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# THE EFFECT OF CIGARETTE SMOKING ON THE FUNCTIONS OF THE LIVER, KIDNEYS, AND BLOOD ELECTROLYTES

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**Abstract:**
Cigarette smoking is associated with a variety of disorders via effecting various processes, factors, and mechanisms. This study aimed to inspect the differences in renal function tests, liver enzymes, and serum electrolytes among smokers and non-smokers in Iraqi males. In this study, 75 specimens were collected, during the period between January to March 2024, from Males who attended the private clinical laboratory at Al-Anbar province, Iraq.
There was slightly increased in blood urea level among smokers (27.51±9.78 mg/dl) compared to the non-smokers (23.82±5.23 years), but it was not significantly, with a p-value of 0.08>0.05. While serum creatinine was significantly increased among smokers (0.73±0.24 mg/dl) compared to the non-smokers (0.63±0.13 years), with a p-value of 0.05. When comparing smokers to non-smokers, the levels of both AST and ALT are higher in smokers. Regarding electrolytes, there was not significant increase in potassium (K), and not significant decrease in Chloride (Cl), sodium (Na), magnesium (Mg) levels among smokers compared to non-smokers.

**Keywords:** Smoking, Renal Function Test (RFT), Liver Function Test (LFT), Electrolytes

## INTRODUCTION:

In the world, smoking is the most common addiction. Half of smokers' lives are often cut short by 15 years due to tobacco addiction, which is a chronic, recurrent, and deadly condition [1]. By 2025, there will be 1.3 billion daily smokers worldwide, according to predictions from the World Health Organization (WHO). Currently, there are an estimated 1.1 billion smokers worldwide. One of the main ways that people and possibly other living things are exposed to chemically mediated diseases is through tobacco smoke, which is a complex mixture of over 5000 chemicals, carcinogens, and poisons [2]. Because it affects a number of different processes, causes, and systems, cigarette smoking is linked to a wide range of illnesses. The kidney plays a significant role in maintaining glucose homeostasis [3], which depends on the pancreatic beta cells producing enough insulin and the insulin acting appropriately in peripheral tissues. Cigarettes have a number of compounds in them, including cadmium, and they may harm your kidneys. According to previous research, smoking-associated vascular lesions can result from the interaction of these processes and cadmium in cigarette smoke, which can cause vascular endothelial cell death [4]. The liver is a vital organ that performs many functions. Alcohol, drugs, and other poisons are among the things the liver is in charge of eliminating from the body. Although smoking does not directly damage the liver, excessive smoking produces toxins that increase the likelihood of hepatic lesions (fibrosis and activity scores) linked to hepatitis B or C virus infection [5]. excessive smoking also stimulates necroinflammation. One set of changes brought on by smoking is an imbalance in electrolytes. Electrolyte changes happen at the cellular, molecular, and systemic levels. Numerous researchers have examined how smoking affects electrolyte levels [6].

## MATERIALS AND METHODS

About 3 mL of venous blood was collected from all participants and then dispensed into a gel tube, centrifuged at 3000 rpm for 10 min after obtaining clear, transparent sera that have been moved to another labelled tube to measurement these tests: renal function tests (RFT), Liver Function Test (LFT), and serum electrolytes. All parameters had conducted by using Cobas diagnostic kit (Roche/Hitachi Cobas system) at private central lab at Al-Anbar, Iraq.
**The test principle of the B. Urea test** was estimated by Kinetic test with urease and glutamate dehydrogenase [7]. Urease hydrolyzes urea to produce carbonate and ammonium.

$$Urea + 2H_2O \xrightarrow{urease} 2NH_4^+ + CO_3^{2-}$$

In the subsequent step, glutamate dehydrogenase (GLDH) and the cofactor NADH facilitate the reaction between ammonium and 2 oxoglutarate, resulting in the production of L-glutamate. For every mole of urea that is hydrolyzed in this process, two moles of NADH are oxidized to NAD+.

$$NH_4^+ + 2-oxoglutarate + NADH \xrightarrow{GLDH} L-glutamate + NAD^+ + H_2O$$

Photometric measurement is used to determine the rate of decline in NADH concentration, which is directly proportional to the urea content in the material.

**The test principle of the S. Cr test:** This S. Cr test is based on the Jaffé method and its test principle was determined using a kinetic colorimetric assay. A yellow-orange combination between creatinine and picrate is formed in an alkaline solution. The content of creatinine in the sample directly correlates with the rate of dye production. To reduce bilirubin interference, the test employs "rate-blanking." Serum or plasma findings are corrected by (-0.3 mg/dL) to account for non-specific response induced by serum/plasma pseudo-creatinine chromogens, such as proteins and ketones [8].

$$Creatinine + picric\ acid \xrightarrow{Alkaline\ pH} yellow-orange\ complex$$

**Test principle of ALT**

This assay had performance and stability optimized while adhering to IFCC guidelines. The reaction between L-alanine and 2-oxoglutarate is catalyzed by ALT. Lactate dehydrogenase (LDH) catalyzes a process that reduces the pyruvate produced to L-lactate and NAD+.

$$L-Alanine + 2-oxoglutarate \xrightarrow{ALT} pyruvate + L-glutamate$$

$$Pyruvate + NADH + H^+ \xrightarrow{LDH} L-lactate + NAD^+$$

There exists a direct correlation between the rate of NADH oxidation and the catalytic ALT activity. It is determined by calculating the absorbance drop.

**Test principle of AST**

Performance and stability were maximized in this assay while following IFCC regulations.

AST in the sample catalyzes the transfer of an amino group between L-aspartate and 2-oxoglutarate to produce L-glutamate and oxaloacetate. Next, the oxaloacetate joins forces with NADH in the presence of malate dehydrogenase (MDH) to produce NAD+.

$$L-Asparatete + 2-oxoglutarate \xrightarrow{AST} Oxaloacetate + L-glutamate$$

$$Oxaloacetate + NADH + H^+ \xrightarrow{MDH} L-malate + NAD^+$$

The catalytic activity of AST is directly correlated with the rate of NADH oxidation. By calculating the absorbance drop, it is ascertained.

**Test principle of ALP**

colorimetric assay carried out using a defined protocol. Phosphatases break down p-nitrophenyl phosphate into phosphate and p-nitrophenol when magnesium and zinc ions are present.

$$p-nitrophenyl\ phosphate + H_2O \xrightarrow{ALP} phosphate + p-nitrophenol$$

The catalytic ALP activity is directly correlated with the amount of p-nitrophenol released. By monitoring the rise in absorbance, it is ascertained.

**Test principle of Calcium**

Calcium ions react with 5-nitro-5'-methyl-BAPTA (NM-BAPTA) under alkaline conditions to form a complex. This complex reacts in the second step with EDTA.

$$Ca^{2+} + \text{NM-BAPTA} \xrightarrow{\text{alkaline pH}} \text{calcium-NM-BAPTA complex}$$

$$\text{calcium-NM-BAPTA complex} + \text{EDTA} \longrightarrow \text{NM-BAPTA} + \text{calcium EDTA complex}$$

Photometric measurement yields a change in absorbance that is directly proportional to the concentration of calcium.

**Test principle of Magnesium**

Magnesium and xylidyl blue diazonium salt combine to generate a purple complex in an alkaline solution. By using photometry, the concentration of magnesium is determined by calculating the decrease in xylidyl blue absorption.

**Test principle of ISE indirect Na-K-Cl**

To generate an electrical potential (also known as an electromotive force, or EMF) for the purpose of measuring ions in solution, an Ion-Selective Electrode (ISE) uses the special characteristics of an ion-selective membrane. Both an internal filling solution and the test solution come into touch with the selective membrane. The EMF is only influenced by the ions that need to be measured because of the membrane's selectivity. The concentration differential between the test ion in the test solution and the internal filling solution yields the membrane EMF.

**Table 1 The normal range of the study parameters**

| Test | Normal Value |
|---|---|
| **B. Urea** | 15-45 mg/dl |
| **S. Creatinine** | 0.6-1.4 mg/dl |
| **S. ALP** | 32-111 U/L |

| | |
|---|---|
| **S. ALT (GPT)** | Up to 41 U/L |
| **S. AST (GOT)** | Up to 40 U/L |
| **S. Calcium (Ca)** | 8.4-10.2 mg/dL |
| **S. Chloride (Cl)** | 96-106 mmol/L |
| **S. Potassium (K)** | 3.5-5.5 mmol/L |
| **S. Sodium (Na)** | 135-145 mmol/L |

## Statically Analysis

The data were analysed using Microsoft Excel 2024 and IBM SPSS v-26.0. The results reported in this study were expressed as mean ± Standard Deviation (SD), and frequencies were expressed as percentages (%). For comparisons among distributed groups, ANOVA was performed. Probability values less than 0.05 were considered a biologically significant difference, while if less than 0.01 were regarded as highly significant.

## RESULTS AND DISCUSSION

As presented in the table and figure below, most of the smokers in this study those aged elderly than 30 years (40%), while most non-smokers in this study ranged between 25 and 30 years (76%).

**Table 1: The distribution of the data based on age categories**

| Age Categories | Stat | Smokers | Non-smokers | Total |
|---|---|---|---|---|
| **<25yrs** | n | 18 | 6 | 24 |
| | % | 36.00% | 24.00% | 32.00% |
| **25-30yrs** | n | 12 | 19 | 31 |
| | % | 24.00% | 76.00% | 41.30% |
| **>30yrs** | n | 20 | 0 | 20 |
| | % | 40.00% | 0.00% | 26.70% |
| **Total** | n | 50 | 25 | **75** |
| | % | 100.00% | 100.00% | **100.00%** |

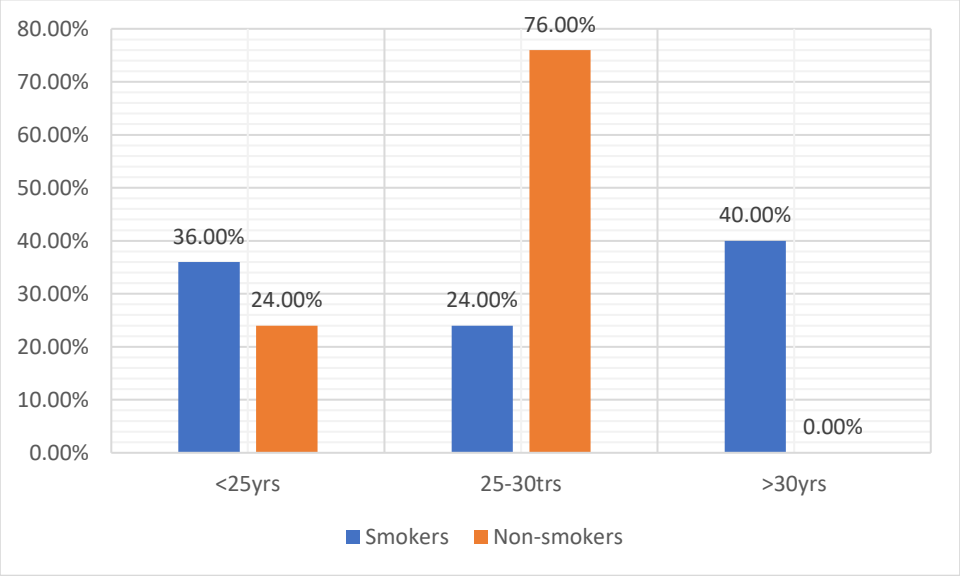


**Figure 1: The distribution of the data based on age categories**

As presented in the table below, the mean age of the smokers in the study were (28.72±5.09 years) elderly than non-smokers (25.96±2.03 years), and there was significant difference between both groups, with a p-value of 0.01. There was slightly increased in blood urea level among smokers (27.51±9.78 mg/dl) compared to the non-smokers (23.82±5.23 mg/dl), but it was not significantly, with a p-value of 0.08>0.05. While serum creatinine was significantly increased among smokers (0.73±0.24 mg/dl) compared to the non-smokers (0.63±0.13 mg/dl), with a p-value of 0.05.

Regarding LFT, there was slightly increased in GOT level among smokers (25.18±17.13 U/L) compared to the non-smokers (20±6.87 U/L), but it was not significantly, with a p-value of 0.1>0.05, while GPT level was significantly increased among smokers (19.67±9.09 U/L) compared to the non-smokers (9.09±3.88 U/L), with a p-value of 0.05. Lastly, ALP level was decreased level among smokers (131.32±6.20 U/L) compared to the non-smokers (145.64±4.96 U/L), but it was not significantly, with a p-value of 0.1>0.05.

Ca level was increased among smokers (11.96±1.59 mg/dL) compared to the non-smokers (8.82±1.25 mg/dL), but it was not significantly, with a p-value of 0.1>0.05.

Regarding electrolytes, the Chloride (Cl) level among smokers was decreased among smokers (94.50±9.46 mmol/L) compared to non-smokers (97.88±7 mmol/L), but it was not significantly, with a p-value of 0.1>0.05. The potassium (K) level was slightly increased among smokers (4.40±0.93 mmol/L) compared to non-smokers (4.18±0.46 mmol/L), but it was not significantly, with a p-value of 0.1>0.05. The sodium (Na) level was slightly decreased among smokers (133.82±1.39 mmol/L) compared to non-smokers (137.88±3.79 mmol/L), but it was not significantly, with a p-value of 0.1>0.05. Also, the magnesium (Mg) level was slightly decreased among smokers (1.83±0.2 mmol/L) compared to non-smokers (1.91±0.16 mmol/L), but it was not significantly, with a p-value of 0.1>0.05.

**Table 2: Average effects of smoking on the studied variables**.

| Variables | Groups | N | Mean | SD | SEM | F | Sig. |
|---|---|---|---|---|---|---|---|
| Age (years) | Smokers | 50 | 28.72 | 5.09 | 0.72 | 6.77 | **0.011** |
| | Non-smokers | 25 | 25.96 | 2.03 | 0.41 | | |
| | Total | 75 | 27.80 | 4.50 | 0.52 | | |
| Urea | Smokers | 50 | 27.51 | 9.78 | 1.38 | 3.104 | 0.082 |
| | Non-smokers | 25 | 23.82 | 5.23 | 1.05 | | |
| | Total | 75 | 26.28 | 8.67 | 1.00 | | |
| S. Cr | Smokers | 50 | 0.73 | 0.24 | 0.03 | 3.98 | **0.05** |
| | Non-smokers | 25 | 0.63 | 0.13 | 0.03 | | |
| | Total | 75 | 0.70 | 0.21 | 0.02 | | |
| GOT | Smokers | 50 | 25.18 | 17.13 | 2.42 | 2.105 | 0.151 |
| | Non-smokers | 25 | 20.00 | 6.87 | 1.37 | | |
| | Total | 75 | 23.45 | 14.69 | 1.70 | | |
| GPT | Smokers | 50 | 19.67 | 9.09 | 1.29 | 5.39 | **0.023** |
| | Non-smokers | 25 | 15.25 | 3.88 | 0.78 | | |
| | Total | 75 | 18.20 | 8.00 | 0.92 | | |
| ALP | Smokers | 50 | 131.32 | 6.20 | 8.80 | 1.003 | 0.32 |
| | Non-smokers | 25 | 145.64 | 4.96 | 9.93 | | |
| | Total | 75 | 136.09 | 5.83 | 6.74 | | |
| Ca | Smokers | 50 | 11.96 | 1.59 | 2.26 | 0.962 | 0.33 |
| | Non-smokers | 25 | 8.82 | 1.25 | 0.25 | | |
| | Total | 75 | 10.91 | 13.09 | 1.51 | | |
| Cl | Smokers | 50 | 94.50 | 9.46 | 1.34 | 2.499 | 0.118 |
| | Non-smokers | 25 | 97.88 | 7.00 | 1.40 | | |
| | Total | 75 | 95.63 | 8.82 | 1.02 | | |
| K | Smokers | 50 | 4.40 | 0.93 | 0.13 | 1.246 | 0.268 |
| | Non-smokers | 25 | 4.18 | 0.46 | 0.09 | | |
| | Total | 75 | 4.33 | 0.81 | 0.09 | | |
| Na | Smokers | 50 | 133.82 | 1.39 | 1.98 | 2.02 | 0.159 |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| | Non-smokers | 25 | 137.88 | 3.79 | 0.76 | | |
| | Total | 75 | 135.17 | 1.17 | 1.36 | | |
| Mg | Smokers | 50 | 1.83 | 0.20 | 0.03 | 3.008 | 0.087 |
| | Non-smokers | 25 | 1.91 | 0.16 | 0.03 | | |
| | Total | 75 | 1.86 | 0.19 | 0.02 | | |

## DISCUSSION

As additional research reveals, smoking can harm and cause renal disorders, it is a well-known risk factor for a number of serious illnesses, such as those of the nervous system, heart, and lungs. Nicotine is a significant part of tobacco smoke and plays an important role in the development of numerous diseases. It causes oxidative damage to the kidney, lungs, liver, and heart. It is a likely oxidant that is capable of producing free radicals and receptive oxygen species [9]. For individuals suffering from chronic conditions such diabetes, hypertension, polycystic kidney disease, and kidney transplant-related illness, smoking exacerbates the severity of renal disorders. Furthermore, even in healthy individuals without a prior history of chronic kidney disease (CKD), smoking can result in de novo renal disease and damage [10]. There was slightly increased in blood urea level among smokers (27.51±9.78 mg/dl) compared to the non-smokers (23.82±5.23 mg/dl), but it was not significantly, with a p-value of 0.08>0.05. While serum creatinine was significantly increased among smokers (0.73±0.24 mg/dl) compared to the non-smokers (0.63±0.13 mg/dl), with a p-value of 0.05. The following are some potential mechanisms by which smoking may specifically affect renal function: smoking may lead to oxidative stress, hardening of glomeruli, and chronic endothelial dysfunction; in vivo research indicates that nicotine inhalation stimulates mesangial cell proliferation [11]; combinations of these mechanisms may result in a reduction in renal function.

Nitric stress, a condition that arises when the production of highly reactive nitrogen-containing chemicals like nitrous oxide surpasses the human body's capacity to neutralize and eliminate them, may be the cause of the increase in liver enzymes. Nitric stress can lead to reactions that alter protein structure and thus interfere with normal body functions. Cigarette smoke contains countless synthetic substances with the potential to poison the liver, including nicotine [12]. This study supported prior studies that found that smokers have greater levels of both AST and ALT when compared to non-smokers. Elevation of the levels of the liver enzymes aspartate aminotransferase (AST or GOT) and alanine aminotransferase (ALT or GPT) due to the harmful effects of tobacco smoke and its constituents on liver cells, which either cause the release of the enzymes through inflammatory pathways or exacerbate the pathogenic effects of other compounds on the liver. Also, another study by Al-Mousawi, et al., (2021) reported that there was a morally high $p<0.05$ in the effectiveness of the enzymes (GOT and GPT) in smokers than controls due to excess nitric oxide effort.

Moreover, it could happen as a result of nitrosative stress, a condition that develops when the body processes more compounds containing highly reactive components than it can neutralize and eliminate, such nitrous oxide. Nitrosative stress disrupts regular bodily processes by initiating the processes that change the structure of proteins. Nicotine is among the numerous hepatotoxic substances found in cigarette smoke.

Significant alterations in the AST and ALT hepatic enzyme activity concentrations between smokers and non-smokers rise in direct proportion to smoking time. According to, a significant concentration of cellular oxidative radicals was released by the liver enzymes AST and ALT, which rose as a result of exposure to smoke or alcohol. Alanine aminotransferase is a crucial enzyme for the synthesis of energy (ALT or GPT). Although it is present in smaller quantities than in the liver, this enzyme can also be found in other tissues in addition to the liver. For instance, the heart and skeletal muscles contain it. They are more useful in the diagnosis of liver disorders such cirrhosis and hepatitis than other liver enzymes [13].

Sodium and potassium assist in maintaining the balance between the body's electrolytes and water. Some important roles of Na+ and K+ are in nerve conduction and muscle contraction. Regarding electrolytes, there was not significant increase in potassium (K), and not significant decrease in Chloride (Cl), sodium (Na), magnesium (Mg) levels among smokers compared to non-smokers in this study. Additionally, a different study by found no significant differences in serum Na+, K+, and Cl-in smokers compared to controls. These findings are consistent with those of Al-Harbi, who discovered that since people consume enough salt in their diets, there are no significant differences in the concentration of Na+ and K+ levels in smokers' blood plasma compared to controls [14].

## CONCLUSION

Smoking is the most widespread addiction worldwide, and it is a preventable risk factor for premature morbidity and mortality.

There was increase in blood urea and serum creatinine levels among smokers compared to the non-smokers. Both AST and ALT have a higher level in smokers if we compare them with non-smokers. Regarding electrolytes, there was not significant increase in potassium (K), and not significant decrease in Chloride (Cl), sodium (Na), magnesium (Mg) levels among smokers compared to non-smokers.

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