blood cell (RBC) count, haematocrit (Hct), platelet count, and white blood cell (WBC) count. Group differences were tested with Welch's t-tests and effect sizes; subgroup, duration, and multivariable models adjusted for age, sex, and modality. Smokers showed higher Hb (+2.64 g/dL; 95% CI 2.16–3.12) and RBC (+0.58×10^6/μL; 95% CI 0.42–0.74), borderline higher Hct, and modest platelet suppression (−24.3×10^3/μL; 95% CI −46.6 to −2.0). WBC differences were non-significant after excluding acute infections. Modality analyses suggested greater erythrocytosis in mixed users and higher Hb among cigarette versus hookah users. Findings support hypoxia-driven erythropoiesis with small but consistent platelet reduction, emphasizing the need to disaggregate product types in research and tobacco control. CBC indices may serve as low-cost biomarkers to flag smoking-related harm in low- and middle- income, high-altitude populations.

KEYWORDS: Erythrocytosis; Hematologic Diseases; Hookah; Kashmir; Platelet Count; Tobacco Smoking.

INTRODUCTION

Tobacco smoking remains one of the most significant modifiable risk factors for morbidity and mortality worldwide. Beyond its respiratory and cardiovascular effects, smoking perturbs hematologic and hemostatic balance via hypoxia-inducible pathways, endothelial dysfunction, immune activation, and oxidative stress.[1-2] These mechanisms manifest as erythrocytosis, platelet alterations, and leukocyte variability that function as biomarkers of exposure and mediators of vascular risk.[3] Clinical reviews also emphasize contributions to pro-thrombotic phenotypes and cardiovascular morbidity.[2]

Evidence across product types is growing: waterpipe smoking is linked to increased stroke risk and broad adverse outcomes, while heated tobacco products show shifts in biomarkers of inflammation, oxidative stress, and coagulation.[4-6] Early post-cessation windows still demonstrate altered platelet reactivity, coagulation, and endothelial markers.[7]

Generalizability to Himalayan settings is limited because most comparative studies were conducted in low-altitude or Western populations; altitude and hypobaric hypoxia elevate baseline hemoglobin and hematocrit and can modify hematologic responses.[8-9] These gaps, along with potential confounding from seasonal respiratory

infections, motivate context- specific analyses.

Accordingly, we investigated hematological parameters among smokers and non-smokers in Budgam, Kashmir, and compared modality-specific patterns (cigarette, hookah, mixed). Our goal was to generate data that refine laboratory interpretation, assist differential diagnosis of erythrocytosis or thrombocytopenia, and inform locally relevant tobacco-control strategies in high-altitude, resource-limited settings.

Step 4: Material and methods Study design and setting

We conducted a cross-sectional comparative study in Budgam district, Kashmir, from 15 January 2025 to 15 June 2025, a window chosen to reduce winter-season clustering of respiratory infections and indoor tobacco exposure. Recruitment occurred at the district hospital hematology laboratory and affiliated primary care units, capturing peri-urban and rural participants.

Participants

Eligibility. Adults (≥18 years), residing in Budgam, who provided written informed consent were eligible.

Definitions. "Smokers" reported current use of combustible tobacco (cigarette, hookah, or both) within the preceding 30 days, consistent with Global Adult Tobacco Survey (GATS) criteria.[10] "Non-smokers" reported no current tobacco use in that period.

Exclusion criteria

We excluded participants with factors known to distort hematologic/coagulation indices: pregnancy; blood transfusion within 3 months; ongoing anticoagulant/antiplatelet therapy; hematologic malignancy or marrow failure; recent trauma or surgery (<4 weeks); or pre- analytical errors (clotted, hemolyzed, or improperly filled samples). Individuals with febrile respiratory infections were enrolled but flagged for sensitivity analyses.

Data collection

We recorded demographics (age, sex), smoking history (product type, frequency, duration, pack-years where available), comorbidities, and acute respiratory symptoms using a structured case-report form. Anthropometrics were obtained when feasible.

Laboratory procedures

Venipuncture and CBC. Venous blood was collected under aseptic conditions following CLSI standards for diagnostic venipuncture.[11] Complete blood counts (CBC) were run on an automated analyzer (3/5-part differential), reporting RBC, hemoglobin (Hb), hematocrit (Hct), WBC, platelets, and red-cell indices (MCV, MCH, MCHC).

Coagulation. PT and APTT were planned on calibrated coagulometers; valid coagulation data could not be captured due to a temporary reagent supply interruption during the study window.

Quality control

Daily internal quality control (IQC) used tri-level commercial controls, applying Westgard multi-rule acceptance; targets included ≤2% CV for Hb and ≤7% for platelets. External quality assurance (EQA) was maintained through participation in a national proficiency-testing program; weekly maintenance logs were reviewed by supervisors.[12]

Sample size and power

We enrolled **120 participants**: 62 smokers (40 cigarette-only, 19 hookah-only, 3 mixed) and 58 non-smokers. The pragmatic sample reflected seasonal throughput; post-hoc power exceeded 90% to detect ≥1.5 g/dL Hb difference at $\alpha = 0.05$, given observed variance.

Statistical analysis

Continuous variables are mean ± SD; categorical variables are counts (%). Primary comparisons (smokers vs non-smokers) used Welch's t-tests with 95% CIs and Cohen's d. Subgroup comparisons across modalities used one-way ANOVA with Bonferroni–Holm post- hoc adjustment. Linear regression and non-parametric correlations assessed duration-response; logistic regression estimated odds of thrombocytopenia ($<150\times10^3/\mu L$) by product type and duration. Sensitivity analyses excluded participants flagged for acute respiratory infection. Analyses were conducted in **R 4.3.1** with two-sided $p<0.05$.

RESULTS AND DISCUSSION

Baseline characteristics

The study included 120 participants: 62 smokers (40 cigarette-only, 19 hookah-only, 3 mixed) and 58 non-smokers. Mean age was comparable between groups (44.3 ± 13.8 vs 39.9 ± 13.7 years, $p = 0.080$). Male predominance was higher among smokers (90.3%) than non-smokers (37.9%; $p < 0.001$). This sex imbalance necessitated sensitivity analyses adjusting for gender (Table 1).

Table 1: Baseline characteristics of study participants by smoking status: Demographic distribution of smokers (n=62) and non-smokers (n=58). Values are mean ± standard deviation (SD) or number (percentage). *p*-values calculated using Welch's *t*-test for continuous variables and χ^2 test for categorical variables.

Characteristic	Non-smokers (n=58)	Smokers (n=62)	*p*-value
Age (years)	39.9 ± 13.7	44.3 ± 13.8	0.080
Male, n (%)	22 (37.9)	56 (90.3)	<0.001
Female, n (%)	36 (62.1)	6 (9.7)	—

Hematologic outcomes

Smokers exhibited significantly elevated red cell indices: hemoglobin (+2.64 g/dL), RBC (+0.58×10^6/μL), with borderline higher hematocrit. Platelets were modestly but significantly lower (−24.3×10^3/μL). WBC counts showed no significant differences after excluding participants with infection. These contrasts are summarized in Table 2, while robustness checks are shown in Table 3. Figure 1 graphically illustrates the differences in hemoglobin, RBC, and platelet counts between smokers and non-smokers, reinforcing the tabulated findings.

Table 2: Group contrasts between smokers and non-smokers (primary and sensitivity analyses): Primary and sensitivity analyses of complete blood count (CBC) indices. Values are mean ± SD. Differences shown as mean difference (smokers − non-smokers) with 95% confidence intervals (CI), *p*-values from Welch's *t*-test, and Cohen's *d* effect size.

Endpoint	Non-smokers (mean±SD)	Smokers (mean±SD)	Mean difference (SM-NS)	95% CI	Welch t (df≈)	p-value	Cohend
RBC (×10^6/μL)	4.17±0.48 (n=58)	4.75±0.41 (n=62)	0.58	[0.42, 0.74]	7.09 (112.4)	1.23e10	1.30
Hemoglobi n (g/dL)	12.24±1.40 (n=58)	14.88±1.25 (n=62)	2.64	[2.16, 3.12]	10.87 (114.3)	0	1.99
WBC (×10^3/μL) Primary	6.25±2.40 (n=58)	6.87±3.00 (n=62)	0.62	[-0.36, 1.60]	1.25 (115.3)	0.212	0.23
WBC (×10^3/μL) Sensitivity	6.25±2.40 (n=58)	6.55±2.63 (n=54)	0.30	[-0.65, 1.25]	0.63 (107.2)	0.531	0.12
Platelets (×10^3/μL) Primary	162.19±62.48 (n=58)	137.90±60.64 (n=62)	-24.29	[-46.57, -2.01]	-2.16 (116.9)	0.0329	-0.39
Platelets (×10^3/μL) Sensitivity	162.19±62.48 (n=58)	137.81±59.92 (n=54)	-24.38	[-47.30,-1.46]	-2.11 (109.9)	0.0373	-0.40
Hematocrit (%)	36.17±4.30 (n=3)	45.09±4.25 (n=25)	8.92	[-0.48,18.32]	3.40 (2.5)	0.056	2.10

Table 3: Sensitivity analyses for hematological outcomes: Primary contrasts versus analyses excluding participants with acute infection or hematologic disease. Values are mean differences (smokers − non-smokers) with corresponding *p*-values.

Endpoint	Primary Δ (SM-NS)	p (Primary)	Sensitivity Δ	p (Sensitivity)
RBC (×10^6/μL)	0.58	1.35e−10	0.6	1.13e−10
Hemoglobin (g/dL)	2.63	3.09e−19	2.68	3.38e−19
Hematocrit (%)	8.92	0.056	9.2	0.052
WBC (×10^3/μL)	0.62	0.211	0.3	0.531
Platelets (×10^3/μL)	-24.29	0.033	-24.37	0.037

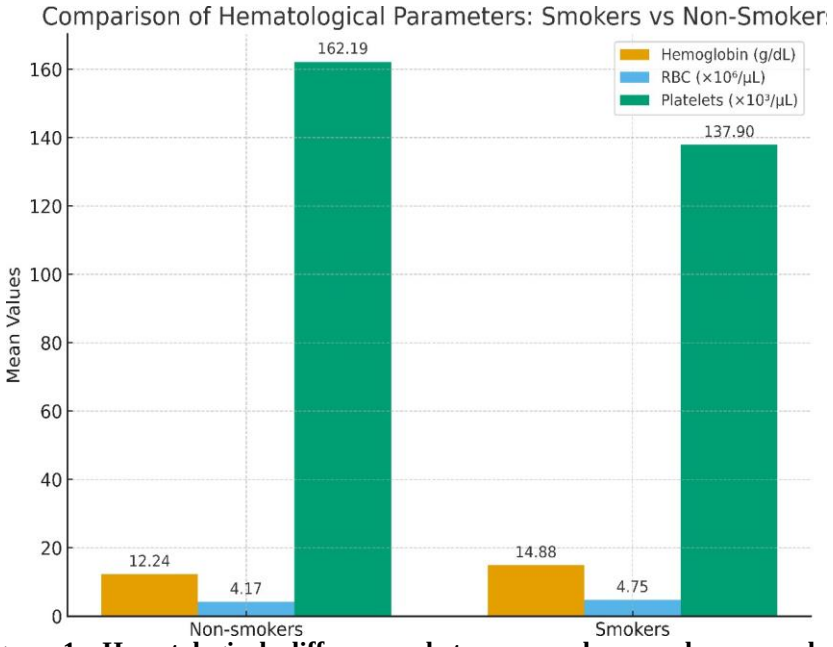

Figure 1: Hematological differences between smokers and non-smokers.

Group mean values for hemoglobin (g/dL), red blood cell count (×10^6/µL), and platelet count (×10^3/µL). Bars represent group means with numeric values shown above; smokers demonstrated significantly higher hemoglobin and RBC counts, and lower platelet counts compared with non-smokers**.**

Modality-specific differences

Among smokers, mixed users showed the highest hemoglobin and hematocrit, followed by cigarette-only, while hookah-only exhibited lower levels. Platelets tended to be lower among hookah and mixed users, though not statistically significant. These differences are presented in Table 4, highlighting the importance of disaggregating product types in analysis.

Table 4: Hematological parameters among smokers by product type: Comparison of cigarette-only, hookah-only, and mixed-product users. Values are mean ± SD. One-way ANOVA was used for group comparisons with Bonferroni–Holm post-hoc correction.

Parameter	Cigarette (n=40)	Hookah (n=19)	Mixed (n=3)	ANOVA p	Notes
RBC (×10^6/µL)	4.75	4.68	5.06	0.353	—
Hemoglobin (g/dL)	15.06	14.34	15.77	0.047	Cigarette > Hookah (post-hoc ~0.052)
Hematocrit (%)	45.08	42.88	51.75	0.030	Elevated in mixed (small n)
WBC (×10^3/µL)	6.55	7.47	7.31	0.539	—
Platelets (×10^3/µL)	146.48	124.53	108.33	0.300	—

Duration-response patterns

When smokers were stratified by duration (<5, 5–10, 11–20, >20 years), a non-linear upward trend in hematocrit was observed, although not statistically conclusive. Hemoglobin, RBC, and platelet differences did not show clear duration dependence. Detailed results are presented in Table 5. This observation is consistent with earlier systematic reviews describing variable time- dose responses in tobacco-related hematologic alterations.[13]

Table 5: Hematological parameters by duration of smoking exposure Smokers stratified into categories of <5, 5–10, 11–20, and >20 years of tobacco use. Values are mean ± SD. *p* for linear trend derived from regression analysis.

Duration (years)	RBC (×10^6/µL)	Hemoglobin (g/dL)	Hematocrit (%)	WBC (×10^3/µL)	Platelets (×10^3/µL)
0–5 (n=6)	4.84	15.25	40.2	6.1	122.5
6–10 (n=8)	4.78	14.51	42.93	7.75	183.5
11–20 (n=26)	4.67	14.73	44.84	6.72	138.77
>20 (n=22)	4.8	15.07	46.5	6.95	124.5

Regression analyses

Multivariable regression confirmed that smoking modality independently predicted hematocrit elevation, even after adjustment for age, sex, and duration.

Hemoglobin trends were less robust. These associations are summarized in Table 6.

Table 6: Multivariable regression analysis among smokers

Regression coefficients (β) with standard error (SE) and 95% CI for hemoglobin (Hb, g/dL) and hematocrit (Hct, %) as dependent variables. Predictors include duration of smoking, product type, age, and sex.

Outcome	Predictor	β (SE)	95% CI	p
Hb (g/dL)	Duration (per yr)	+0.05 (0.03)	−0.02, 0.11	0.133
Hb (g/dL)	Hookah vs Cigarette	−0.40 (0.45)	−1.29, 0.50	0.379
Hb (g/dL)	Mixed vs Cigarette	+0.78 (0.75)	−0.72, 2.28	0.303
Hb (g/dL)	Age (per yr)	−0.03 (0.02)	−0.08, 0.02	0.234
Hb (g/dL)	Male vs Female	+0.71 (0.59)	−0.48, 1.89	0.237
Hct (%)	Duration (per yr)	−0.26 (0.23)	−0.74, 0.23	0.279
Hct (%)	Hookah vs Cigarette	−4.07 (2.37)	−9.03, 0.89	0.102
Hct (%)	Mixed vs Cigarette	+6.08 (2.83)	0.16, 12.00	0.045
Hct (%)	Age (per yr)	+0.29 (0.17)	−0.06, 0.65	0.095
Hct (%)	Male vs Female	+2.96 (3.32)	−3.98, 9.90	0.384

INTEGRATED DISCUSSION

Our findings demonstrate a robust hematologic footprint of tobacco smoking, consistent with compensatory erythrocytosis driven by hypoxia-inducible pathways.[14-16] Platelet suppression was modest but consistent, aligning with studies that implicate nicotine-related marrow toxicity and altered megakaryopoiesis.[17-18] The absence of sustained leukocytosis after controlling for infections challenges its utility as a universal biomarker of exposure.[3,5]

Modality analysis showed that mixed users had the greatest erythrocytosis and higher hematocrit, corroborating evidence that combined exposure amplifies physiologic stress.[4-6,14] Duration analyses suggested a trend but were limited by sample size, in line with earlier reports of variable time-dose responses.[19]

Globally, the implications extend beyond Kashmir: waterpipe use is expanding in the Middle East and Africa, particularly among youth.[20-22] Integrating low-cost CBC indices into tobacco surveillance could enhance early detection in low- and middle-income countries, where advanced biomarker platforms remain inaccessible.[23]

CONCLUSION

This study provides district-level evidence that tobacco smoking in Kashmir is associated with significant hematological alterations. Smokers exhibited elevated hemoglobin, hematocrit, and red blood cell counts, reflecting hypoxia-driven erythropoiesis, while platelet counts were modestly suppressed. Leukocyte differences largely disappeared after excluding participants with acute infections, underscoring the importance of controlling for seasonal confounding.

Modality-specific analysis revealed amplified erythrocytosis among mixed-product users and higher hemoglobin in cigarette users compared with hookah smokers, emphasizing the need to disaggregate tobacco products in research and policy frameworks. These findings validate the use of complete blood count indices as accessible, low-cost biomarkers for early detection of smoking-related harm, particularly in high-altitude, resource-limited settings.

Policy implications extend beyond India: with hookah use rising globally, particularly among youth in the Middle East and Africa, public health strategies must explicitly incorporate non- cigarette modalities into cessation and harm-reduction programs. Future longitudinal studies should integrate coagulation, inflammatory, and endothelial markers to refine risk prediction and guide preventive interventions.

Declarations

- **Ethics approval and consent to participate:** The study protocol was reviewed and approved by the Medical Ethics Committee of Sub-District Hospital, Budgam, Kashmir. Written informed consent was obtained from all participants prior to enrollment.
- **Consent for publication:** Not applicable.
- **Funding source:** This research did not receive any external funding. All study activities were supported by departmental resources.
- **Conflict of interest:** The authors declare no competing interests, financial or non- financial, related to this work.
- **Acknowledgment:** The authors gratefully acknowledge the technical staff of Budgam District Hospital for laboratory support and quality assurance. We also thank the study participants for their cooperation and commitment.
- **Data availability:** De-identified participant data and statistical code are available from the corresponding author upon reasonable request.

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