A Comparative Study of Biomarker Levels for Iraqi Individuals' Smokers and Non-Smokers Depending on the Duration of Smoking

by 40 Perpustakaan UMSIDA

Submission date: 25-Jul-2024 11:57AM (UTC+0700)
Submission ID: 2422139641
File name: Cek_Plagiasi_Comparative_Study.doc (422K)
Word count: 3530
Character count: 20028

A Comparative Study of Biomarker Levels for Iraqi Individuals' Smokers and Non-Smokers Depending on the Duration of Smoking

ABSTRACT

Smoking is a widespread public health challenge globally, impacting societal health and economy while elevating the risk of cardiovascular and peripheral vascular diseases. This study aims to examine hematological parameters (HB, PCV%, WBC, RBC, PLT, ESR), liver and kidney function, lipid profile, and electrolytes (K+, Na+, Ca++) in male smokers aged 20-55 from Baghdad City, subdivided by smoking duration (≥5 years and <5 years), compared to non-smoking controls. A total of sixty participants were recruited across three areas (Diyala Bridge, Mada'in, Talbieh), with each group comprising thirty individuals. Venipuncture was performed for blood sample collection. Results revealed statistically significant elevations in total WBC and RBC counts, hematocrit, hemoglobin, total cholesterol, and liver enzymes (GOT, GPT, ALP) among smokers compared to non-smokers ($p<0.05$ or $p<0.001$). Serum uric acid, urea, and creatinine levels were significantly higher in smokers ($p<0.001$), while triglyceride levels showed no significant difference ($p>0.05$) between groups. Smokers with longer duration (>5 years) exhibited higher HB, triglyceride, and Na+ levels compared to shorter-term smokers. Sensitivity analysis indicated Na+ as a potentially effective biomarker for smoking-related health impacts, complementing PCV% and urea sensitivity. The study underscores the association between smoking and oxidative stress, notably reflected in elevated GOT and GPT levels, suggesting implications for antioxidant therapies in managing smoking-induced oxidative damage. These findings contribute novel insights into biomarker responses influenced by smoking duration and advocate for targeted public health strategies and further research into antioxidant interventions to mitigate smoking-associated health risks effectively.

Keywords: Hematological parameters, Kidney function, Lipid profile, Electrolytes.

INTRODUCTION

The biggest issue about public health is smoking. Numerous investigations have demonstrated their detrimental impacts on several bodily systems, particularly the cardiovascular and respiratory systems. With 6,000 compounds, it includes, it has pharmacological, carcinogenic, mutagenic, toxic, and inflammatory effects Messner et al. (2014). Numerous oxidants and free radicals found in cigarette smoke can damage proteins, lipids, DNA, carbohydrates, and other macromolecules. It has been shown that smoking affects hematopoietic systems, hormone release, and several metabolic and biological processes Inal et al. (2014). Hematological components develop in peripheral blood tissue after starting their development in the bone marrow. As a result, numerous toxic substances that harm bone marrow and peripheral blood tissue have an impact on them. Research has demonstrated that alterations in hematological parameters, particularly hemoglobin level and WBC count, are linked to conditions like smokers' disease, peripheral vascular disease, and ischemic heart disease Açık (2020). One of the liver's many important jobs is to break down and remove poisons from the body, which includes digesting narcotics, alcohol, and other substances. The liver is among the many organs that suffer harm from cigarette smoking even though they are not in close touch with the smoke. It is well known that liver cancer risk is increased by smoking. Additionally, Burns demonstrated that smoking may impede the development of chronic liver diseases and raise the risk of cirrhosis Pessione et al. (2001). Smoking has clear biological effects because it alters the activity of liver enzymes. GOT, GPT, and ALP activity increased significantly Abdul-Razaq (2013).

Tobacco contains a variety of toxic chemicals, making the kidneys another essential organ affected by smoking, Among these substances that can be obtained by smoking, both actively and passively, is nicotine Orth et al. (2001). Tests for renal function are crucial for diagnosing renal disease, detecting renal malfunction, tracking the course of the illness, and evaluating the effectiveness of treatment. In medicine, renal function is a gauge of the kidney's health and physiological role in the body (nephrology). The majority of medical professionals utilize the plasma concentrations of urea and creatinine to assess renal function. These measurements are sufficient to ascertain if a patient has a renal illness Cooper (2006), early diagnosis of renal affection is crucial for enabling early therapies. Smoking has the potential to cause kidney impairment through various pathways. It has both acute impacts, such as sympathetic stimulation (It decreases GFR and renal plasma flow, increases heart rate and blood pressure, and increases renal vascular resistance), and chronic effects, compromised endothelial cell functioning Cooper (2006). Passive smoking exposure has been linked to heart and blood vessel illnesses, markedly elevated lipid peroxidation in the liver, and elevated kidney catalase activity.

These conditions eventually result in aberrant renal affections and renal blood flow Franceschini et al. (2016). Cigarette smoking also negatively affects the blood lipid profile (LP) levels is seen. Nicotine profoundly impairs the regulation and metabolism of lipids. It is hypothesized that smoking affects smoking-induced atherosclerosis through lipid and lipoprotein abnormalities. Researchers have found that smoking significantly increases triglyceride and total cholesterol (TC) levels Batic-Mujanovic et al. (2008). Nicotine activates the sympathetic adrenal system, which increases catecholamine secretion, lipolysis, and plasma-free fatty acid (FFA) concentration. These factors also increase the release of hepatic triglycerides and free fatty acids, as well as VLDL-C, into the bloodstream. Smoking causes a drop in estrogen levels, which further lowers HDL cholesterol. Because smokers with hyperinsulinemia had decreased lipoprotein lipase activity, their levels of cholesterol, LDL-C, VLDL-C, and TG were greater Alsalhen et al. (2014)

Electrolytes play a vital function in metabolism by acting as catalysts in cellular enzymes. According to reports, electrolytes play a significant part in nerve conduction, blood coagulation, muscle contractions, maintaining normal acid-base balance, and regulating bodily fluid levels. Renal failure, endocrine abnormalities, hepatic illness, and coronary heart disease can all be brought on by electrolyte imbalance ohn (2007). Carbon monoxide, one of the chemicals emitted while smoking, combines with hemoglobin to generate carboxyhemoglobin, which results in hypoxia. This procedure lowers oxyhemoglobin levels, which speeds up blood acidity and disrupts Casasola et al. (2002). The primary goal of the current investigation was to evaluate the connection between the biochemicals' states and cigarette smoking to investigate the changes in hematological parameters (HB, PCV%, WBC RBC, PLT, ESR), liver enzymes (GOT, GPT, ALP), electrolytes like (K+, Na+, Ca++), tests for kidney function that measure creatinine, uric acid, and serum urea levels.

MATERIALS AND METHODS

Study population :

In this study, samples were randomly collated from different areas of Baghdad (Diyala Bridge, Mada'in, and Talbieh) included (30) male individuals who smoked and (30) male non-smokers. The age and gender of the smoker group and the control group were the same. The involved individuals were selected randomly from relatives, family, and friends. The research was carried out between February and June of 2023, with the participants being split into two groups according to the length of time they had been smokers: less than five years (<5) and more than five years (>5). For every case, eight milliliters of blood were drawn into a test tube. The blood was performed in an EDTA-coated tube to evaluate the hematological parameters and the rest of the blood was drowned in a gel

tube, the serum was separated after 30 minutes by centrifuging it for 10 minutes at 3500

rpm/min. The serum was stored in append-rove tubes at 20°C in refrigerators until the test was conducted, estimation of the serum lipid profile, Triglycerides, and Cholesterol respectively Tietz (1999) and Allain et al. (1974), as liver function (GOT, GPT, and ALP), were assessed based on the Reitman and Frankel approach and the Kind and King method respectively. Kidney function (uric acid, urea, and creatinine) provided by Fossati et al. (1980); Searcy et al. (1967) respectively. In UV IS Spectrophotometer SP-3000 nano. Electrolytes such as (Na+, K+, and Ca++) in Electrolyte Analyzer Convergys ISE Comfort Germany. Evaluation of the hematological parameter analyzed by fully automatic hematological analyzers (Drew-D3, USA).

Statistical Analysis :

An independent-sample t-test was used to determine if a difference was highly significant ($p<0.001$), significant ($p<0.05$), or non-significant ($p>0.05$) for comparison. Furthermore, Pearson's correlation analysis is performed to ascertain the associations among all the variables under investigation.

RESULT AND DISCUSSION

When comparing the hematological parameters of smokers and non-smokers, Table (1) and Figure (1) show that smokers have significantly higher levels of hemoglobin and hematocrit ($P<0.001$), as well as a larger count of total white blood cells and red blood cells ($P<0.05$). When compared to the non-smokers, the remaining parameters in a study on the impact of smoking on hematological parameters did not demonstrate statistical significance ($P>0.05$). It has been discovered that smokers have an increased risk of cardiovascular disease, polycythemia, atherosclerosis, and chronic obstructive pulmonary disease. The levels of WBC, Red Blood Cells, HB, and PCV% were seen to be significantly elevated. Atherosclerosis, polycythemia vera, chronic obstructive pulmonary disease, and cardiovascular disorders have all been related to these alterations Asif et al. (2013). A surge in erythropoietin would raise hematological parameters, primarily due to the hypoxia caused by the CO-bound HB's inability to transport O2 to tissues properly Herath et al. (2021) These results indicate a statistically significant increase ($P<0.05$) in the hematological parameters and previous studies Malenica et al. (2017); Herath et al. (2021).

Table 1. The statistical disparities among Smokers and Non Smokers.

	Non-Smokers		Cigarette Smokers		
Parameters	Mean	SD	Mean	SD	P-value
Hb (g/dl)	13.41	1.052	14.903	0.977	0.001* S
PCV %	43.77	2.431	48.33	2.482	0.001* S
WBC 103/µL	6946.66	1331.51	7856.66	1880.33	0.034* S
RBC 106/µL	5.233	0.525	5.469	0.357	0.045* S
ESR (mm/hr.)	14.033	7.393	13.033	9.852	0.658 NS
PLT (109/L)	265.97	91.96	282.033	51.56	0.407 NS
Uric acid (mg/dl)	5.833	1.103	5.5233	0.961	0.250 NS
Urea (mg/dl)	28.167	3.374	34.266	3.311	0.001* S
Creatinine (mg/dl)	0.633	0.092	0.790	0.088	0.001* S
GOT (IU/L)	20.167	3.375	24.86	3.963	0.001* S
GPT (IU/L)	16.767	4.384	22.60	3.191	0.001* S
ALP (IU/L)	40.13	10.798	51.033	13.66	0.001* S
T.G (mg/dl)	104.8	28.392	115.20	29.012	0.165 NS
Cholestrol (mg/dl)	151.63	21.924	167.10	18.55	0.004* S
Na+1 (mEq/L)	129.6	9.0003	142.80	2.398	0.001* S
K+1 (mEq/L)	4.193	0.717	4.853	0.405	0.001* S
Ca+2 (mg/dl)	8.747	1.666	8.996	0.848	0.466 NS

*Significant at P<0.05, NS: Non-Significant.

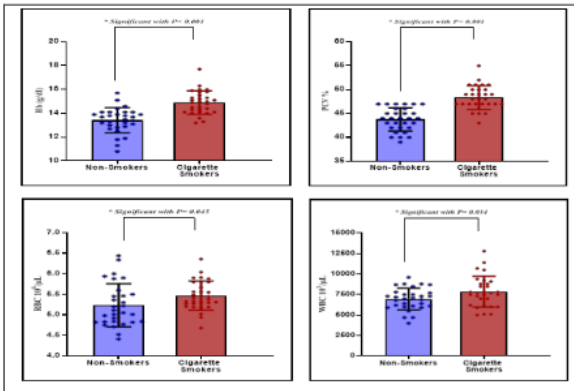

Figure 1. Comparison of blood parameters in male smokers and non-smokers

When we examined the effects of years of smoking on the hematological parameters in our study, we found that male smokers who had smoked for five or more years had significantly higher HB counts ($P<0.001$). However, we were unable to find any effects of smoking on the other hematological parameters. In contrast to men who have smoked for under five years (Table (2) and Figure (2)). our study agreements with Malenica et al. (2017); Jaafar (2020), and Açık (2020) demonstrated that long-term cigarette smoking has a significant negative impact on hematological parameters (such as hemoglobin). This effect may be particularly correlated with the length of smoking, and these changes may be associated with a higher risk of

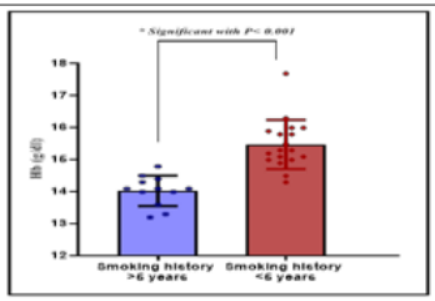

Figure 2. Comparison of HB in male smokers according to duration of smoking.

developing atherosclerosis, polycythemia vera, cardiovascular disease, and/or chronic obstructive pulmonary disease.

It is additionally clear that the values of renal functions which included Serum urea, and creatinine levels were statistically significantly higher, (P<0.001)), but there was no significant (P>0.05) in serum uric acid in the smokers group in comparison to nonsmokers (Table (1) and Figure (3)) also the kidney function levels were no statistically significant in males smoking of 5 or more years in comparison to male smokers for less than 5 years (Table (2)). These results correspond with other earlier studies El Sayed (2013) This might be because tobacco smoking reduces the filtration rate in glomerular tissue (GFR), or the kidney's ability to filter plasma blood by raising resistance to renovascular activity. Lindenfeld et al. (2011).

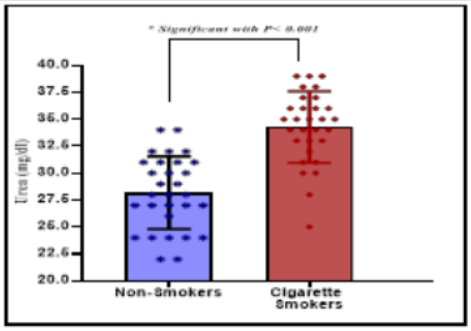

Figure 3. Comparison of urea in male smokers and non-Smokers.

Additionally, when GFR declines, urea reabsorption rises, and the distal tubule's rate flow drops. Another reason why smoking cigarettes may be harmful to the kidneys is because nicotine inhales certain chemicals, like lead (Pb) and cadmium (Cd), which can accumulate in high concentrations and induce nephrotoxicity. Prior research has associated smoking with reduced sympathetic nervous system stimulation, which can result in renal vasoconstriction, smooth muscle cell proliferation, and ultimately renal dam Munzir et al. (2015). This study follows the previous study Tayeb et al. (2020); Kumar et al. (2022). In which they found that there was an elevated value of kidney function in smoking individuals.

Table 2. The statistical disparities among smoking for less and more than 5 years.

	Smoking history >5 years		Smoking history <5 years		
Parameters	Mean	SD	Mean	SD	P-value
Hb (g/dl)	14.03	0.471	15.48	0.771	0.001* S
PCV %	47.58	1.975	48.83	2.706	0.181 NS
WBC 103 µL	7683.33	2032.39	7972.22	1822.99	0.687 NS
RBC 106 µL	5.40	0.302	5.5122	0.392	0.429 NS
ESR (mm/hr.)	14.41	10.569	12.11	9.542	0.539 NS
PLT (109/L)	276.41	47.114	285.77	55.329	0.634 NS
Uric acid	5.22	1.265	5.605	0.787	0.306 NS
		(mg/dl))			
Urea (mg/dl)	33.33	4.119	34.666	2.425	0.273 NS
Creatinine	0.77	0.096	0.794	0.08	0.553 NS
		(mg/dl)			
GOT (IU/L)	23.75	3.816	26.055	3.99	0.126 NS
GPT (IU/L)	21.83	2.406	23.33	3.548	0.211 NS
ALP (IU/L)	49.08	13.681	51.89	13.36	0.581 NS
T.G (mg/dl)	103.66	27.684	128.0	30.11	0.033* S
Cholestrol	163.42	19.355	170.66	19.08	0.319 NS
		(mg/dl)			
Na+1 (mEq/L)	141.00	1.348	144.0	2.196	0.001* S
K+1 (mEq/L)	4.892	0.355	4.855	0.450	0.817 NS
Ca+2 (mg/dl)	8.892	0.789	9.05	0.893	0.622 NS

*Significant at P<0.05, NS: Non-Significant.

The findings demonstrated that serum GOT, GPT, and ALP levels were significantly high (P<0.001) in the smoker's group in comparison to non-smokers (Table (1) and Figure (4)), No statistically significant differences (P>0.05) were noted in GOP, GPT and ALP levels for smoker more 5 years' group comparing with less 5 years' group (Table (2)). Smoking cigarettes can impact the functions and efficiency of the liver cigarette smoke contains countless toxic substances that cause toxic cells of the liver, including nicotine, because it contains free radicals, leading to the events of oxidant effort and increasing the oxidation of fat. This could be due to nitric stress, which occurs when the body produces more highly reactive nitrogen-containing compounds than it can neutralize and eliminate, such as nitrous oxide Pannuru Padmavathi et al. (2009); Alsalhen et al (2014). Our results are similar to what the researchers Hamza et al. (2020); Al-Mousaw et al. (2021) found that the smoke of cigarettes may have some chemicals that play to disturbing liver function and effects of tobacco smoke's chemical components on liver cells that cause them to secrete these enzymes more highly through inflammatory pathways or because other substances' harmful effects on the liver are exacerbated.

A level of lipid profile concentration in both smokers' and non-smokers' blood serum (Table (1)) indicates and graphs (5) the emergence of statistically significant differences (P<0.05) in the concentration of cholesterol serum of smokers compared to control. While

we observed no significant differences (P>0.05) in Triglyceride Levels Table (1).

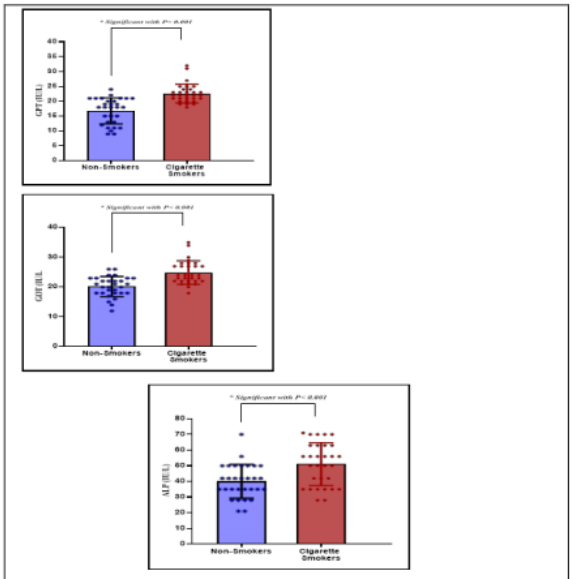

Figure 4. Comparison of liver enzyme in male smokers and non-smokers.

The change in the serum Triglyceride became more marked with the duration time of cigarettes smoked per more than 5 years where statistically significant variations were noted (P<0.05) compared to than less 5 years while Cholesterol was non-significant (P>0.05) according to the time duration smoking (Table (2) and Figure (5)). According to a prior study, smoking excessively and for an extended period might aggravate lipid profile changes and raise blood cholesterol levels, which can result in dyslipidemia Lakshmanan et al. (2014). One possible mechanism for dyslipidemia is that nicotine from cigarettes stimulates the adrenal cortex to release more adrenalin, leading to increased concentrations of free fatty acids and stimulating the liver to synthesize and secrete more cholesterol, followed by the secretion of lipoproteins, proteins that combine and transport fats and lipids in the bloodstream. Another way smoking affects the lipid profile is that it causes hyperinsulinemia by increasing insulin resistance. Hyperinsulinemia is well-known for raising triglyceride and lipoprotein levels. Eliasson (1997). Smokers with hyperinsulinemia have higher cholesterol, LDL, and TG because lipoprotein lipase activity is lowered Bhatt (2003). According to the current study, compared to non-smokers, smokers' total cholesterol levels were noticeably higher. These results agreed with the findings of the previous experiment Devaranavadagi et al. (2012); Hassan (2013); Al-Mousawi et al. (2021).

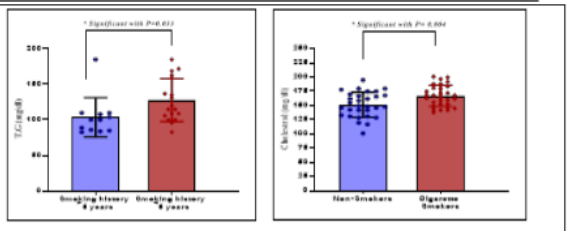

Figure 5. Comparison of lipid profile in male smokers and non-smokers.

Table (1) shows electrolytes (Na+, K+, and Ca++) in the serum of control and cigarette smokers. Cigarette smoking significantly (p< 0.001) affected electrolytes when compared to the non-smokers group. While we detected significant increases (p< 0.001) in Na+ serum levels for smokers in the more 5 years group compared with the less 5 years group also we found No statistically significant differences (P>0.05) in K+ and Ca++ (Table (2) and Figure (6)).

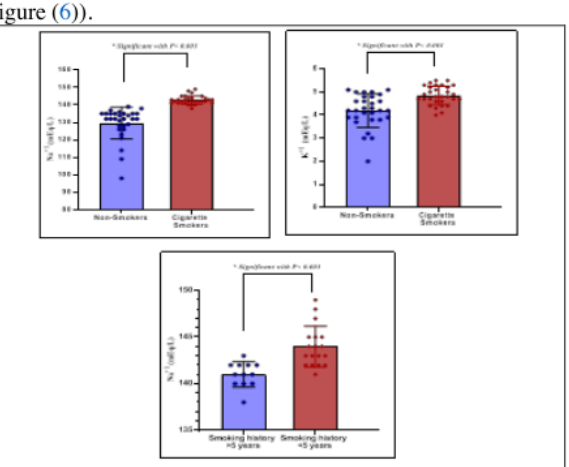

Figure 6. Comparison of electrolytes in male smokers and non-smokers.

Sodium and potassium are essential for the contraction of muscles and the transmission of nerve signals. Hypoxia is a stressor that causes cell development arrest and damage, most likely due to a reduction in the blood's ability to carry oxygen Hang et al. (2005). Respiratory acidosis and electrolyte imbalance may have been caused by elevated carboxyhemoglobin and decreased oxyhemoglobin. Together, potassium and sodium help the body's electrolyte and water balance Nguyen et al. (2004). Furthermore, potassium and sodium are essential for muscular contraction, neuronal conduction, and the passage of molecules across membranes Marsano et al. (1989). It is possible that chronic cigarette smoking changes the transparency of organs' and tissues' membranes, altering signal transmission and maybe causing an electrolyte imbalance Padmavathi et al. (2009). The data from this study concurs with the findings of the research work of Egoro et al. (2018) who reported an elevation in the serum concentration of electrolytes in cigarette smokers. This, however, may be due to tissue and

cell damage caused by chemicals in cigarette smoke, which in turn resulted in cellular contents leakage along with electrolytes into the plasma.

Table 3. The significant Smoker Parameters ROC outcomes.

urameters	AUC	SE	P-value	Cut-off value	Sensitivity	Specificity
b	0.869	0.046	<0.001	13.95	90 %	73.3 %
PCV	0.911	0.035	<0.001	46.5	83.3 %	80 %
WBC	0.647	0.072	0.050	7400	63.3 %	66.7 %
RBC	0.669	0.073	0.025	5.14	90.0 %	53.3 %
ESR	0.433	0.076	0.375	24.5	20.0 %	96.7 %
PLT	0.587	0.077	0.249	200	96.7 %	33.3 %
Uric acid	0.42	0.076	0.287	4.75	83.3 %	26.7 %
Urea	0.903	0.039	<0.001	32.5	76.7 %	93.3 %
Creatinine	0.877	0.044	<0.001	0.75	70.0 %	93.3 %
GOT	0.816	0.053	<0.001	21.5	83.3 %	60.0 %
GPT	0.872	0.044	<0.001	21.5	60.0 %	93.3 %
ALP	0.726	0.066	0.003	53.0	50.0 %	93.3 %
T.G	0.601	0.074	0.181	99.0	73.3 %	53.3 %
Cholestrol	0.693	0.067	0.010	151.0	76.7 %	53.3 %
Na+1	0.998	0.002	<0.001	193.5	96.7 %	100 %
K+1	0.792	0.058	<0.001	4.35	90.0 %	60.0 %
Ca+2	0.624	0.076	0.098	8.95	70.0 %	63.3 %

*Significant at P≤ 0.05, NS: Non-Significant.

According to the ROC results in Table (3) and Figure (7), the electrolytic parameters Na+ had the maximum area under the curve (AUC) at AUC=0.998, which means that it has the highest sensitivity (96.7%) and specificity (100%) that there is sensitivity in PCV%, had a higher diagnostic efficacy among other electrolyte parameters in smoking, while it was less (AUC) for Ca++ and less sensitive.

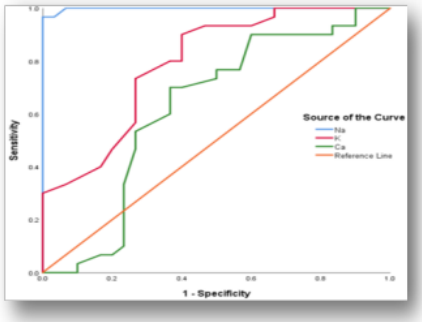

Figure 7. The significant electrolyte Smoker Parameters ROC outcomes.

Following the hematological parameters PCV% where (AUC=0.911) thus has the highest sensitivity (83.3%) and the highest specificity (80%), while it was less (AUC) for ESR and less sensitive (Table (3) and Figure (8)).

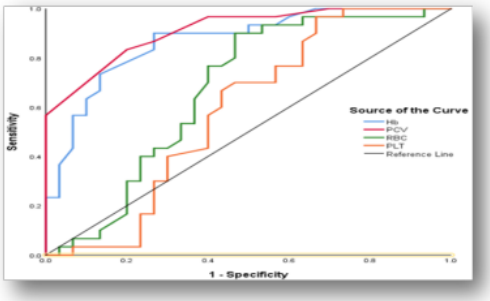

Figure 8. The significant hematological Smoker Parameters ROC outcomes.

The kidney function parameters, urea were (AUC= 0.903) and the highest sensitivity (76.7) and the highest specificity (93.3), while the rest parameters were less sensitive and less specificity (Table (3) and Figure (9)).

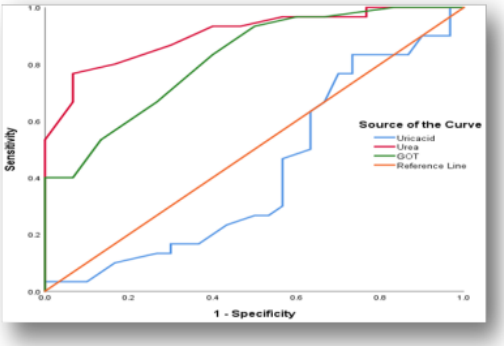

Figure 9. The significant kidney Smoker Parameters ROC outcomes Conclusion.

The correlations were as in the following Table (4) and the highest correlation was between the GOT, and GPT level (r = 0.710** p<0.001) among studied participants in Figure (10).

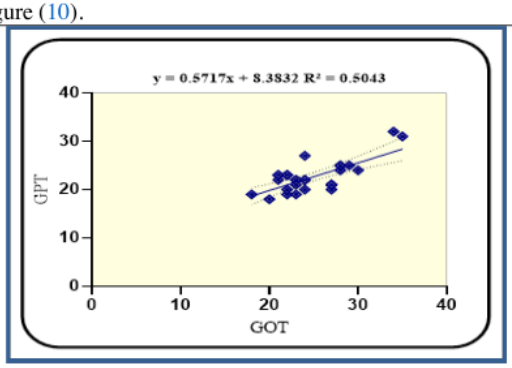

Figure 10. Correlation between GOT and GPT among smokers.

Table 4. The significant correlation parameters in the cigarette smokers group.

Parameters	r	p-value
Hb/PCV	0.585**	0.001
Hb/RBC	0.370*	0.044
PCV/RBC	0.567**	0.000
RBC/Cholesterol	0.417*	0.022
PLT/Cholesterol	-0.457*	0.011
PLT/T.G	-0.396*	0.030
Uricacid/Na+	-0.48**	0.043
Urea/Creatinine	0.680**	0.000
GOT/GPT	0.710**	0.000
GOT/Na+	0.461*	0.010
GPT/ Na+	0.395*	0.031
T.G/ Cholesterol	0.530**	0.003

*Correlation is significant at the 0.05 level (2-tailed).

**Correlation is significant at the 0.01 level (2-tailed).

CONCLUSION

60 oking has negative effects on public health due to the presence of toxic substances that cause the formation of free radicals that affect the effectiveness of the body's organs. In this study, we found an effect on liver function (GOT, GPT, and ALP), hematological parameters (RBCs, HB, and WBCs), lipid profile (triglyceride cholesterol), and kidney function (urea, uric acid, and creatinine). Also, the present study has shown that men who smoke cigarettes for a period of >5 years have significant effects on HB, triglyceride, and Na+. Regarding the ROC results in our result, sensitivity in Na+ had a higher diagnostic efficacy among other parameters in smoking. Following the PCV% and urea, the rest of the parameters are less sensitive and less specific. Further studies are needed to determine whether ıntioxidant enzymes are efficient in identifying the free radicals that smoking produces.

A Comparative Study of Biomarker Levels for Iraqi Individuals' Smokers and Non-Smokers Depending on the Duration of Smoking

ORIGINALITY REPORT

2%	2%	1%	1%
SIMILARITY INDEX	INTERNET SOURCES	PUBLICATIONS	STUDENT PAPERS

PRIMARY SOURCES

1	fdocuments.in Internet Source	1%
2	www2.mdpi.com Internet Source	1%
3	www.mdpi.com Internet Source	1%

Exclude quotes	On	Exclude matches	< 1%
Exclude bibliography	On		