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Comparative Analysis of Heavy Metal Exposure and Liver Enzyme Levels in Hookah and Cigarette Smokers

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ABSTRACT:

Tobacco consumption, including cigarettes and hookah, remains a major global health concern. This study investigates the impact of cigarette and hookah smoking on liver function and the levels of heavy metals in serum among smokers compared to non-smokers. This project was executed from October 2024 to February 2025 in the Kurdistan area. This time was used to assess the impact of cigarette and hookah smoking on the deposition of heavy metals in blood and liver parameters of smokers compared to nonsmokers. The study involved 200 participants, comprising 80 non-smokers and 120 smokers (60 cigarette and 60 hookah users) aged between 18-70 years. Blood samples were collected, and serum levels of liver enzymes (ALT, AST, ALP, GGT), bilirubin, albumin, were analyzed using a fully automated biochemical analyzer (Mindray BS 240) and trace elements (Ni, Fe, Hg, As, Pb, Cd) were analyzed using Inductively Coupled Plasma Optical Emission spectroscopy (ICP-OES). The findings revealed that hookah and cigarette smokers exhibited significantly higher levels of liver enzymes (ALT, AST, GGT) and heavy metals (Hg, Pb, Cd) compared to non-smokers. Conversely, ALP and albumin levels were significantly lower among smokers. Notably, the levels of Ni showed no significant differences between groups. The study highlights the heightened exposure to toxic heavy metals and liver dysfunction associated with tobacco use, with hookah smoking posing comparable or even greater risks than cigarette smoking. These results underscore the need for increased awareness and regulatory measures to mitigate the health risks of both cigarette and hookah smoking.

Keywords: Cigarette, Heavy metal, Hookah, Liver enzyme, Smoker.

1 INTRODUCTION

Tobacco is ingested by several methods, including cigarettes, hookahs, and chewing. Tobacco use is a pervasive global concern that not only imposes significant social and economic burdens but also contributes to severe public health challenges. Tobacco use is the second largest cause of mortality globally [1]. It is linked to approximately 8 million deaths annually, primarily due to health complications and co-morbidities associated with smoking [2]. The primary Harmful and Potentially Harmful Constituents (HPHCs) are carbon monoxide (CO), nicotine, particulate matter (PM), volatile organic compounds (VOCs), acrolein, arsenic, and heavy metals. Figure 1 illustrates a comparison of the fold differences of some (HPHCs) between smoking one cigarette (1 g of tobacco) and one session of waterpipe (8–12 g of tobacco) [3]. The plasma nicotine levels after a single session of waterpipe smoking are approximated to be comparable to smoking (2–3) cigarettes [4]. Despite the particulate matter (PM) size supplied during waterpipe smoking being less than that of cigarettes (0.04–0.15 µm against 0.15–0.5 µm), the quantity of PM inhaled in a single waterpipe breath (1L) may attain up to 70 × 10-9 particles, in contrast to a cigarette breath (45 ml) which contains 9.2 × 10-9 particles [5]. An estimated 1.1 billion individuals globally use tobacco products, with cigarettes being the predominant choice (82%) [6]. Hookah is a tobacco product that is becoming popular in the United States and other nations, especially among the young. Water pipe smoking (WPS), commonly referred to as hookah in Arab and British contexts, shisha in German, narghile in Armenian and Bulgarian, hubble-bubble in India, goza in Egypt, chichi in France, qalyan in Persian, madaa in Yemen, and gaya in Ethiopia, constitutes a method of tobacco consumption practiced by both men and women across the Middle East, Africa, and Asia [7].

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Smoking hookah has many harmful effects due to the presence of 4,000 chemical substances in hookah smoke, the majority of which are generated during combustion and are associated with over 40 carcinogenic compounds [8]. Hookah blows more smoke than cigarettes. Cigarettes create 500-600 ml smoke, whereas hookah produces 5000. The use of hookah has gained popularity among adults in Asia and Africa, particularly in the Middle East and Arab nations [9]. Hookah smoke contains more harmful substances than cigarettes. A daily hookah smoker has the same nicotine level as a 10-cigarette smoker. Hookah smoking is linked to cancer, cardiovascular disease, and nicotine addiction, although epidemiological studies are required [10]. Hookah smoke contains significant quantities of carcinogenic substances, including hydrocarbons and heavy metals [11]. Waterpipe use produces elevated levels of carbon monoxide (CO), mostly due to charcoal combustion; substituting charcoal with an electric heater decreases CO emissions from waterpipes by 90% [12]. The carbon monoxide breathed during a waterpipe session is believed to exceed the quantity exhaled after consuming a full pack of cigarettes [13]. The use of waterpipes may generate a significant quantity of volatile organic compounds (VOCs). Employing a standardized smoking machine procedure, a single session of waterpipe smoking resulted in elevations of formaldehyde (27-fold), acrolein (19-fold), methacrolein (4-fold), and propionaldehyde (9-fold) [14]. The origin of heavy metals, including lead (Pb) and chromium (Cr), is believed to be the charcoal used in waterpipe smoking [15].

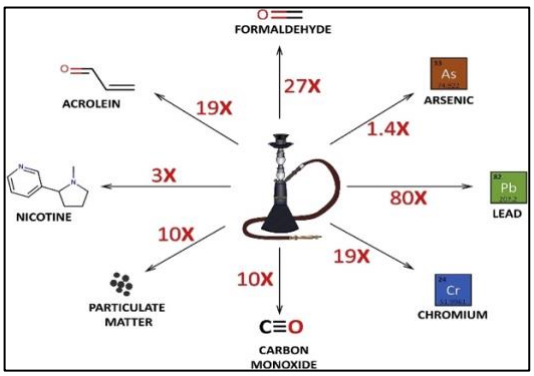

FIGURE 1. Relative amounts of harmful substances in one session of waterpipe tobacco smoke (8–12 g) versus smoking a single cigarette (1g of tobacco)

Heavy metal is characterized as a metal having a specific weight of more than 5 g/cm³ or an atomic number above twenty. Certain heavy metals, such as copper, are requisite and vital for biological functions, metabolism, development, and reproduction [16]. However, they are hazardous at elevated concentrations, including lead, copper, zinc, chromium, and cobalt, which are found at low levels inside human tissue (e.g. ppm) [17, 18]. Liver enzymes, including ALT (Alanine Aminotransferase), AST (Aspartate Aminotransferase), ALP (Alkaline Phosphatase), total bilirubin, albumin, and GGT (Gamma-Glutamyl Transferase), serve as indicators of hepatic function. The harmful consequences of smoking include toxic compounds that induce oxidative stress by lipid peroxidation, resulting in the activation of hepatic stellate cells, liver fibrosis, and heightened production of inflammatory cytokines [19].

2 MATERIAL AND METHODS

2.1 STUDY GROUPS

The study involved 200 participants: 80 controls and 120 smokers (60 hookah smoker and 60 cigarette smoker). All participants were aged between 18-70 years. 5 ml blood sample was drawn from the vein of the participates and centrifuged at 5000 rpm for 10 minutes. The separated serum samples were used to measure the levels of liver function test including (AST, ALT, ALP, GGT, T. Bilirubin and albumin) by full automated biochemical analyzer (Mindray bs-230) brand. Also, the trace elements were measured by using ICP-OES (Agilent Technologies). The Koya University ethical committee (KOUFSDHM2024-002) granted ethical approval for this research on 24/9/2024.

2.2 SAMPLE PREPARATIONS AND DIGESTION

Digestion of samples for heavy metal measurement, (1 ml) of serum from each sample was digested with (9 ml) of 65% nitric acid and (1 ml) of hydrogen peroxide (Merck, Germany). The samples were then immersed in a water bath at 98 °C for 4–6 hours to complete the digestive process and get a translucent solution. Upon completion of digestion, the samples were allowed to cool to ambient temperature for one hour, after which they were diluted with double-distilled water to a final volume of 25 ml. The produced samples were filtered in sterile containers. The processed samples were then transferred for elemental analysis using the ICP-OES instrument. All measurements were conducted by qualified professionals in the laboratory with identical equipment. The concentration of components in the serum was expressed as (µg/l and mg/l).

2.3 STATISTICAL ANALYSIS

GraphPad Prism Statistics version 10.4.1 was used to analyze the data. The normality test was evaluated by (Shapiro-Wilk and Kolmogorov-Smirnov test) to assay the homogeneity of variance for variables. One-way (ANOVA) test was used to compare the study group. Pearson's correlation test was used to establish a relationship between the antioxidants and the biomarker for oxidative stress. The results were displayed as the mean and standard deviation (SD). A p-value of less than 0.05 was deemed statistically significant.

4 RESULT AND DISCUSSION

Age, economic position, marital status, body mass index, and participant family history are among the general characteristics of the study's groups, as shown in Table 1.

The duration of hookah use was 7.06 ± 4.57 years. The duration of cigarette smoking was 15.08 ± 5.78 years, and each person was smoked 43±15.81 cigarette per day.

Table 1. characteristics of the study population

Age (year)	Non smoker No. (80)	Hookah smoker No. (60)	Cigarette smoker No. (60)
Age (year)	25.58 ± 6.712	29.64 ± 10.83	40.29 ± 9.145
	Habitat		
Rural	46 (% 57.5)	34 (% 56.7)	27 (% 45)
Urban	34 (% 42.5)	26 (% 43.3)	33 (% 55)
Cigarettes/day	-	-	43±15.81
Time using hookah or cigarette/hours	-	7.06 ± 4.57	15.08 ± 5.78
BMI (Kg/m^2)	26.45 ± 6.714	27.54 ± 10.80	35.32 ± 9.442

Results are express as mean± SD

The serum of the participants in the current investigation was examined for the presence of some trace elements (heavy metals) as shown in table 2 and figure 2. In the group of non-smokers, all trace elements were showed lower than to another group (Hookah and cigarette) smoker. Heavy metals, including nickel (Ni), iron (Fe), mercury (Hg), arsenic (As), lead (Pb), and cadmium (Cd), may build in the body over time, with serum levels being used as biomarkers to evaluate exposure and associated health hazards [20]. Heavy metal poisoning from smoking may lead to oxidative damage [21]. The concentrations of these metals in blood fluctuate markedly among non-smokers, hookah users, and cigarette smokers, attributable to variations in exposure sources and smoking duration [22]. (Ni) was showed no significant difference between non-smoker (0.0152±0.003 mg/l), cigarette smoker (0.0156±0.0028 mg/l) and hookah smoker (0.0175±0.007 mg/l), this result was accepted with pervious study [23], as in table 2 and figure 1. The iron level was decreased in cigarette (0.1095±0.0890 mg/l) and hookah (0.11950±0.0856 mg/l) smoker as compared to non-smoker (0.2110±0.1071 mg/l), this result was accepted with pervious study [23-25]. The oxygen-carrying capacity of hemoglobin is reduced by the binding of carbon monoxide in tobacco smoke, which in turn affects iron metabolism. Smoking can result in chronic inflammation, which disrupts iron metabolism and absorption [26].

Table 2 also showed a statistically increase in the Hg mean for hookah smokers (1.4750±0.2971 µg/L), cigarette smoker group (1.7500±0.32687 µg/L) respectively as compared to Non-smokers group (0.0358±0.00759 µg/L). The oxygen-carrying capacity of hemoglobin is reduced by the binding of carbon monoxide in tobacco smoke, which in turn affects iron metabolism. Smoking can result in chronic inflammation, which disrupts iron metabolism and absorption [27]. Arsenic level of cigarette and hookah smoker (8.5000±1.9868 µg/L) (7.2222±1.5635 µg/L) respectively was showed non-significant elevated as compare to non-smokers group (6.4000±1.8468 µg/L). The risk of lung, kidney, and bladder malignancies, as well as cardiac disease, is elevated by arsenic exposure from tobacco smoking [23, 28].

The results showed a statistically increase in the Pb value of (10.600±4.115 µg/L) for hookah smokers, cigarette smoker group (11.690±5.040 µg/L) as compared to Non-smokers group (3.5444±1.08410 µg/L). The use of hookah and smoking cigarettes results in the exposure of consumers to lead (Pb), a toxic heavy metal that poses substantial health risks [29]. Smoking both cigarettes and hookahs leads to elevated blood lead levels and increased lead exposure. The fumes may contain even higher concentrations of heavy metals due to the additional use of charcoal in hookahs [30].

The results showed significant increase in the Cd level in hookah smokers (1.5000±0.4218 µg/L), cigarette smoker group (1.8950±0.3219 µg/L) as compared to Non-smokers group (0.45556±0.1810 µg/L). According to research, the concentration of Cd in the blood of tobacco consumers is several times higher than that of non-smokers [20, 31]. The quantity of cadmium in tobacco is contingent upon the analytical method employed to ascertain cadmium, as well as the variety and provenance of the plant [32]. The tobacco plant (Nicotiana tabacum) naturally accumulates cadmium, which is why it is known to be substantially absorbed in cigarette smoke [33, 34]. In tobacco leaves, the concentration of

cadmium ranges from 1 to 2 µg × g−1 of dried matter, which is equivalent to 0.5–1 µg of cadmium per cigarette, and the absorption of cadmium is increased by smoking 20 cigarettes per day [35]. The average daily Cd intake from 20 cigarettes is approximately 1 µg. Nevertheless, it is presumed that modifications to the design of cigarettes and the refining of tobacco can mitigate the ingestion of cadmium from smoking. Smoking cigarettes for an extended period of time (e.g., 20 years) results in the introduction of approximately 15 mg of cadmium into the smoker's body [36]. that cigarette consumption may lead to higher levels of cadmium than hookah smoking [37]. Cadmium exposure is linked to a variety of health hazards, such as an elevated risk of cancer, bone demineralization, and renal injury. Cadmium accumulation in the body is influenced by the use of both cigarettes and hookahs [38].

Table 2. Levels of trace elements in serum of non-smoker, hookah smoker and cigarette smokers

Heavy metals	Non smoker	Hookah smoker	Cigarette smoker	P value
Ni (mg/L)	0.0152±0.003a	0.0175±0.007a	0.0156±0.0028a	0.3742
Fe (mg/L)	0.2110±0.1071a	0.11950±0.0856b	0.1095±0.0890b	0.0212
Hg (µg/L)	0.0358±0.00759a	1.4750±0.2971b	1.7500±0.32687c	0.0007
As (µg/L)	6.4000±1.8468ab	8.5000±1.9868b	7.2222±1.5635a	0.0035
Pb (µg/L)	3.5444±1.08410a	11.690±5.040b	10.600±4.115b	0.0002
Cd (µg/L)	0.45556±0.1810a	1.5000±0.4218b	1.8950±0.3219c	<0.0001

Results are express as mean± SD
same letters mean non-significant different
different letters mean significant different

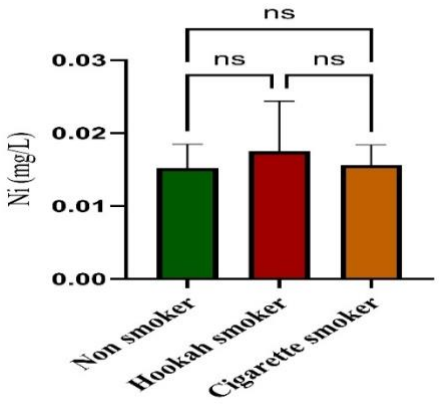

FIGURE 2. Ni level in smoker and nonsmoker

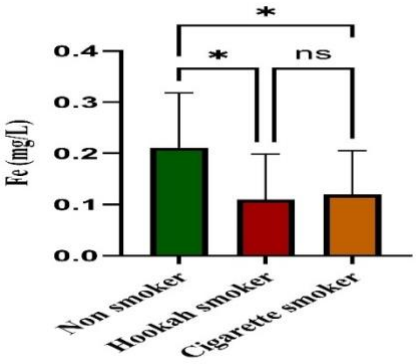

FIGURE 3. Fe level in smoker and nonsmoker

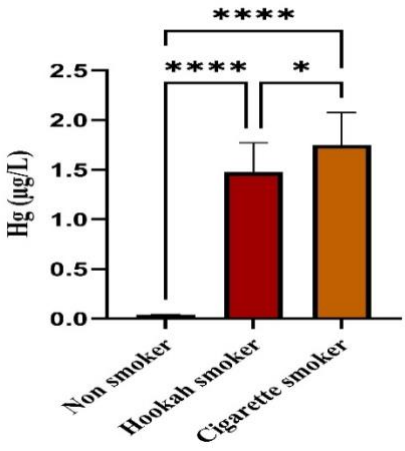

FIGURE 4. Hg level in smoker and nonsmoker

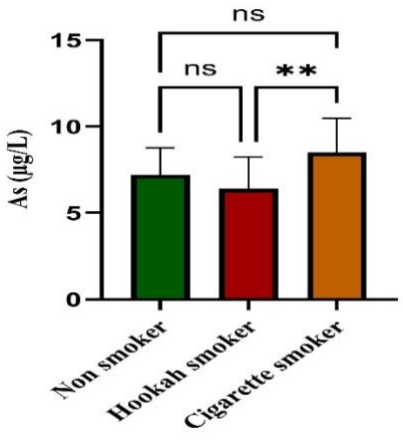

FIGURE 5. As level in smoker and nonsmoker

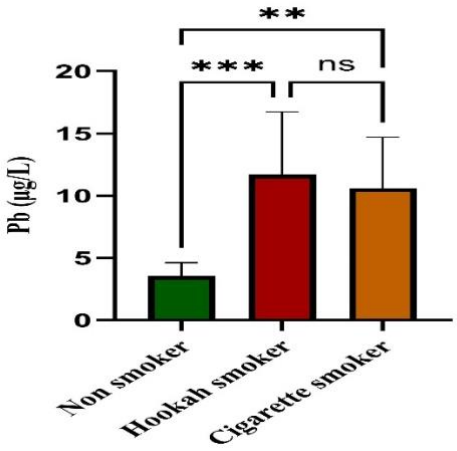

FIGURE 6. Pb level in smoker and nonsmoker

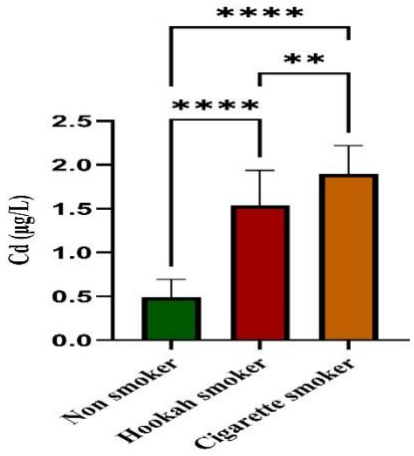

FIGURE 7. Cd level in smoker and nonsmoker

Smoking had an effect on the liver. table 3 and figure 3, shows that compared to non-smokers; smokers liver function was much higher. As an example, blood tests for AST, ALT, total bilirubin, GGT, and albumin were all higher in smoker samples than in nonsmokers, although ALP was lower in smoker samples from both cigarette and hookah use. The effects of smoking on liver function may be responsible for this increase in levels as compared to non-smokers. ALT, AST, TB and GGT was elevated in cigarette and hookah smoker as compared to non-smoker. ALT in non-smoker was (20.64±4.816 IU/L) and in cigarette smoker was (30.08±10.43 IU/L) and hookah smoker was (28.53±10.24 IU/L), this result showed that ALT was significantly increased in cigarette and hookah smoker as compared to non-smoker [39]. Liver damage or inflammation may be suggested by elevated ALT levels. The consequences of smoking, including both cigarettes and hookah [7]. This result showed that AST was significantly increased in cigarette and hookah smoker (55.86±18.10 IU/L) (57.25±21.29 IU/L) respectively, as compared to non-smoker (35.82±6.546 IU/L). Studies demonstrate that smoking correlates with the exacerbation and advancement of liver disorders, notably fibrosis and hepatocellular carcinoma, illnesses often characterized by heightened AST and ALT levels [40]. ALP was decreased in cigarette and hookah smoker (174.3±51.27 IU/L, 163.3±52.17 IU/L), respectively as compared to non- smoker (235.1±39.39 IU/L). Contrary to the results of our study, another study suggest that ALP increased in smoker as compared to non-smoker [39]. Smoking may diminish the absorption of vital nutrients such as zinc and magnesium, which serve as cofactors for ALP synthesis [41]. Heavy smokers may exhibit a poor diet, resulting in decreased protein levels, which may subsequently reduce ALP [42]. Total bilirubin in non-smoker was (0.5722±0.1466 mg/dl) and in cigarette and hookah smoker was (0.7750±0.2221 mg/dl) (0.7611±0.2831 mg/dl) respectively. Cigarette and hookah users often have marginally raised total bilirubin levels, since the carbon monoxide included in these substances may hinder oxygen transport, resulting in enhanced hemoglobin degradation and, thus, increased bilirubin production [39]. As the results showed that GGT was significantly elevated in cigarette and hookah smoker (32.27±12.07 U/L) (34.38±21.62 U/L) respectively as compared to non-smoker (21.39±7.962 U/L). Cigarette smoke contains many heavy metals, including lead, cadmium, and mercury. Oxidative stress in the liver induced by alcohol intake and metabolism attains a critical threshold more readily when compounded by generalized oxidative stress from concomitant smoking. Smoking increases oxidative damage, prompting the creation of GGT as a defensive mechanism [43]. Certain research indicates that serum GGT activity rises after cadmium exposure. Diet is the primary source of environmental cadmium exposure [44]. Tobacco smoking leads to blood cadmium levels in smokers that are 4 to 7 times greater than those in nonsmokers [45].

Table 3. Liver parameters in serum of non-smoker, Hookah smoker and Cigarette smokers.

Liver parameters	Non smoker	Hookah smoker	Cigarette smoker	P value
ALT (IU/L)	20.64±4.816^{b}	28.53±10.24^{a}	30.08±10.43^{a}	0.0179
AST (IU/L)	35.82±6.546^{b}	57.25±21.29^{a}	55.86±18.10^{a}	0.0008
ALP (IU/L)	235.1±39.39^{a}	163.3±52.17^{b}	174.3±51.27^{b}	<0.0001
TB (mg/dl)	0.5722±0.1466^{b}	0.7611±0.2831^{a}	0.7750±0.2221^{ab}	0.0208
GGT (U/L)	21.39±7.962^{b}	34.38±21.62^{a}	32.27±12.07^{ab}	0.0218
Albumin (gm/dl)	5.092±0.2951^{a}	4.840±0.2157^{b}	4.746±0.1458^{b}	0.0004

Results are express as mean± SD
same letters mean non-significant different
different letters mean significant different

Serum cadmium content independently correlates with elevated serum GGT levels. Additionally, serum mercury buildup may have been associated with elevated blood GGT levels [46].

The mean albumin level in cigarette and hookah smoker (4.746±0.1458 mg/dl) (4.840±0.2157 mg/dl) was significantly lower than that of non-smoker (5.092±0.2951 mg/dl). The oxidative stress and inflammatory responses that tobacco smoke induces may be responsible for the decrease in albumin levels among smokers, which can impede protein synthesis and liver function [47].

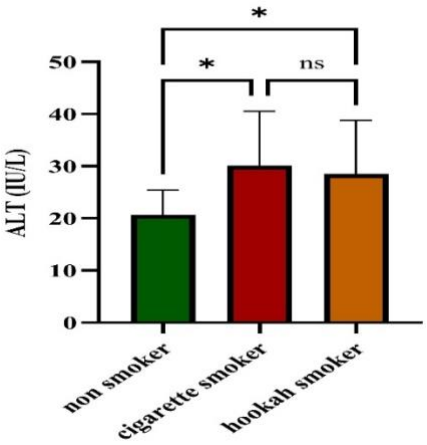

FIGURE 8. ALT level in smoker and non-smoker

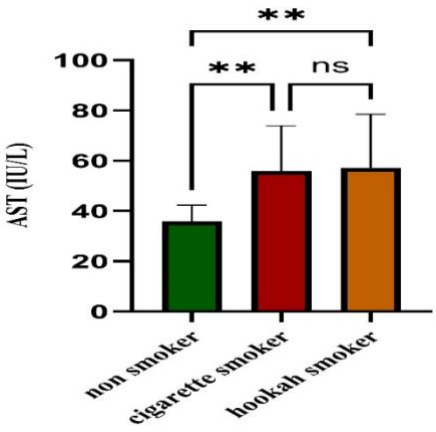

FIGURE 9. ALT level in smoker and non-smoker

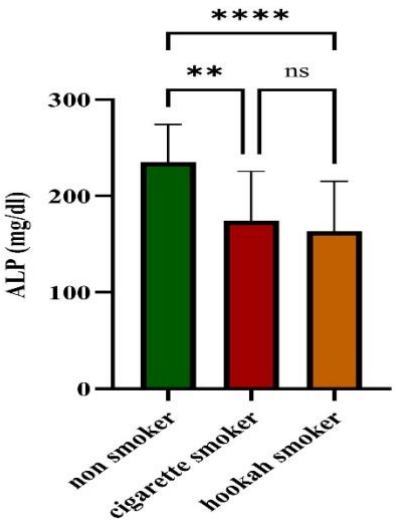

FIGURE 10. ALP level in smoker and non-smoker

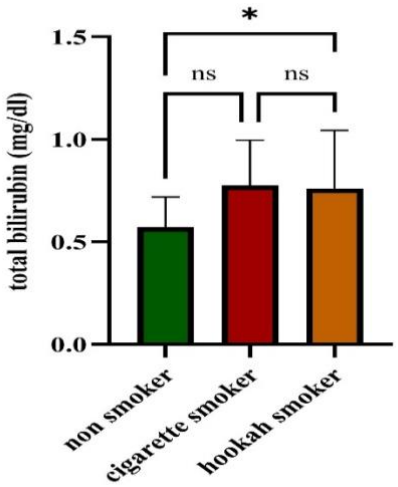

FIGURE 11. TB level in smoker and non-smoker

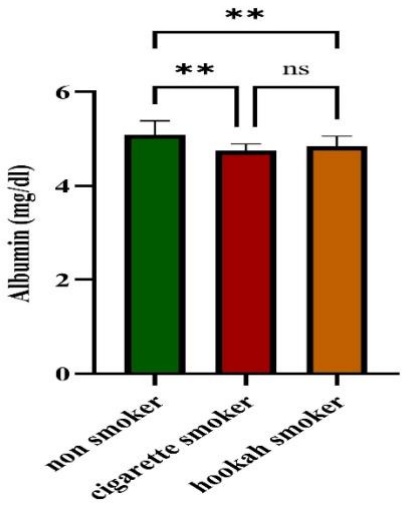

FIGURE 12. Albumin level in smoker and non-smoker

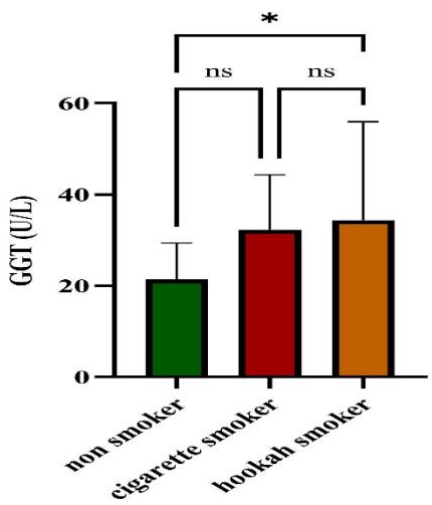

FIGURE 13. GGT level in smok.er and non-smoker

CONCLUSION

This study concludes that hookah and cigarette smoking pose serious health concerns. Many people think hookah is safer than cigarettes, yet regular hookah smoking exposes you to dangerous compounds for a long time. Hookah smoking may cause respiratory infections, cardiovascular issues, and cancer due to chemical inhalation. Hookah smoking has equivalent or higher risks than cigarette smoking for toxic heavy metals and liver damage, according to the research.

These results highlight the need for increased knowledge and regulation to decrease cigarette and hookah health risks. Hookah and cigarette users had greater liver enzymes (ALT, AST, GGT) and heavy metals (Hg, Pb, Cd) than non-smokers. Smokers had considerably lower ALP and albumin levels. Notably, Ni levels were similar across groups.

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