

## Research article

## Effects of hookah smoking on liver functions, lipid profile, and adult blood count

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## ABSTRACT

**Introduction and Aim:** Smoking is the most common habit and the largest health risk among people who have an impact on the human body. The goal of our study was to examine the effects of hookah (Shisha) on the liver, lipid profile, and blood content in adult smokers to adult non-smokers.

**Materials and Methods:** A total of 50 men between the ages of 30 and 60 years took part in the study. These samples were divided into two groups: 30 smokers and 20 non-smokers. Every male is subjected to a complete medical history survey as well as frequent tests to rule out any underlying illnesses or diseases. Blood samples were taken in quantities of 10 mL to examine the blood. Blood samples were placed on the red roof when blood samples were used with anticoagulant factor. The identification of biological parameters was done using enzyme methods and a Random kit.

**Results:** The results showed that Hb, PCV, WBC, ESR, platelets, and RBC were all elevated, whereas ALP and transaminase enzymes were not. Although not significantly, TC and TG increased the lipid profile findings. Our analysis yielded a wide range of outcomes. Various factors could be to blame for these disparities.

**Conclusion:** We describe how future research will focus on the relationship between organ functions and their working tests for smoking effects based on our findings. Age, sex, nutrition, and hereditary behavior must all be included in future studies.

**Keywords:** Smoker; lipid profile; blood; shisha.

## INTRODUCTION

Shisha (Hookah) is a method of smoking tobacco that was developed by Hakim Abul-Fath Gilani in the sixteenth century. The purpose of using this apparatus was to drive smoke through water in an attempt to 'decontaminate' the smoke, a dubious notion that has been addressed by the clinical society on several occasions. Since the beginning of time, the essential instrument of a shisha framework has been constant. Regardless, the structure has been greatly simplified (1). Shisha consists of an upper and a lower part associated with a channel. The top is made up of a bowl that holds tobacco or molasses. It is then covered with pierced aluminum foil, which is then covered with eating charcoal. On the instrument's base is a gasket-secured container with a tube and an extraction valve (utilized for disposing of static smoke; 2). Shisha is also known as the borry, argyle, hookah, whirlwind, or water pipe (Fig. 1) is a tobacco tube that draws smoke through the water in a bowl and has a lengthy but adjustable cylinder. Water tube usage has existed for thousands of years in northwestern India and is now gaining popularity in the United States and Europe, extending to Iran, the Arab world, and Turkey, as an obvious occurrence in the West (3, 4). In addition, the Shisha act differed in a number of ways. Smoked materials have expanded beyond simple tobacco, such as apples, grapes, and mint. For example, shisha smoking has been more accessible as a result of an increase in the number of shisha service outlets and their modest shoreline. The

fact that the smoke is separated by water yet develops evidence that successfully smoking shisha may be harmful or more than smoking cigarettes, when opposed to cigarettes, is generally thought to be a safer option (5).

Shisha smoking causes oxidative stress by increasing free radicals, which interact with biological molecules to cause oxidative stress, that causes biological molecules to become more stressed. (6) Hookah smoking is also a major source of heavy metals that are harmful to human health. Heavy metals could be caused by the tobacco blends used in shisha.

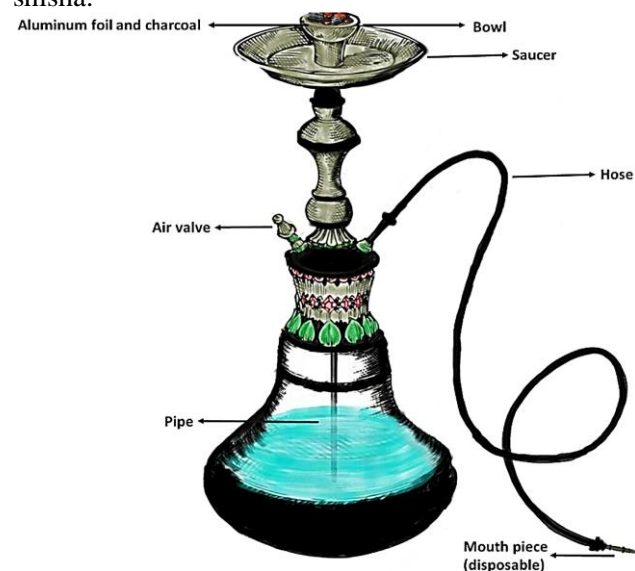


Fig. 1: A typical Shisha apparatus

Hookah pipes, for example, which contain roughly 30% tobacco and 70% a mixture of flavorings, glycerol, and sweeteners, may have different tobacco percentages (7). Furthermore, several studies have indicated that shisha smoking for 30 minutes causes high blood pressure, respiration, impaired lung functions, decreased blood oxygenation, brain damage, and fainting (8). For the purpose of evaluating the effects of harmful smoking on human blood content and organ functions, our research focuses on the following targets:

- Performance of blood tests (Hb, PCV, WBC, ESR, and other parameters) on smokers and compares their results to nonsmokers to determine the impact of shisha smoking.
- Calculation of the lipid profile in shisha smokers' blood compared to nonsmokers' (TC, TG, HDL, LDL, and VLDL).
- Comparison of the liver functions and other key enzymes evaluated in the blood of shisha smokers to non-smokers (ALK, AST, ALT, albumin, TSP, urea, CK, CPK, LDH, and glucose).

The aim of this study was to identify the harmful effects of smoking shisha by examining blood count, liver functions, and lipid profile.

### MATERIALS AND METHODS

Fifty men, ranging in age from 30 to 60, participated in our study. The "30-Shisha Smoker" group and the "20-Non-smokers" group were established at random and given the names "30-Shisha Smoker" and "20-Non-smokers" respectively. To rule out any illnesses or abnormalities, all males must fill in a detailed medical history questionnaire and undergo a series of routine examinations. Three milliliters of blood samples were collected. Of these, for analysis, three milliliters of blood were placed in a red tube containing anticoagulant factor. Three milliliters of blood were deposited in dry tubes, and the serum was centrifuged and frozen at -8°C for later examination. Following a 12-hour fasting, all individuals were tested for TC, TGs, HDL, LDL, and VLDL. Liver function and other important enzymes were regularly

examined (ALK, AST, ALT, albumin, TSP, urea, CK, CPK, LDH, and glucose).

The Random kit, for example, examines biochemical markers such as TC, TG, HDL, ALK, AST, ALT, albumin, TSP, urea, CK, CPK, LDH, and glucose using enzymatic assays.

### Statistical analysis

The data were presented as a mean value with standard deviation. All statistical analysis were performed using SAS-2002 software, and significant differences between groups were assessed using the Duncan-test (9).

### RESULTS

Table 1 shows the complete blood count (CBC) in two groups, shisha smoking group and nonsmoking group, which include (Hb, PCV, RBC, WBC, ESR, Plat., LYM, Monocyte, Neutrophil, Eosinophil, Basophil) (control). The results showed that smokers had the highest hemoglobin (Hb) levels ( $16.2 \pm 0.523$  g/dL) and non-smokers had the lowest ( $15.0 \pm 0.452$  g/dL), but there were no significant differences between their averages, in another hand, there was a significant increase in PCV level at ( $P \leq 0.05$ ) for smokers ( $45.8 \pm 0.544$ ) compared to non-smokers ( $40.1 \pm 1.26$ ). Our findings also demonstrate that Shisha smokers ( $244 \pm 6.59 \times 10^3 / \mu\text{L}$ ) had an insignificant increase in platelet levels as compared to non-smokers ( $224 \pm 15.7 \times 10^3 / \mu\text{L}$ ), the People who smoked Shisha had a higher number of leukocytes (WBC) ( $6.73 \pm 0.260 \times 10^3 / \mu\text{L}$ ) than non-smokers ( $5.84 \pm 0.446 \times 10^3 / \mu\text{L}$ ) in leukocytes (WBC) tests. In addition, smoking resulted in a substantial increase in basophil ( $0.684 \pm 0.0419 \times 10^3 / \mu\text{L}$ ) as compared to non-smoking ( $0.400 \pm 0.0774 \times 10^3 / \mu\text{L}$ ). Also, we discovered a drop in erythrocytes in smokers ( $5.07 \pm 0.0808$  million cells/  $\mu\text{L}$ ) compared to non-smokers ( $5.16 \pm 0.202$  million cells/  $\mu\text{L}$ ) in our study. ESR value (Erythrocyte Sedimentation rate) in smokers was ( $10.34 \pm 0.591$  mm) and in non- smokers it was ( $9.2 \pm 1.23$  mm).

**Table 1:** The impact of shisha smoke on the contents of the entire bloodstream (CBC)

Parameters	Groups		P-values
	Smokers	Controls	
PCV%	$45.8 \pm 0.544^a$	$40.1 \pm 1.26^b *$	0.0001
Hb%	$16.2 \pm 0.523$	$15.0 \pm 0.452$	NS**
WBC ( $10^3 / \mu\text{L}$ )	$6.73 \pm 0.260$	$5.84 \pm 0.446$	NS
RBC (million cells/ $\mu\text{L}$ )	$5.07 \pm 0.0808$	$5.16 \pm 0.202$	NS
Platelets ( $10^3 / \mu\text{L}$ )	$244 \pm 6.59$	$224 \pm 15.7$	NS
ESR (mm/hour)	$10.34 \pm 0.591$	$9.2 \pm 1.23$	NS
Lymphocyte ( $10^3 / \mu\text{L}$ )	$33.9 \pm 1.47$	$32.9 \pm 2.45$	NS
Monocyte ( $10^3 / \mu\text{L}$ )	$8.51 \pm 0.661$	$6.35 \pm 0.846$	NS
Neutrophil ( $10^3 / \mu\text{L}$ )	$5.04 \pm 0.308$	$5.16 \pm 0.596$	NS
Eosinophil ( $10^3 / \mu\text{L}$ )	$2.64 \pm 0.138$	$2.18 \pm 0.242$	NS
Basophil ( $10^3 / \mu\text{L}$ )	$0.684 \pm 0.0419^a$	$0.400 \pm 0.0774^b$	0.0063

\*Refers to means  $\pm$  standard deviation, \*\*NS Not significant between study groups in ( $P \leq 0.05$ ), a, b: significant differences in ( $P \leq 0.05$ )

In comparison to non-smokers, smokers' liver function had improved. Table 2 shows that the key liver function enzymes (AST and ALT) have increased in smokers compared to non-smokers. AST ( $13.6 \pm 0.347$  KAU/L) in smokers compared to non-smokers ( $10.0 \pm 1.32$  KAU/L) and ALT  $10.5 \pm 1.07$  in smokers compared to nonsmokers  $10.1 \pm 1.07$ ). The alkaline phosphatase enzyme (ALP) is also an excellent indicator of liver health; in our study, smokers ( $96.4 \pm 3.41$  KAU/L) had higher ALP levels than non-smokers ( $76.7 \pm 3.98$  KAU/L). Total protein and albumin levels are influenced by shisha smoking-related liver damage. As shown in the graph, there was a significant increase in their level in the cigarette group compared to the non-smoker group (Table 2). We discovered that smokers'

glucose levels had increased significantly ( $100.2$  mg/dL) as compared to nonsmokers' ( $97$  mg/dL). Other measurements, such as blood urea and creatinine, were also raised in our study, with no significant difference between smokers and non-smokers, as shown in table 2. We identified differences in serum lipid profile (TC, TG, LDL, HDL, and VLDL) levels between study groups that included non-smoking shisha smokers and non-smoking controls in our current study. Although the findings suggest that smokers are more likely than nonsmokers to develop coronary heart disease, we also found that the smoking group had greater levels of TC, TG, LDL, VLDL, and HDL than the nonsmoking group.

**Table 2:** Some serum biochemical markers are affected by shisha smoke.

Parameters	Groups		P-values
	Smokers	Controls	
Albumin(g/dL)	$4.94 \pm 0.138$	$4.71 \pm 0.437^*$	NS**
Urea (mg/dL)	$12.3 \pm 0.581 \pm$	$12.6 \pm 1.68$	NS
Creatinine(mg/dL)	$0.858 \pm 0.0459$	$0.780 \pm 0.0757$	NS
ALP KAU/L	$96.4 \pm 3.41^a$	$76.7^b \pm 3.98^b$	0.0153
AST KAU/L	$13.6 \pm 0.347$	$10.0 \pm 1.32$	NS
ALT KAU/L	$10.5 \pm 0.439$	$10.1 \pm 1.07$	NS
CPK (mcg/L)	$167 \pm 8.38$	$135 \pm 18.8$	NS
LDH (U/L)	$141 \pm 3.56$	$140 \pm 6.65$	NS
TC (mg/dL)	$198 \pm 4.26$	$178 \pm 6.99$	NS
TG (mg/dL)	$128 \pm 4.14$	$116 \pm 11.1$	NS
HDL (mg/dL)	$42.7 \pm 1.73$	$40.7 \pm 5.24$	NS
LDL (mg/dL)	$97.0 \pm 2.76$	$98.5 \pm 10.0$	NS
VLDL (mg/dL)	$23.7 \pm 0.828$	$23.3 \pm 2.22$	NS
Protein (mg/dL)	$7.72 \pm 0.171$	$6.92 \pm 0.266$	NS
Glucose (mg/dL)	$100.2 \pm 2.20$	$97 \pm 5.82$	NS

\*Refers to mean  $\pm$  standard deviation, \*\*NS Non-significant between studies groups in ( $P \leq 0.05$ ), a, b: significant differences in ( $P \leq 0.05$ ). HDL: High-density lipoprotein, LDL: Low-density lipoprotein, TG: Triglyceride, TC: Total cholesterol, VLDL: Very low-density lipoprotein, LDH: Lactate dehydrogenase, CPK: Creatinine kinase, ALP: Alkaline phosphatase, (ALT, AST): Transaminase enzymes.

## DISCUSSION

According to the findings, our results indicate that in the levels of hemoglobin (Hb) there was no significant difference in their averages between smoker and non-smoker, although there was a significant increase in PCV level among same groups, as documented in prior research ( $P \leq 0.05$ ; 10). An increased level of hemoglobin in the blood due to a decrease in the amount of oxygenated blood and increased CO in the smoking blood, which may also lead to atherosclerosis and acute coma (11), which is balanced by the production of hemoglobin in the blood. The findings also show non-significant increase in platelet level in both shisha smokers and non-smokers, the effects of smoking on platelet levels could be linked to the sharing factor. Increased nicotine and cotinine levels caused by smoking, for example, lead to a prothrombotic state in smokers due to increased platelet-dependent thrombogenesis (12, 13).

People who smoked shisha had a higher number of leukocytes than non-smokers; there were no significant differences, although our findings were consistent with those of other investigations (14) which found greater white blood cell count in smokers compared to nonsmokers. The generation of catecholamines by nicotine may result in an increase in leukocytes, as demonstrated in table 1. This could result in an increase in blood lymphocytes. There was also a substantial rise in basophil in smokers, which could explain the same (15). In our investigation, we also found that there is no significant decrease in erythrocytes in both smokers and non-smokers. This result agrees with other studies (16). However, other research has found that the amount of red blood cells in smokers is significantly lower than in non-smokers (17). Because our testing revealed a non-significant reduction in RBCs, this could be due to RBC haemolysis caused by smoking. The ESR was somewhat higher in smokers than the average ESR of

nonsmokers, but there was no discernible increase. Another report discovered that smoking induces an increase in the mean ESR; this followed a progressive pattern as the number of cigarettes smoked increased (18). The biochemical analyses for serum lipid profile in two groups were summarized in table 2; Smokers' liver function enzymes, AST and ALT were increased when compared to non-smokers. The increase in our sample may contribute to the influence of smoking on liver function. This has also been discovered in prior investigations (19). For example, AST levels have been reported to higher among smokers compared to nonsmokers (20). Other findings demonstrated that smoking causes an increase in ALT (21). On the other hand, the results have shown that smokers had a higher level of ALP than non-smokers; these increases were significantly higher in smokers than non-smokers. There is a relationship between smoking and liver disease, according to several research (22), although it is assumed that smoking can influence the body biochemical protein levels, particularly in liver tissue (23) Smoking has been found to affect the morphology and life processes of the liver and other organs in various investigations (24). Also, observed the shisha smoking caused liver abnormalities that affected total protein and albumin levels, with a considerable increase in smokers compared to non-smokers, as shown (table 2). Glucose levels had increased significantly when compared to nonsmokers.

Smoking affects insulin sensitivity, causing a decrease in glucose tolerance and an increase in blood glucose. This finding echoes prior research (25). The levels of urea and creatinine in our study were elevated, with no discernible difference between smokers and non-smokers (Table 2), According to previous research, smoking significantly increased creatinine and blood urea levels (23); this could be due to other factors influencing our results, such as sample size. In our current study, we found changes in blood lipid profile values, the findings imply that smokers are more likely to develop coronary heart disease than nonsmokers due to increased levels of TC, TG, LDL, VLDL, and HDL (22). This occurs because nicotine activates the adrenal-sympathetic nervous system, which produces adrenocorticotrophic hormone (ACTH), which acts on the sympathetic nervous system and increases lipolysis, resulting in increased serum free fatty acid, cholesterol synthesis, and liver secretion (18). It could be linked to a problem with lipoprotein metabolism, which reduces the ability of blood vessel walls (22) The enzymes LDH and CPK are the most sensitive and specific indicators of an acute myocardial infarction due to the side effects of smoking. In comparison, the smoking group (shisha) had slightly greater CPK and LDH levels than the control group (non-smoking).

## CONCLUSION

We describe how future research will focus on the relationship between organ functions and their working tests for smoking effects based on our findings. Age, sex, diet, and hereditary behaviour must be included in future studies.

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## CONFLICT OF INTEREST

Authors declare that there is no conflict of interest among authors.

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