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The Effect of Electronic Cigarettes Compared to Regular Smoking on Some Blood Characteristics and Kidney Function

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ABSTRACT: Background: Cigarette smoking remains a leading preventable cause of morbidity, while electronic cigarettes are promoted as safer alternatives despite limited comparative evidence.

Objective: To compare the effects of conventional and electronic cigarette use on hematological parameters, lipid profile, and kidney function.

Methods: Sixty participants formed three groups: cigarette smokers, electronic cigarette users (vapers), and healthy controls (n = 20 per group). Complete blood count, cholesterol, triglycerides, urea, and creatinine were measured and analyzed by one-way ANOVA (P < 0.05).

Results: Groups were age-matched (P = 0.307). RBC, hemoglobin, and platelet counts were significantly higher in both smoking groups than controls; vapers had the highest RBC (6.20 ± 0.08). Cholesterol (274.85 ± 20.16 mg/dL) and triglycerides (201.80 ± 5.42 mg/dL) peaked in vapers, whereas urea and creatinine were highest in smokers (40.80 and 1.62 mg/dL).

Conclusion: Electronic cigarettes are not physiologically inert, inducing hematological, lipid, and renal alterations comparable to conventional smoking.

1. INTRODUCTION

The worldwide epidemic of smoking-related diseases causes about eight million deaths annually [1]. Traditional cigarette smoking is significantly linked to cardiovascular diseases, respiratory dysfunction, and chronic kidney disease (CKD) progression [2]. Essential constituents—including nicotine, carbon monoxide, heavy metals, and reactive oxygen species—are swiftly absorbed into the bloodstream, producing systemic oxidative stress, inflammation, endothelial dysfunction, and cellular damage that ultimately compromise organ performance.

In the circulatory system, tobacco smoke's carbon monoxide causes tissue hypoxia, rising hemoglobin and hematocrit levels through enhanced erythropoiesis [3]. Smoking also increases white blood cell counts, showing a chronic inflammatory response, and enhances platelet activation [4]. Renally, toxins harm tissues directly or indirectly. Nicotine stimulates the sympathetic nervous system, resulting in elevated blood pressure and renal vasoconstriction, which impairs glomerular filtration [5] and speeds up nephropathy progression toward renal failure [6].

Recently, electronic cigarettes have grown in popularity as a combustion-free alternative perceived to be safer [7]. However, scientific studies reveal that e-cigarette aerosols contain harmful contaminants, such as heavy metals, volatile organic

compounds, and cytotoxic flavorings [8]. These compounds mimic traditional smoke by triggering oxidative stress and inflammatory responses that can alter hematopoiesis, modify white blood cell counts, and damage red blood cell integrity [9].

Similarly, vaping-delivered nicotine induces renal vasoconstriction, reducing blood flow, while aerosol exposure generates inflammatory mediators that damage renal tissues [10]. Although long-term data remain limited, comparing the effects of electronic and traditional cigarettes on blood parameters and renal function is crucial. Utilizing these physiological measurements as biomarkers helps detect systemic toxicity before clinical symptoms appear, ultimately guiding public health policy, clinical practice, and user awareness.

Taken together, the literature establishes the harms of combustible tobacco with reasonable certainty but leaves the comparative position of electronic cigarettes unresolved, particularly when hematological, lipid, and renal outcomes are examined simultaneously in the same participants. The present study was therefore designed to address this gap by comparing the three domains concurrently in conventional smokers, e-cigarette users, and healthy non-smoking controls.

Aims of the Study

1. **Quantification:** To measure and evaluate specific blood characteristics and kidney function parameters within the study groups.
2. **Comparative Analysis:** To compare the physiological effects of electronic cigarette usage directly against regular tobacco smoking.
3. **Control Evaluation:** To assess how much both smoking methods deviate from normal baseline values (healthy controls).
4. **Impact Assessment:** To determine the severity of biological alterations caused by e-cigarettes on hematological profiles and renal health.

2. MATERIALS AND METHODS

2.1 Study Design and Participants

This comparative cross-sectional study was conducted on 60 adult male volunteers, who were allocated to three equal groups according to their nicotine-exposure status: conventional cigarette smokers ($n = 20$), with a habit of at least five cigarettes per day for a minimum of two consecutive years; exclusive electronic cigarette users (vapers, $n = 20$), with daily use for at least six months and no history of conventional smoking; and healthy non-smoking controls ($n = 20$), with no history of tobacco or electronic cigarette use. Participants with known cardiovascular disease, diabetes mellitus, renal or hepatic disease, active inflammatory conditions, or any medication liable to influence hematological, lipid, or renal parameters were excluded. All participants gave informed consent before enrolment, and the protocol was approved by the institutional research ethics committee.

2.2 Blood Sample Collection

All participants' blood is drawn in the morning and before breakfast. About (10–15) milliliters of venous blood was drawn from patients and control individuals and left at room temperature for (15) minutes in a gel tube (without anticoagulant), after which serum was separated by centrifugation at (3000 xg) for (15) minutes, divided into (4) aliquots, and stored frozen at (-20°C) until utilized for biochemical determination.

2.3 Estimation of Cholesterol

Principle of the test: Serum cholesterol level was estimated by using the enzymatic method, according to the instruction of the company (Bicon Burbach / Germany), by the following reactions:

Cholesterol esters $\xrightarrow{\text{Chol. Esterase}}$ Cholesterol + free fatty acids

Cholesterol + $\text{O}_2 \xrightarrow{\text{Chol. Oxidase}}$ Cholesten-4-one + H_2O_2

$\text{H}_2\text{O}_2 + \text{Phenol} + 4\text{-aminoantipyrine} \xrightarrow{\text{POD}}$ Quinoneimine + $4\text{H}_2\text{O}$

The intensity of the pink / red color formed is proportional to the cholesterol concentration.

Reagents

Reagents	Composition
Reagent 1 (Buffer solution)	Phosphate buffer
Reagent 2 (Enzymes)	Cholesterol oxidase, cholesterol esterase, peroxidase, 4-aminoantipyrine, cholic acid salt.
Reagent 3 (Standard)	Cholesterol (200 mg / dl)

Procedure

Reagents	Test	Standard	Blank
1 - Serum	10 μ l	—	—
2 - Standard	—	10 μ l	—
3 - Working solution (R1 + R2)	1 ml	1 ml	1 ml
4 - Mixed and incubate for 5 min at 37 °C, then measure the absorbance of test and standard at 500 nm against the blank.			

Calculation

Cholesterol conc. (mg / dl) = [(Test reading – Blank reading) / (Standard reading – Blank reading)] $\times$ 200

2.4 Estimation of Triglyceride

Principle: The concentration of triglyceride was measured by enzymatic colorimetric method supplied by (Bicon Burbach / Germany). Triglyceride enzymatically hydrolyzed to glycerol and free fatty acids by effect of lipoprotein lipase LPL. The hydrogen peroxide formed reacts, under catalysis of glycerol kinase GK, glycerol-3-phosphate oxidase GPO and peroxidase POD, with P-chlorophenol and 4-aminoantipyrine to form quinoneimine dye as indicator. Quinoneimine is a red-colored dye that was measured colorimetrically at 500 nm.

Triglycerides

Glycerol + fatty acids

Glycerol + ATP

Glycerol – 3 – phosphate + ADP

Glycerol – 3 – phosphate + O₂

ADP (Dihydroxyaceton phosphate) + H₂O₂

2H₂O₂ + parachlorophenol + 4-aminoantipyrine

Quinoneimine + 4H₂O

Reagents

Reagents	Composition
Reagent 1 (Buffer)	* p – chlorophenol * Pipes buffer (pH = 7.2)
Reagent 2 (Enzymes)	(LPL, GK, GPO, POD, ATP) 4 - aminoantipyrine
Reagent 3 (Standard)	Glycerol (200 mg / dl)

Procedure

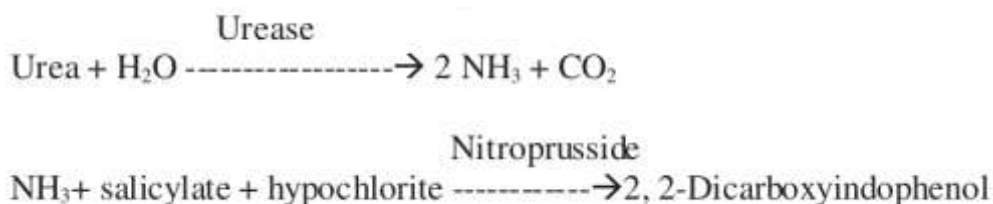
Reagents	Test	Standard	Blank
1 - Serum	10 µl	—	—
2 - Standard	—	10 µl	—
3 - Working solution (R1 + R2)	1 ml	1 ml	1 ml
4 - Mixed and measured after incubation at 37 °C for 5 min, then read the absorbance of test and standard against reagent blank.			

Calculation

Triglycerides conc. (mg / dl) = [(Test reading – Blank reading) / (Standard reading – Blank reading)] × 200

2.5 Estimation of Urea

Principle: Urease catalyses the conversion of urea to ammonia. In a modified Berthelot reaction, the ammonium ions react with a mixture of salicylate, hypochlorite and nitroprusside to yield a blue-green dye (Indophenol). The intensity of this dye is proportional to the concentration of urea in the sample.



PROCEDURE

Pipette into cuvettes	Blank	Standard	Sample
Reagent-1	1000 µL	1000 µL	1000 µL
Sample	--	--	10 µL
Standard	--	10 µL	--
Mix and incubate for 5 minutes at 20-25°C or 3 minutes at 37°C			
Reagent-2	1000 µL	1000 µL	1000 µL
Mix and incubate for 10 minutes at 20-25°C or 5 minutes at 37°C Measure the absorbance of the sample (As) and the standard (Astd) against the reagent blank			

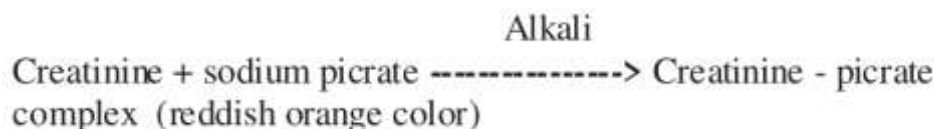
CALCULATION

$$\text{Urea Conc. (mg/dL)} = \frac{\Delta A \text{ sample}}{\Delta A \text{ standard}} \times 80 \text{ (Std.conc.)}$$

Urea (g/24 urine) = mg/dL X volume of 24-hour urine
To convert mg/dL to mmol/L divide by 6.01

2.6 Estimation of Creatinine

Principle: In the Jaffe reaction, Creatinine reacts with alkaline picrate to produce a reddish-orange color, the intensity of which at 520 nm is directly proportional to the Creatinine concentration.



PROCEDURE

Pipette into cuvettes	Blank	Standard	Sample
Distilled Water	1000 μ L	---	---
Standard	---	1000 μ L	---
Sample	---	---	1000 μ L
TCA	1000 μ L	1000 μ L	1000 μ L
Mix well and centrifuge at 5000 rpm X 10 minutes			
supernatant	1000 μ L	1000 μ L	1000 μ L
Working reagent	1000 μ L	1000 μ L	1000 μ L
Mix well and wait exactly 20 minutes after adding working reagent, read absorbance against reagent blank.			

CALCULATION

$$\text{Serum Creatinine (mg/dL)} = \frac{\Delta A \text{ sample}}{\Delta A \text{ standard}} \times 2 (\text{Std.conc.})$$

To convert mg/dL to μ mol/L multiply by 88.4

NORMAL RANGE

Serum Creatinine

Male	0.7-1.4 mg/dL	62-124 μ mol/L
Female	0.7-1.2 mg/dL	62-106 μ mol/L

2.7 Statistical Analysis

All data were processed using the Statistical Package for the Social Sciences (SPSS). Continuous variables are expressed as mean $\pm$ standard error (SE). Differences among the three study groups were examined by one-way analysis of variance (ANOVA), and a probability value of $P < 0.05$ was considered statistically significant.

3. Results

3.1 Demographic Characteristics of Study Groups

The present study included 60 participants, who were divided into three groups according to smoking status: cigarette smokers ($n = 20$), electronic cigarette users (vapers) ($n = 20$), and a healthy control group ($n = 20$). The mean age of the participants in the three groups was compared using One-Way Analysis of Variance (ANOVA). The results showed no significant difference in age among the study groups ($P = 0.307$), indicating that the groups were age-matched, which reduces the potential influence of age as a confounding factor (Table 1).

Table 1: Comparison of Age Among Study Population.

Groups	N (60)	Mean	Std. Error	P-value
Cigarette smokers	20	35.6000	1.11262	0.307
E-Cigarette Users (Vapers)	20	34.6000	1.11262	
Control Group	20	36.000	3.44734	
* <i>P value</i> < 0.05 * <i>Values are expressed as mean ± standard error (SE)</i> * <i>One-Way ANOVA</i>				

3.2 Comparison of Hematological Parameters (CBC) Among Study Groups

The results revealed significant differences in complete blood count (CBC) parameters among the study groups ($P < 0.05$). The mean red blood cell (RBC) count was higher in e-cigarette users (6.20 ± 0.08) and cigarette smokers (5.72 ± 0.09) compared with the control group (4.95 ± 0.13). Hemoglobin (Hb) levels were also elevated in cigarette smokers (16.19 ± 0.27) and e-cigarette users (15.08 ± 0.26) relative to the control group (13.46 ± 0.30).

In contrast, the mean corpuscular volume (MCV) was highest in cigarette smokers (87.03 ± 1.75), followed by the control group (81.60 ± 0.92), while the lowest value was observed in e-cigarette users (78.65 ± 0.83). Regarding white blood cell (WBC) count, the control group showed the highest level (8.38 ± 0.46), followed by e-cigarette users (7.09 ± 0.13) and cigarette smokers (6.35 ± 0.14). Platelet (PLT) counts were higher among e-cigarette users (291.6 ± 13.42) and cigarette smokers (284.7 ± 15.91) compared with the control group (236.3 ± 7.32). These findings indicate that both conventional cigarette smoking and e-cigarette use are associated with significant alterations in hematological parameters (**Table 2**).

Table 2: Comparison of Complete Blood Count (CBC) Parameters Among Study Groups.

CBC Tests	Groups	N	Mean	Std. Error	P value
RBC	Cigarette smokers	20	5.72	.09306	< 0.05
	E-Cigarette Users (Vapers)	20	6.20	.08347	
	Control Group	20	4.95	.13189	
Hb	Cigarette smokers	20	16.19	.27250	< 0.05
	E-Cigarette Users (Vapers)	20	15.08	.25724	
	Control Group	20	13.46	.29624	
MCV	Cigarette smokers	20	87.03	1.75202	< 0.05
	E-Cigarette Users (Vapers)	20	78.65	.83330	
	Control Group	20	81.60	.92463	
WBC	Cigarette smokers	20	6.35	.13582	< 0.05
	E-Cigarette Users (Vapers)	20	7.09	.13208	
	Control Group	20	8.38	.45593	
PLT	Cigarette smokers	20	284.7	15.91013	< 0.05
	E-Cigarette Users (Vapers)	20	291.6	13.42431	
	Control Group	20	236.3	7.32124	
* P value < 0.05 * Values are expressed as mean $\pm$ standard error (SE) * One-Way ANOVA					

Expressed against the control group, the two exposed groups diverge in both magnitude and direction. The red blood cell count was 15.6 % higher in smokers and 25.3 % higher in vapers, whereas hemoglobin followed the reverse order (+20.3 % in smokers against +12.0 % in vapers). Platelet counts rose comparably in both (+20.5 % and +23.4 %, respectively). The mean corpuscular volume separated the groups most sharply: it stood 6.7 % above control in smokers but 3.6 % below control in vapers, the only parameter in the entire panel on which the two exposed groups moved in opposite directions. The total white blood cell count was reduced in both exposed groups (−24.2 % in smokers, −15.4 % in vapers).

3.3 Comparison of Lipid Profile Among Study Groups

The results revealed a significant difference in serum lipid profile parameters among cigarette smokers, e-cigarette users, and the control group ($P < 0.05$). The mean total cholesterol level was highest in e-cigarette users (274.85 ± 20.16 mg/dL), followed by cigarette smokers (244.40 ± 15.61 mg/dL), while the lowest level was observed in the control group (147.85 ± 4.20 mg/dL). Similarly, triglyceride levels were elevated in both e-cigarette users (201.80 ± 5.42 mg/dL) and cigarette smokers (191.75 ± 4.24 mg/dL) compared with the control group (126.95 ± 2.64 mg/dL). These findings indicate that both conventional smoking and electronic cigarette use are associated with significant alterations in lipid profile parameters (**Table 3 and Figure 1**).

Table 3: Comparison of Lipid Profile Parameters Among Study Groups.

Lipid Profile Tests	Groups	N	Mean	Std. Error	P value
Cholesterol	Cigarette smokers	20	244.4000	15.60759	< 0.05
	E-Cigarette Users (Vapers)	20	274.8500	20.16071	
	Control Group	20	147.8500	4.20293	
Triglycerides	Cigarette smokers	20	191.7500	4.23698	< 0.05
	E-Cigarette Users (Vapers)	20	201.8000	5.41713	
	Control Group	20	126.9500	2.64224	
* P value < 0.05 * Values are expressed as mean ± standard error (SE) * One-Way ANOVA					

Relative to the control group, total cholesterol was elevated by 65.3 % in smokers and by 85.9 % in vapers, and triglycerides by 51.0 % and 59.0 %, respectively. On both lipid parameters it is the electronic cigarette group, not the combustible cigarette group, that is displaced furthest from the healthy baseline.

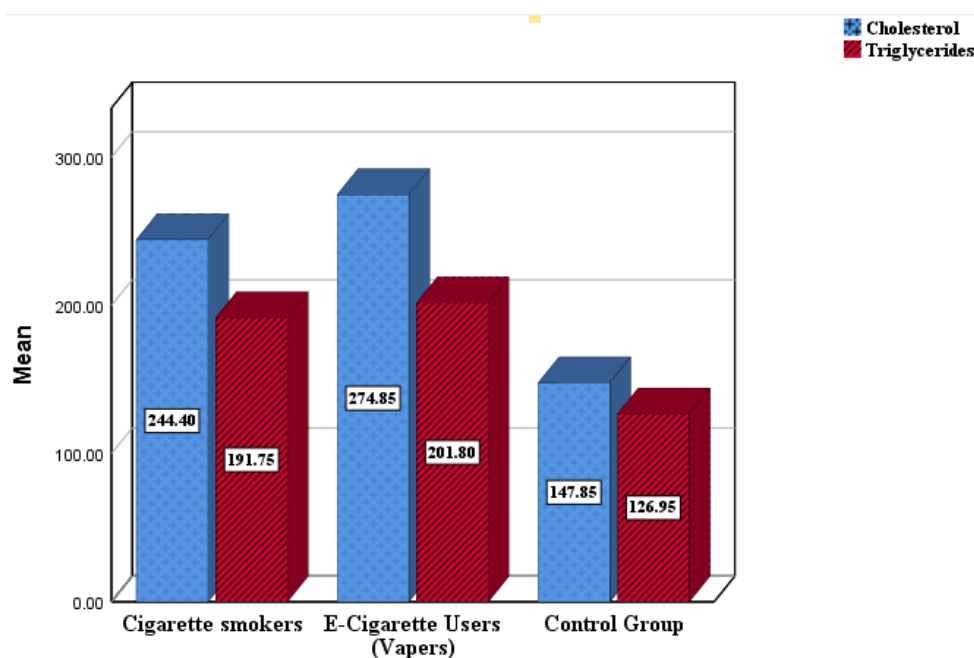


Figure 1: Comparison of Lipid Profile Parameters Among Study Groups.

3.4 Comparison of Kidney Function Parameters Among Study Groups

The results revealed a significant difference in kidney function parameters among the study groups ($P < 0.05$). The mean serum urea level was higher in cigarette smokers (40.80 ± 2.19 mg/dL) and e-cigarette users (39.80 ± 1.07 mg/dL) compared with the control group (33.20 ± 1.58 mg/dL). Similarly, serum creatinine levels were elevated in cigarette smokers (1.62 ± 0.05 mg/dL) and e-cigarette users (1.41 ± 0.06 mg/dL) relative to the control group (0.81 ± 0.05 mg/dL). These findings suggest that both conventional smoking and electronic cigarette use may be associated with alterations in kidney function parameters (**Table 4**, **Figure 2**, and **Figure 3**).

Table 4: Comparison of Kidney Function Parameters Among Study Groups.

Kidney Function Tests	Groups	N	Mean	Std. Error	P value
Urea	Cigarette smokers	20	40.8000	2.19281	< 0.05
	E-Cigarette Users (Vapers)	20	39.8000	1.07263	
	Control Group	20	33.2000	1.58214	
Creatinine	Cigarette smokers	20	1.6200	.04565	< 0.05
	E-Cigarette Users (Vapers)	20	1.4150	.05585	
	Control Group	20	.8150	.05093	

* P value < 0.05 * Values are expressed as mean $\pm$ standard error (SE) * One-Way ANOVA

Relative to the control group, serum urea was raised by 22.9 % in smokers and 19.9 % in vapers, while serum creatinine was raised by 98.8 % and 73.6 %, respectively. The ordering is identical for both renal markers: conventional smokers are displaced furthest, vapers occupy an intermediate position, and both exposed groups sit clearly above the healthy baseline.

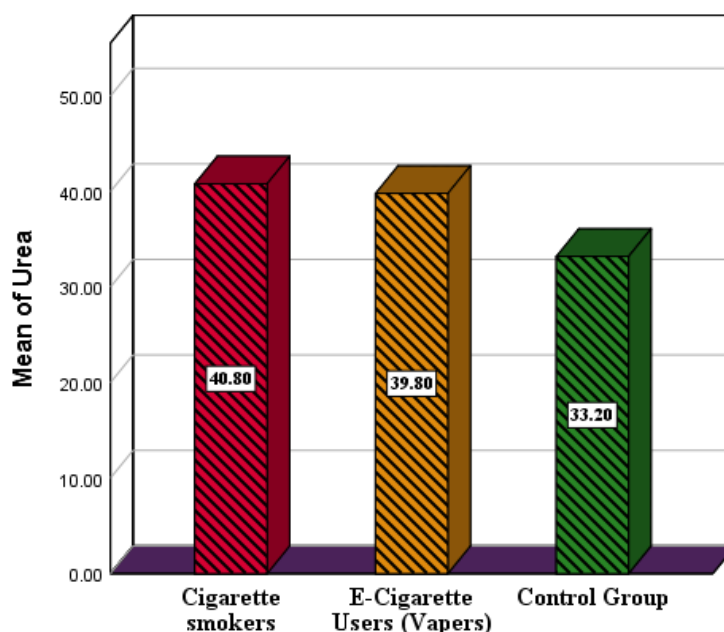


Figure 2: Comparison of Serum Urea Among Study Groups.

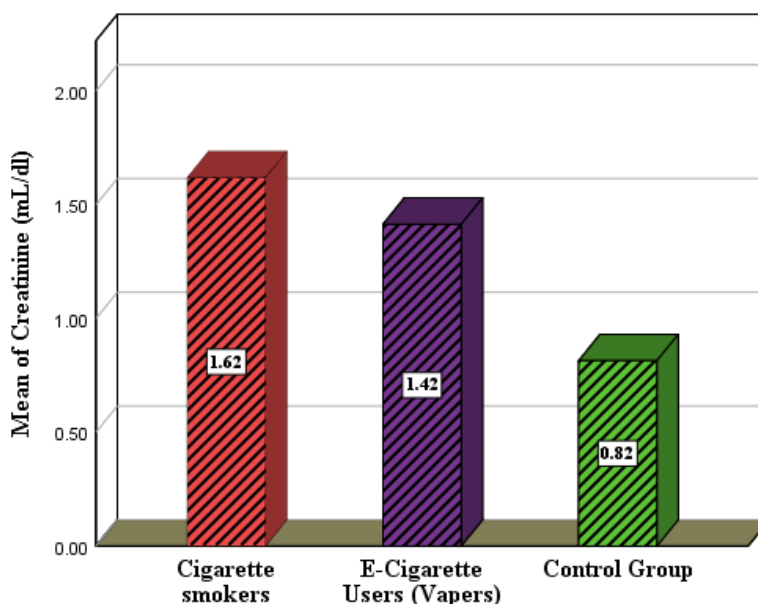


Figure 3: Comparison of Serum Creatinine Among Study Groups.

3.5 Summary of Deviations from the Control Group

Table 5 consolidates the nine measured parameters as percentage deviations from the healthy control group, so that the direction and the magnitude of change in the two exposed groups can be compared side by side. The values are derived arithmetically from the group means already reported in Tables 2 to 4 and introduce no new measurement.

Table 5: Percentage Deviation of Each Exposed Group from the Control Group.

Parameter	Cigarette smokers	E-cigarette users	Pattern
RBC	+15.6 %	+25.3 %	Raised in both; higher in vapers
Hb	+20.3 %	+12.0 %	Raised in both; higher in smokers
MCV	+6.7 %	−3.6 %	Opposite directions
WBC	−24.2 %	−15.4 %	Reduced in both
PLT	+20.5 %	+23.4 %	Raised in both; near-identical
Cholesterol	+65.3 %	+85.9 %	Raised in both; higher in vapers
Triglycerides	+51.0 %	+59.0 %	Raised in both; higher in vapers
Urea	+22.9 %	+19.9 %	Raised in both; higher in smokers
Creatinine	+98.8 %	+73.6 %	Raised in both; higher in smokers

* Percentage deviation of each group mean from the control-group mean, computed from Tables 2–4.

Two features of this summary deserve notice before the findings are interpreted. First, on seven of the nine parameters the two exposed groups move in the same direction, and on the two lipid parameters and on the red blood cell count the electronic cigarette group is displaced further from the baseline than the combustible cigarette group. Second, the largest single deviation in the whole panel is the near-doubling of serum creatinine in conventional smokers. Both observations are internal to the present dataset and are reported here as description, not as a claim about causation.

4. DISCUSSION

The present study set out to compare, in the same participants and under the same laboratory conditions, the hematological, lipid, and renal consequences of conventional cigarette smoking and of electronic cigarette use. Four principal findings emerged. The three groups were successfully matched for age, so that age may be excluded as a confounding factor. Both exposed groups differed significantly from the healthy control group on every parameter measured except age. The electronic cigarette group was displaced further from the baseline than the conventional smoking group on the two lipid parameters and on the red blood cell count. The conventional smoking group, in turn, was displaced further on the two renal parameters. Taken together, these results give no support to the proposition that electronic cigarettes are physiologically inert.

4.1 Age Matching and Internal Validity

The findings of the present study demonstrated no statistically significant difference in age among the three groups ($P = 0.307$), indicating effective age-matching across participants. This baseline homogeneity eliminates age as a confounding variable, thereby enhancing the reliability of the comparative outcomes between traditional smokers, vapers, and controls [11]. This approach aligns with similar comparative cohorts where age-matching is vital to accurately isolate the direct impacts of electronic and conventional smoking [12].

The practical value of this homogeneity is considerable. Age is among the strongest determinants of renal function and of the lipid profile, and an imbalance of even a few years between the groups would have compromised the interpretation of the creatinine, urea, and cholesterol results. Because the age comparison is the only one in the study that did not reach statistical significance ($P = 0.307$, against $P < 0.05$ for every other parameter), the differences reported below cannot be attributed to a difference in the ages of the groups. The remaining sources of confounding are addressed in the limitations.

4.2 Hematological Parameters

The significant elevations in RBC and Hb levels among traditional smokers and vapers ($P < 0.05$) likely reflect a compensatory tissue hypoxia induced by chronic carbon monoxide or nicotine exposure, forcing an increase in erythropoiesis [12]. Conversely, the variations in MCV and WBC levels, alongside elevated platelet counts in substance users, underscore systemic hematological adaptations and inflammatory responses associated with aerosol inhalation [13]. These hematological alterations strictly corroborate previous literature establishing that both vaping and smoking alter baseline hematological profiles, challenging the assumption that e-cigarettes lack systemic hematological impact [14].

The two exposed groups reached their elevated red cell counts by what may be different routes. Vapers recorded the highest red cell count of the three groups (6.20 ± 0.08 , against 5.72 ± 0.09 in smokers and 4.95 ± 0.13 in controls), even though electronic cigarettes involve no combustion. The platelet elevation was likewise near-identical in the two exposed groups (291.6 ± 13.42 in vapers versus 284.7 ± 15.91 in smokers), while controls were markedly lower (236.3 ± 7.32). Read strictly on the present data, these two observations are compatible with a mechanism common to both products rather than one specific to combustion. The cross-sectional design cannot test that proposition, and it is advanced here only as an interpretation of the values obtained, not as an established mechanism.

Two findings merit particular caution. First, the total white blood cell count was lowest in conventional smokers (6.35 ± 0.14) and highest in the control group (8.38 ± 0.46), a direction opposite to the one anticipated. Within the present dataset several explanations remain open: samples were drawn in the fasting state after an interval of abstinence; the participants were young and free of comorbidity; and twenty participants per group limits the precision of the estimate. Second, the mean corpuscular volume was lowest in vapers (78.65 ± 0.83) and highest in conventional smokers (87.03 ± 1.75), with controls intermediate (81.60 ± 0.92). Neither observation can be regarded as settled on the present evidence, and both require replication in larger cohorts with measurement of iron status and reticulocyte indices.

4.3 Lipid Profile

The significantly elevated levels of total cholesterol and triglycerides observed in both traditional smokers and vapers ($P < 0.05$) highlight a clear link between nicotine inhalation and disrupted lipid metabolism. Nicotine triggers the release of adenylyl cyclase-activated lipolysis, which increases free fatty acid flux to the liver, subsequently driving up the synthesis of hepatic triglycerides and cholesterol [15]. These findings closely mirror previous international cohorts, confirming that e-cigarette aerosols, despite being marketed as safer alternatives, still exert a profound atherogenic effect similar to conventional tobacco products [16].

That vapers recorded the highest cholesterol of the three groups (274.85 ± 20.16 mg/dL, against 244.40 ± 15.61 mg/dL in smokers and 147.85 ± 4.20 mg/dL in controls) is the most striking metabolic finding of this study. Expressed against their own control group, the vaping group carries a mean total cholesterol 85.9 % higher and a mean triglyceride concentration 59.0 % higher, exceeding the smoking group on both measures (+65.3 % and +51.0 %, respectively). The deviation is therefore not marginal, and it is the electronic cigarette group, not the combustible cigarette group, that is displaced furthest from the healthy baseline on both lipid parameters. Whether this reflects the nicotine dose delivered by contemporary devices, constituents of the e-liquid other than nicotine, or unmeasured differences in diet and physical activity cannot be determined from the present data.

4.4 Kidney Function

The significant elevations in serum urea and creatinine levels among both conventional smokers and e-cigarette users ($P < 0.05$) highlight the potential nephrotoxic impact of nicotine and aerosol chemical constituents. These inhalants induce renal oxidative stress and cause systemic vasoconstriction, which subsequently impairs the glomerular filtration rate (GFR) and alters baseline renal biomarkers [17]. These findings align with recent literature demonstrating that e-cigarette use, similar to traditional tobacco smoking, contributes to renal cellular injury and early manifestations of renal impairment [18].

The magnitude of the renal change deserves emphasis. Serum creatinine in conventional smokers (1.62 ± 0.05 mg/dL) was approximately twice that of the control group (0.82 ± 0.05 mg/dL), and serum urea was raised by roughly a quarter (40.80 ± 2.19 against 33.20 ± 1.58 mg/dL). Vapers occupied an intermediate position (creatinine 1.42 ± 0.06 mg/dL; urea 39.80 ± 1.07 mg/dL), significantly above controls yet below conventional smokers. The ordering is identical for both renal markers, which strengthens the internal coherence of the finding. On the present data the lower creatinine among vapers is best read as a difference of degree rather than of kind, since the vaping group is displaced from its control group on every renal parameter measured, and it was on average the youngest of the three groups.

4.5 Limitations of the Study

1. The cross-sectional design permits association but not causal inference; the observed differences cannot be attributed to smoking or vaping with certainty.
2. The sample of twenty participants per group is adequate to detect large effects only, and smaller yet clinically meaningful differences may have been missed.
3. Only male participants were studied, so the findings may not generalize to women.
4. Smoking and vaping status were self-reported and were not verified biochemically (for example, by serum cotinine or exhaled carbon monoxide), leaving the possibility of misclassification and of undisclosed dual use.
5. Nicotine concentration, device power, and puff topography were not quantified, so exposure could not be related to the magnitude of the physiological response.
6. The panel of parameters was limited: HDL and LDL cholesterol, inflammatory markers, and urinary albumin excretion, which is a more sensitive index of early glomerular injury, were not measured.
7. Each parameter was measured at a single time point, and residual confounding from diet, physical activity, and occupational or environmental exposures cannot be excluded.

5. CONCLUSION

The present study compared the impacts of electronic cigarettes and traditional smoking on hematological parameters, lipid profiles, and renal function against a healthy control group. Age was equivalent across all research groups, confirming it was not a confounding factor in the results. Both traditional smokers and e-cigarette users exhibited significant abnormalities compared to controls. Elevated red blood cell, hemoglobin, and platelet counts were observed, likely due to a compensatory reaction to hypoxia or systemic smoking effects, while altered white blood cell counts and mean corpuscular volume indicated affected immune responses and red blood cell morphology. E-cigarette users showed significantly higher total cholesterol and triglycerides, indicating a potential risk of dyslipidemia and cardiovascular diseases comparable to traditional smoking. Both smoking groups showed significantly higher serum urea and creatinine levels, though the harmful effects were marginally

stronger in traditional cigarette smokers. Electronic cigarettes are shown to be just as harmful as traditional cigarettes regarding blood parameters, lipid metabolism, and kidney function.

6. RECOMMENDATIONS

There is a critical need for public health initiatives and increased awareness to reduce both traditional and electronic cigarette consumption. On the basis of the present findings, the following measures are proposed:

1. Marketing claims presenting electronic cigarettes as a safe or harmless alternative should be restricted until long-term safety data become available.
2. Health-warning requirements for electronic cigarettes should extend beyond addiction to include the potential hematological, metabolic, and renal effects documented here.
3. Clinicians should ask about vaping as routinely as they ask about smoking, including among patients who have never smoked, and should consider periodic assessment of the lipid profile, renal function, and complete blood count in long-term users.
4. National health surveys should record device type, nicotine concentration, and frequency and duration of use, so that population-level exposure can be monitored.
5. Future research should prioritize prospective cohorts with larger and mixed-sex samples, biochemical verification of exposure, and quantification of nicotine dose, in order to establish dose–response relationships and to determine whether the alterations reported here are reversible upon cessation.

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