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Comparison Of Some Kidney And Liver Function Tests Between Current And Former Iraqi Cigarettes Or Arghila Smokers

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ABSTRACT

Smoking is the world's largest single cause of death protection. It is a common trigger for cardiovascular diseases, as well as a range of cancers and other disabilities. Approximately half of the regular smokers will eventually be killed by their smoking habit, with many of these deaths occurring in the middle ages. As long as it causes premature death, tobacco consumption greatly reduces the quality of life, affecting family, friends and colleagues as well as smokers themselves. The current study included 30 Iraqi smokers in three different groups (cigarette smokers, arghila (hookah) smokers and former smokers) (age range= 19-53 years). The cases were selected from family, friends, college students and coffee shops the costumers, from November 2015 to March 2016. Ten healthy, non-smoker males of matched age were also included as a control group throughout this study. A careful history was obtained from each volunteer including age, smoking duration, type of smoking (cigarettes, arghila, or former), and family history of diseases (such as diabetes mellitus and thyroid disease). In the sera of the groups under study, some biochemical parameters such as (AST, ALT, ALP, creatinine, and urea) were estimated. The results showed significant differences ($p<0.05$) in the studied parameters in: (cigarette and arghila smokers), and (former smokers) groups, when compared to the control group.

Keywords: smoking, kidney function, liver function, transaminases, ALP.

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INTRODUCTION

Tobacco smoke contains thousands of chemicals like nitrogen oxides, tar, hydrogen cyanide, arsenic (white ant poison), phenol (paints), ammonia (floor cleaner), naphthalene (mothballs) , urethane acetone (paint stripper), cadmium (car batteries), carbon monoxide (carexhaust), dichlorodiphenyltrichloroethane (insecticide) and butane (light fuel), many of which are poisonous [1].

Nicotine is commonly used in cigarettes, pipes, or arghila smoking. Health risks of smoking arghila (hookah) include exposure to toxic chemicals that are not filtered out by water[2] and risk of infectious disease when sharing arghila[3, 4] arghila smokers inhale nicotine that is an addictive chemical. A typical hookah smoking session delivers 1.7 times a single cigarette nicotine dose[5]. The main tobacco alkaloid nicotine is mediated by smoking's addictive liability and pharmacological effects. They are elevated levels of serum lead and cadmium in smokers, resulting in glomerular dysfunction[6]. Nephropathies are accelerated by nicotine with an increased incidence of microalbuminuria progressing to proteinuria, followed by type-1 diabetes mellitus induced renal failure. Cigarette smoke-induced renal damage is due, at least in part, to activation of the sympathetic nervous system resulting in an elevation in blood pressure. Ethanol, nicotine, or concurrent intake significantly increases lipid peroxidation in liver and decreased superoxide dismutase activity and increased catalase activity in the kidney [7, 8].

Cigarette smoke does not come into direct contact with the liver; it incidentally affects the liver. At the end, the chemicals in cigarette smoke make their way to the liver. These chemicals cause oxidative stress, fibrosis and damage to the cells of the liver. The chemicals that are excessive in cigarette smoke prevent the liver from performing its main function. When cigarettes are used, the liver becomes less effective in removing toxins from the body [9].

Patients and methods

Patients

Inclusion criteria: from November 2015 to March 2016, thirty Iraqi smokers in three different groups (cigarette smokers, arghila (hookah) smokers, and former smokers), (age range= 19-53 years) were selected from family, friends, college students, and coffee shop costumers. Ten healthy non-smoker males (age range=21-51years) of matched age were also included as a control group in this study. Careful information was obtained from each volunteer in this study including age, smoking duration, type of smoking (cigarettes, or arghila, or former) and disease family history.

METHODS

A sufficient amount of venous blood (6-8 ml) was obtained from each subject included in this study. Blood samples were allowed to clot naturally at room temperature in plain tubes and then separated for 10 minutes by centrifugation at (1500x g). Haemolysis was avoided and the sera was divided into 5 aliquots, all samples were labeled by serial numbers and the name of the person, the serum was immediately frozen at -34C for further processing, once thawed, refreezing was avoided.

Urea and creatinine were estimated based on the randox reagent kit manufacturer procedure, while linear kit manufacturers determined aspartate aminotransferase (AST), alanine aminotransferase (ALT), and alkaline phosphatase (ALP).

Statistical Analysis: The data were analyzed by Duncan's multiple range test at [($p \leq 0.05$) was accepted as statistically significant and highly signific

ant when using the SPSS software ($p \leq 0.001$)]. All analyzes were repeated three times.

RESULTS

The mean age of the volunteers included in the current study was (29.15± 9.5 years) with age range between 19-53 years. Table (1) showed the distribution of subjects under study according to the types and states of smoke. The mean values of the biochemical parameters studied (AST, ALT, ALP, creatinine, and urea) showed significant increases in the group of volunteers smoking cigarettes compared to the group of non-smoking volunteers as shown in table (2).

Table (1) Distribution of subjects under study by age (mean values ± SD) and their smoking status

	Control group			Cigarettes Smokers group			
	Range	Mean	±SD	Range	Mean	±SD	P-Value
ALT (IU/L)	10-32	20.8	7.49	12-235	81.7	72.1	P<0.05
AST (IU/L)	4-43	16.2	11.3	17-135	67.5	42.4	P<0.05
ALP(IU/L)	19-98	56.7	30.4	51-214	95.5	51.8	P<0.05
Creatinine (mg/dL)	0.02-0.7	0.342	0.24	0.02-2.7	0.795	0.92	P<0.05
Urea (mg/dL)	14-48	29.3	10.9	7-75	37.5	22.4	P<0.05

	NO.	Age (year) (mean ± SD)	Smoking Status
Controls	10	(30.01±11.44) (21 - 51)	-
Cigarettes smokers	10	(25.50±4.06) (22 - 32)	12-24 cigarettes per day
Arghila (hookah) smokers	10	(22.00 ± 1.88) (19-26)	1-2 arghila per 2 days
Formers smokers	10	(41.01±12.96) (21 - 53)	Formers smoking from1-20 years

Table (2) Comparison between some biochemical parameters (AST, ALT, ALP, creatinine, and urea) of healthy individual and cigarettes smokers group

The alterations in some biochemical markers that have been measured in the current study were illustrated in table (3); where significant differences ($p<0.05$) were obtained in (AST, ALT, ALP, and urea) and non-significant difference ($p>0.05$) was observed in creatinine concentration when compared between arghila smokers and control groups.

Table (3) Comparison between some biochemical parameters (AST, ALT, ALP, creatinine, and urea) of healthy individual and Arghila smokers group

	Control group			Arghila(hookah) Smokers group			
	Range	Mean	±SD	Range	Mean	±SD	P-Value
AST (IU/L)	10-32	20.8	7.49	16-198	69.5	61.5	P<0.05
ALT (IU/L)	4-43	16.2	11.3	22-107	66.5	27.7	P<0.05

ALP(IU/L)	19-98	56.7	30.4	15-159	69	40.3	P<0.05
Creatinine (mg/dL)	0.02-0.7	0.342	0.24	0.09-0.7	0.22	0.19	P>0.05
Urea (mg/dL)	14-48	29.3	10.9	21-88	52.1	21.3	P<0.05

Table (4) Comparison of some biochemical parameters of healthy individuals and former smokers (AST, ALT, ALP, creatinine, and urea)

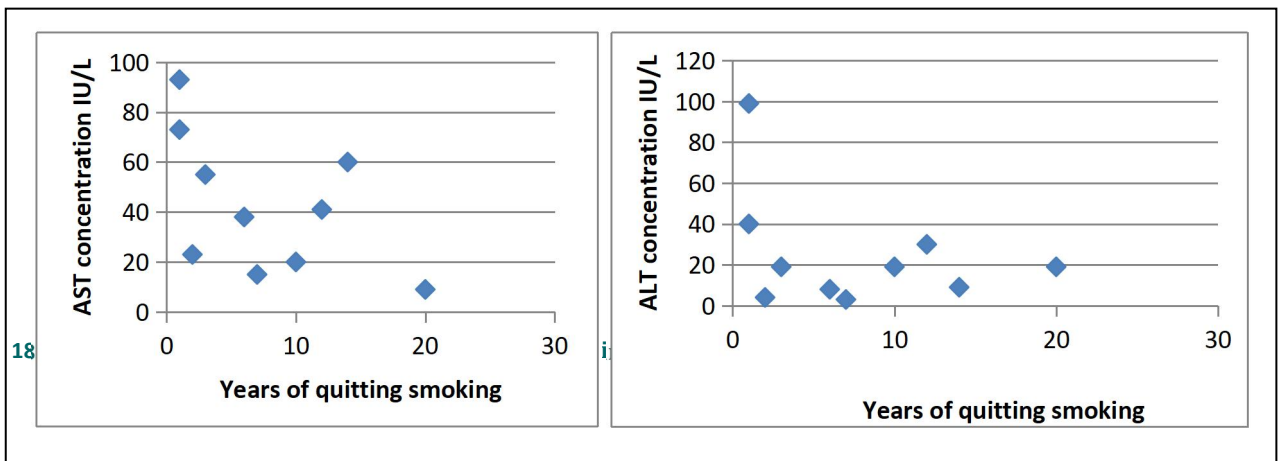
	Control group			Former Smokers group			
	Range	Mean	±SD	Range	Mean	±SD	P-Value
AST (IU/L)	10-32	20.8	7.49	9-93	42.6	29.7	P<0.05
ALT (IU/L)	4-43	16.2	11.3	3-99	25.7	22.7	P<0.05
ALP(IU/L)	19-98	56.7	30.4	23-175	78.5	53.4	P<0.05
Creatinine (mg/dL)	0.02-0.7	0.342	0.24	0.05-1.9	0.733	0.66	P<0.05
Urea (mg/dL)	14-48	29.3	10.9	19-69	45.6	17.5	P<0.05

Table (4) shows significant differences in the parameters studied when compared between former smokers and control groups. While the results in table (5) showed non-significant differences in (ALT and creatinine concentrations) when compared to groups of smokers of arghila and cigarettes ($p>0.05$).

Table (5) Comparison of some biochemical parameters (AST, ALT, ALP, creatinine, and urea) of cigarette smokers and Arghila smokers group.

	Cigarettes Smokers group			Argela Smokers group			
	Range	Mean	±SD	Range	Mean	±SD	P-Value
AST (IU/L)	12-235	81.7	72.1	16-198	69.5	61.5	P<0.05
ALT (IU/L)	17-135	67.5	42.4	22-107	66.5	27.7	P>0.05
-ALP(IU/L)	51-214	95.5	51.8	15-159	69	40.3	P<0.05
Creatinine (mg/dL)	0.02-2.7	0.795	0.92	0.09-0.7	0.22	0.19	P>0.05
Urea (mg/dL)	7-75	37.5	22.4	21-88	52.1	21.3	P<0.05

The results in figure (1) showed noticeable improvement in the parameters studied in the former smokers group as a result of the year's increase in quit smoking.



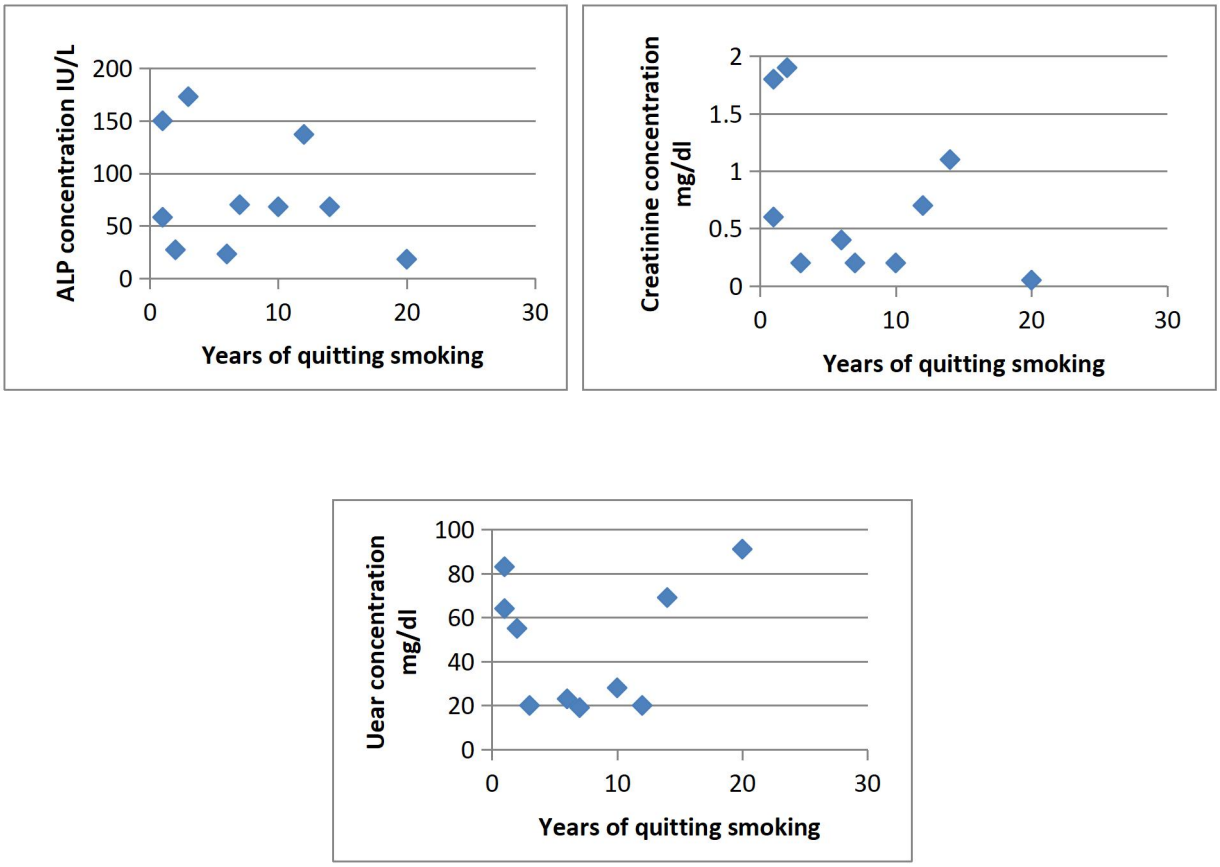


Figure (1) AST, ALT, ALP, Creatinine, and Ura concentrations (IU / L) in the former smokers group based on years of quitting smoking

DISCUSSION

Although cigarette smoke does not come into direct contact with the liver, it incidentally affects the liver. The cigarette chemicals smoke by and by making their way to the liver. These chemicals cause damage to the liver cells and fibrosis. Over time, the liver becomes less effective in removing toxins from the body. This can also prevent the proper use of medicines that may be taken for a particular disease [9].

Liver function tests (LFT): are good clinical practice tools for estimating liver disease, monitoring treatment responses, and predicting the prognosis of patients with liver disease. Most commonly, LFT consists of many tests such as: total protein serum, albumin, total cholesterol (TC), total bilirubin (TB), alkaline phosphatase (ALP), aspartate amino transferase (AST), alanine aminotransferase (ALT), and π-glutamyltransferase (GGT). However, the interpretation of these tests should be inclusive and careful as LFTs can be influenced by many personal and environmental factors, including age, gender, body mass index (BMI) [10, 11], Cigarette smoking[12], alcohol drinking, malnutrition, the presence of extrahepatic diseases such as cardiac, musculoskeletal or endocrine diseases, and the health status of the liver in itself[13]. Serum concentrations of liver enzymes are directly proportional to hepatic cell damage, which includes transferase enzymes: aspartate transferase (AST), alanine transferase (ALT) and alkaline phosphatase (ALP) [9].

ALP is the most routinely measured indicator for diseases of liver bile ducts[14]. AST and ALT enzymes appear regularly in the serum after hepatic cell injury, or sometimes in smaller amounts from destroyed cells. [15]. High levels of liver enzymes may indicate inflammation or damage to hepatic cells[16]. Most people understand clearly the effects of smoking on the heart and lungs, the abundant toxins found in cigarette tobacco lead to chronic inflammation and liver scarring, which in turn increases the risk of damage to liver cells, including diseases such as liver cancer, liver fibrosis, hepatitis B and C, [17]. In addition, smoking affects the function of the liver, such as: alcohol and medication processes, which can increase the risk of alcoholism, as well as the overall levels of drugs and alcohol tolerance [18].,

However, Goya S et al. studied the effect of cigarette smoking and mortality in middle-aged men and found that some cigarette smoking was significantly associated with increased levels of ALP al, in accordance with our findings [19]. Another study conducted by Kurtul N et al. found that there was no statistically significant difference in serum AST levels between smokers and non-smokers, and serum ALT levels were higher in smokers than controls, this result was in agreement with our study on ALT results only. [20]. A potential explanation for increased levels of transaminases in smokers is the interactive effects between smoking and oxidative stress. This presumption has supported this study's significantly higher ALT levels.

Duru et al. suggest that tobacco smoking leads to an increase in blood urea levels due to its effect on the kidney and hepatic cells due to blood contamination by tobacco smoke constituents, and this result agrees with our finding. [21]. In addition to high risk of kidney cancer, smoking can also contribute to additional kidney damage. Smokers are at a significantly higher risk of chronic kidney disease than non - smokers.[22][23]A history of smoking increases the progression of diabetes nephropathy[24, 25].

While all individuals who quit smoking in the current study will benefit from a decreased chance of death by improving their biochemical results, noticeable results have been shown in the above figures.

CONCLUSION

To our knowledge, there are few reports in the literature on the study of biochemical changes in Iraqi smokers who still smoke cigarettes or arghila and formers. Our present study focuses on the relationship between some chemical parameters and the effect of nicotine in cigarette and arghila. The findings suggest that cigarette smoking has a higher effect on liver function compared to arghila, resulting in liver injury indicated by increased levels of liver enzymes. In order to investigate this relationship more deeply in these smokers, further studies must be carried out with a large number of cases.

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