

# CENTRAL ASIAN JOURNAL OF THEORETICAL AND APPLIED SCIENCE



https://cajotas.centralasianstudies.org/index.php/CAJOTAS

*Volume:* 06 *Issue:* 02 | *February* 2024 *ISSN:* 2660-5317

Article

# Effect of Smoking and E-cigarette (Vape) on Liver Enzymes and Lipid Profile in Iraqi People with Dyslipidemia and Liver Disease

Abduqader W. Rasheid\*1

- 1. Ministry of Education/ The Open Educational College, Salah eldin, Iraq.
- \* Correspondence: <a href="mailto:chemistbird2@gmail.com">chemistbird2@gmail.com</a>

**Abstract:** There is an increased trend of e-cigarette but the toxic effects of e-cigarette metabolites are not widely studied especially in liver disease. Study showed evaluate the prevalence and patterns of recent e-cigarette use in a nationally representative sample of Iraqi adults are association with liver disease and revealed a significant association between e-cigarette use, dyslipidemia, in Iraqi adults and adolescents. 88% of e-cigarette users with comorbid liver disease and dyslipidemia exhibited elevated liver enzyme levels, underscoring a potential pathophysiological interplay between lipid dysregulation and hepatotoxicity. Among hepatic biomarkers, serum gammaglutamyl transferase (GGT) demonstrated the strongest independent correlation with lipid profile derangements, positioning GGT and hyperlipidemia as critical biomarkers for monitoring lipidinduced hepatic injury and its clinical progression. liver disease exhibited lower e-cigarette use prevalence compared to non smoker (6.4%) smokers (7.2%), multivariate logistic regression identified e-cigarette use (OR: 1.06; 95% CI: 1.05-1.06, \*p\*<0.0001) and non smoker (OR: 1.50; 95% CI: 1.50-1.51, p<0.0001) as independent risk factors for liver disease history. Paradoxically, despite lower absolute e-cigarette use frequency in liver disease cohorts, the elevated odds ratios highlight a disproportionate risk burden in dyslipidemic individuals. Additionally, traditional cigarette smokers showed significantly higher hemoglobin (Hb) levels than e-cigarette users, implying divergent systemic effects of smoking types.

**Keywords:** E-cigarette, Vape Smoking, Liver Disease, Liver Toxicity, Biochemistry Marker, Dyslipidemia

Citation: Rasheid, A. W. Effect of Smoking and Ecigarette (Vape) on Liver Enzymes and Lipid Profile in Iraqi People with Dyslipidemia and Liver Disease. Central Asian Journal of Theoretical and Applied Science 2025, 6(2), 99-107.

Received: 12th Jan 2025 Revised: 23th Jan 2025 Accepted: 30th Jan 2025 Published: 8th Feb 2025



Copyright: © 2025 by the authors. Submitted for open access publication under the terms and conditions of the Creative Commons Attribution (CC BY) license (https://creativecommons.org/licenses/by/4.0/)

#### 1. Introduction

Liver disease is a significant global health burden, accounting for over two million deaths annually, including cases of cirrhosis, viral hepatitis, and hepatocellular carcinoma [1]. This constitutes approximately 4% of all deaths worldwide, equating to one in every 25 deaths [2]. One-third of liver-related fatalities occur among females. Within this mortality estimate, liver cancer contributes to approximately 600,000 to 900,000 deaths annually [3]. Currently, liver disease ranks as the eleventh-leading cause of death globally, although this may be an underestimation due to challenges in accurate reporting [4]. Cirrhosis remains a leading cause of mortality, ranking tenth in Africa (up from thirteenth in 2019), ninth in South East Asia and Europe, and fifth in the Eastern Mediterranean region. Globally, liver cirrhosis ranks among the top 20 causes of Disability-Adjusted Life Years (DALYs) and Years of Life Lost (YLLs) [5]. Subsequent studies have reported that smoking is an independent risk factor for liver cirrhosis, irrespective of alcohol

consumption [6]. The mechanisms underlying the hepatotoxic effects of smoking are multifaceted, involving the activation of stellate cells via nicotinic acetylcholine receptors [7], increased production of pro-inflammatory cytokines [8], and secondary iron overload due to polycythemia, leading to oxidative stress, necro inflammation, apoptosis, and excess iron deposition in the liver [9]. In recent years, electronic nicotine delivery systems (ENDS), also known as e-cigarettes, have emerged as a popular alternative to traditional smoking, despite a lack of robust clinical evidence regarding their safety. A study by Hassan et al demonstrated that exposure to nicotine aerosol significantly increased hepatic lipid accumulation, oxidative stress, and apoptosis compared to placebo aerosol [10]. Additional studies have reported that nicotine exposure, particularly when combined with a high-fat diet, can exacerbate oxidative stress, hepatocyte apoptosis, and hepatic statuses in animal models [11], [12]. The dearth of research on the impact of e-cigarettes on liver health, particularly in the context of liver fibrosis or cirrhosis, necessitates an investigation into the effects of e-cigarette use on liver disease. This knowledge gap is particularly pertinent, as the global prevalence of e-cigarette use continues to rise [13]. This study aimed to address this knowledge gap by investigating the prevalence and correlates of ecigarette use among respondents with liver disease in Iraq. Specifically, the primary objective was to determine the prevalence of e-cigarette(vape) use, dyslipidemia, and some chronic disease as diabetes and hypertension among respondents with liver disease, the secondary objective was to evaluate the association between e-cigarette(vape) use relation with liver disease and some biochemistry parameters in patients and healthy people, while adjusting for confounding variables and examining the effect of e-cigarette use on liver enzyme concentrations in patients. To last knowledge, this is the first study to investigate e-cigarette use in respondents with liver disease in Iraq, using a well-designed survey that includes information on e-cigarette use and liver disease characteristics. The findings of this study will contribute to the growing body of research on the health effects of ecigarette use, particularly among vulnerable populations such as those with liver disease.

### 2. Materials and Methods

Design of the "study and selection of participants: Between December 2023 and February 2024, a cross-sectional population-based study was carried out in the Department of Chemistry, College of Science, Tikrit University, Iraq. 200 participants in all were chosen at random from among the general patients at the Tikrit Scientific Hospital, including those with liver disease and other illnesses.. There were 112 male individuals and 88 female subjects. The following are requirements for inclusion: (1) both sexes (18 years of age and older), (2) no serious or chronic illnesses, and (3) willingness to take part in the study. Criteria for exclusion: (1) women who are pregnant or nursing (2) individuals with a history of alcoholism or hepatotoxic drug use, either current or previous (3) self-reported viral hepatitis, and (4) participants who did not fill out the questionnaire or donate blood samples. Prior to their involvement in the study, all individuals gave their informed consent. Measurements of body weight, height, hip circumference (HC), and waist circumference (WC) are all part of anthropometry. These measurements were taken using the usual protocols outlined elsewhere [14], [15]. In a nutshell, digital weighing devices (Beurer 700, Germany) were used to measure the body weight to the closest 0.1 kg [16]. Using a measuring tape, the standing body height (stature) was determined to the closest 0.1 cm. A person's BMI was determined by dividing their height in meters squared (m2) by their weight in kilograms (kg). A digital blood pressure monitor (Omron, Tokyo, Japan) was used to record the systolic (SBP) and diastolic (DBP) readings. Each subject's mean blood pressure reading from two consecutive readings was determined and used in the study Sample collection and biochemical analysis: Using disposable needles and syringes, each participant had approximately 5 ml of fasting blood drawn via venipuncture. Following blood sample centrifugation, serum was extracted and kept at -80 °C until biochemical parameter measurement. Standard colorimetric techniques were used to test the serum levels of triglycerides (TG), total cholesterol (TC), low-density lipoprotein (LDL), high-density lipoprotein (HDL), and fasting blood glucose (FBG). Kinetic techniques were used to quantify the evels of serum  $\gamma$ -glutamyltransferase (GGT), alkaline phosphatase (ALP), and alanine and aspartate aminotransferase (ALT and AST). With the exception of the GGT from Aflu (Italy) to assess the biochemical parameters, all of the available diagnostic kits were acquired from Human Diagnostic (Germany) [17]. A semi-automated biochemistry analyzer (Aflu 2000, Italy) was used for all biochemical analyses. Every analysis was carried out in compliance with the guidelines provided by the manufacturer. Regular calibration using the reference standards included in the kits verified the inter-laboratory precision and accuracy" of all measurements.

### 3. Results

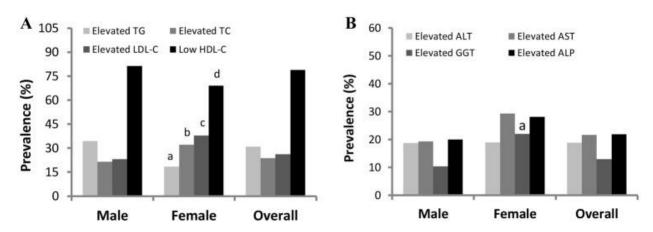
Table 1 provides a summary of the study participants baseline characteristics by gender. Of the 200 people who signed up, 88 were women and 112 were men. Male participants averaged  $40.5\pm12.7$  years, whereas female participants averaged  $40.6\pm13.4$  years. The participants' average age was  $40.5\pm12.9$  years. Serum levels of total cholesterol (TC), triglycerides (TG), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), fasting blood glucose (FBG), and alkaline phosphatase (ALP) were significantly different between the male and female groups, according to biochemical analysis (p < 0.05 for all parameters). Body mass index (BMI), waist circumference (WC), hip circumference (HC), systolic and diastolic blood pressure (DBP), alanine aminotransferase (ALT), aspartate aminotransferase (AST), and gamma-glutamyl transferase (GGT) did not differ statistically significantly between the sexes. The prevalence of diabetes and hypertension was higher in women (46% and 49.4%, respectively) than in men (41.6% and 29.9%). In terms of smoking behaviors, all female participants reported not smoking, while roughly 29% of male participants were avid ecigarette users.

Table 1. Baseline characteristics of the study participants.

Variables	Male (n = 112)	Female (n = 88)	Total $(n = 200)$	<i>P</i> -value
Age (years)	$41.5 \pm 11.7$	$40.6 \pm 13.4$	$40.5 \pm 12.9$	0.949
Weight (kg)	$66.1 \pm 11.7$	$58.3 \pm 9.7$	$65.2 \pm 11.1$	< 0.001
Height (cm)	$164.4 \pm 7.8$	$152.7 \pm 7.9$	$162.7 \pm 8.7$	< 0.001
BMI $(kg/m^2)$	$23.5 \pm 4.4$	$25.1 \pm 4.1$	$24.6 \pm 3.5$	0.183
WC (cm)	$85.0 \pm 11.1$	$85.7 \pm 10.9$	$85.9 \pm 11.8$	0.842
HC (cm)	$93.1 \pm 7.2$	$92.5 \pm 8.9$	$92.2 \pm 8.4$	0.743
SBP (mmHg)	$127.9 \pm 13.5$	$126.2 \pm 18.8$	$126.7 \pm 15.5$	0.714
DBP (mmHg)	$82.6 \pm 10.9$	$83.2 \pm 10.1$	$83.5 \pm 9.9$	0.738
PP (beats/min)	$75.4 \pm 13.5$	$82.3 \pm 12.1$	$77.7 \pm 12.6$	< 0.001
Glucose (mg/dL)	$119.8 \pm 58.4$	$136.8 \pm 70.2$	$122.4 \pm 63.0$	0.013
TG (mg/dL)	$193.6 \pm 113.4$	$153.2 \pm 94.6$	$185.7 \pm 110.0$	0.001
TC (mg/dL)	$204.3 \pm 72.4$	$228.3 \pm 92.0$	$208.7 \pm 78.3$	0.021
LDL (mg/dL)	$134.6 \pm 61.3$	$162.7 \pm 89.8$	$139.9 \pm 70.0$	0.001
HDL (mg/dL)	$31.8 \pm 13.9$	$35.9 \pm 9.7$	$33.4 \pm 12.6$	0.015
ALT (U/L)	$33.5 \pm 16.2$	$31.7 \pm 25.8$	$33.9 \pm 19.4$	0.360
AST (U/L)	$28.0 \pm 11.0$	$29.0 \pm 17.1$	$27.4 \pm 13.4$	0.414
GGT (U/L)	$33.8 \pm 31.2$	$29.6 \pm 28.2$	$32.1 \pm 29.8$	0.380
ALP (U/L)	$101.6 \pm 42.3$	$87.9 \pm 35.0$	$97.6 \pm 39.5$	0.023
Hypertensive (%)	41.4	46.7	43.2	0.480
Diabetic (%)	31.9	48.7	35.2	0.001
Physical activity (%)				
Low	20.3	26.4	21.7	0.336
Medium	68.9	67.8	68.4	

Adequate	10.3	4.7	8.6	
Smoking e-cigar	ette status (%)			
Yes	100	60.8	78	< 0.001
No	29.2	0	22	

Figure 1 shows the prevalence of high liver enzyme levels and e-cigarette use by gender among male and female individuals. Increased triglycerides (TG) at 30.9%, total cholesterol (TC) at 23.7%, low-density lipoprotein cholesterol (LDL-C) at 26.2%, and decreased high-density lipoprotein cholesterol (HDL-C) at 78.8% were the overall prevalence of e-cigarettes. While female individuals had a higher frequency of elevated TC and LDL-C levels, male participants had a higher prevalence of elevated TG levels and decreased HDL-C. Elevated levels of alanine aminotransferase (ALT), aspartate aminotransferase (AST), gamma-glutamyl transferase (GGT), and alkaline phosphatase (ALP) were seen in 18.8%, 21.6%, 12.9%, and 21.9% of cases with liver enzyme abnormalities, respectively. Of these, elevated AST, GGT, and ALP levels were significantly more common in female individuals than in male participants. Figure 1 shows the prevalence of liver enzyme abnormalities and dyslipidemia in various sex groups.



**Figure 1.** Shows the gender group's prevalence of e-cigarettes (A) and high liver enzymes (B). As opposed to the male group, the prevalence in (A) is a,cp<0.05 and b,dp<0.01. When comparing the prevalence to the male group, ap is less than 0.01 in (B). The Chi Square test yields P-values.

Rates of Liver Dysfunction and E-Cigarette Use by Health Status Group The prevalence and mean levels of liver enzymes and lipid markers in various health status categories are compiled in Table 2. Compared to the healthy control group, persons with diabetes and hypertension showed noticeably increased prevalences and levels of aberrant lipid markers as well as elevated liver enzymes.

**Table 2.** Prevalence and Levels of Lipid Markers and Liver Enzymes Across Health Status Groups.

Variables	Overall (n = 200)	Healthy $(n = 65)$	Hypertensive (n = 86)	P-value <sup>a</sup>	Diabetic (n = 82)	P-value <sup>b</sup>
TG (mg/dL)	$185.7 \pm 110.0$	$163.6 \pm 95.1$	$200.3 \pm 116.3$	0.005	$211.0 \pm 127.4$	0.001
Elevated TG, n (%)	125 (30.9)	39 (22.3)	61 (35.5)	0.021	56 (40.6)	0.001
TC (mg/dL)	$208.7 \pm 78.3$	$183.5 \pm 52.0$	$230.4 \pm 91.5$	< 0.001	$235.7 \pm 101.9$	< 0.001
Elevated TC, n (%)	96 (23.7)	17 (9.7)	58 (33.7)	< 0.001	51 (37)	< 0.001
LDL (mg/dL)	$139.9 \pm 70.0$	$120.4 \pm 46.9$	$157.1 \pm 80.9$	< 0.001	$159.9 \pm 94.9$	< 0.001

Variables	Overall (n = 200)	Healthy (n = 65)	Hypertensive (n = 86)	P-value <sup>a</sup>	Diabetic (n = 82)	P-value <sup>b</sup>
Elevated LDL, n (%)	106 (26.2)	27 (15.3)	61 (35.5)	< 0.001	49 (35.5)	< 0.001
HDL (mg/dL)	$33.4 \pm 12.6$	$31.0 \pm 9.0$	$35.3 \pm 14.4$	0.002	$36.6 \pm 16.4$	< 0.001
Low HDL, n (%)	319 (78.8)	150 (85.2)	128 (74.4)	0.010	95 (68.8)	0.001
ALT (U/L)	$33.9 \pm 19.4$	$30.6 \pm 14.5$	$36.6 \pm 23.0$	0.014	$36.7 \pm 22.2$	0.006
Elevated ALT, n (%)	75 (18.8)	27 (15.3)	37 (22)	0.146	33 (25)	0.039
AST (U/L)	$27.4 \pm 13.4$	$23.7 \pm 10.9$	$30.5 \pm 15.7$	0.003	$32.2 \pm 15.2$	< 0.001
Elevated AST, n (%)	53 (21.6)	17 (15.2)	25 (26.3)	0.162	28 (28.9)	0.043
GGT (U/L)	$32.1 \pm 29.8$	$22.6 \pm 13.2$	$41.0 \pm 37.3$	< 0.001	$44.2 \pm 39.8$	< 0.001
Elevated GGT, n (%)	50 (12.9)	6 (3.6)	36 (21.7)	< 0.001	35 (26)	< 0.001
ALP (U/L)	$97.9 \pm 39.5$	$93.5 \pm 30.5$	$100.3 \pm 41.0$	0.505	$103.3 \pm 47.8$	0.150
Elevated ALP, n (%)	53 (21.9)	16 (14.5)	24 (25.8)	0.162	32 (33.3)	0.004

The frequency of elevated liver enzymes in adults with dyslipidemia: The data are displayed as a percentage (%) or mean  $\pm$  SD. The statistical difference between the hypertensive and healthy groups is represented by the P value, whilst the difference between the diabetic and healthy groups is indicated by the P value. The Chi-square test was used to evaluate prevalence data (%) and the independent sample t-test was used to investigate mean concentrations. According to Table 3, almost 73% of e-cigarette users with liver disorders and dyslipidemia—which is characterized by one or more raised lipid levels—had at least one elevated liver enzyme level. Individuals with elevated levels of triglycerides (TG), except for AST and ALP, and total cholesterol (TC), except for ALT, were substantially more likely to have these hepatic enzyme abnormalities than those with normal TC and TG levels (p < 0.05 for all comparisons). Additionally, AST concentrations were substantially greater in those with elevated low-density lipoprotein (LDL) levels than in those with normal LDL levels (p < 0.05).

**Table 3.** Prevalence of Elevated Liver Enzymes in Dyslipidemia Adults.

T		TG	r		TC			LDL				HDL		
Variab	les	Noi	rmal	Elevated	<i>P</i> -value	Normal	Elevated	<i>P</i> -value	Normal	Elevated	<i>P</i> -value	Normal	Low	<i>P</i> -value
Elevated A	ALT	15.6		25.2	0.023	16.8	25.3	0.065	18.7	19.0	0.939	20.5	18.4	0.659
Elevated AST	20.1	25.0	0.391	16.4	32.	5 0.00	)4 17.5	3	0.4 0.0	22 20.3 2	22.0	0.782		
Elevated GGT	9.8	19.7	0.007	9.1	24.	7 < 0	.001 11.5	1	6.5 0.1	97 15.4	12.	.2 0.45	55	
Elevated ALP	18.5	29.7	0.051	18.0	30.	7 0.02	27 19.2	2	8.0 0.1	24 16.4	23.	.5 0.25	59	

Relationship Between E-cigarette Users' Liver Enzymes and Lipid Markers: The Chisquare test was used to calculate the p-values, and the data are presented as percentages (%). All measured liver enzymes, including alanine aminotransferase (ALT), aspartate aminotransferase (AST), gamma-glutamyl transferase (GGT), and alkaline phosphatase (ALP), showed a significant positive correlation with serum triglycerides (TG) in regression models (p < 0.05 for all cases; Table 4). Likewise, there was a substantial positive correlation (p < 0.05) between total cholesterol (TC) and ALT, AST, and GGT. ALT and

GGT were positively correlated with low-density lipoprotein cholesterol (LDL-C) (p < 0.05 in all cases).

High-density lipoprotein cholesterol (HDL-C), on the other hand, only significantly correlated negatively with GGT (p < 0.01 in model 1; p < 0.05 in models 2–3), suggesting that HDL levels and GGT activity are inversely related.

**Table 4.** Association Between Lipid Markers and Liver Enzymes.

-	ALT		AST		GGT		ALP	
	OR (95% CI)	P for trend	OR (95% CI)	P for trend	OR (95% CI)	P for trend	OR (95% CI)	P for trend
				TG				
Model 1	1.06 (1.00– 1.17)	0.007	1.08 (1.00– 1.13)	0.008	1.11 (1.08– 1.14)	0.006	1.04 (1.01– 1.06)	0.008
Model 2	1.07 (1.01– 1.17)	0.022	1.08 (1.01– 1.16)	0.025	1.12 (1.08– 1.14)	0.021	1.04 (1.00– 1.08)	0.026
Model 3	1.20 (1.22– 1.48)	0.024	1.10 (1.22– 1.37)	0.022	1.13 (1.03– 1.24)	0.022	1.05 (1.00– 1.07)	0.018
				TC				
Model 1	1.04 (1.00– 1.22)	0.009	1.07 (1.01– 1.13)	0.004	1.10 (1.08– 1.14)	0.008	0.98 (0.98– 1.00)	0.284
Model 2	1.08 (1.01– 1.13)	0.024	1.07 (1.01– 1.13)	0.026	1.10 (1.08– 1.14)	0.023	0.98 (0.97– 1.01)	0.212
Model 3	1.05 (1.01– 1.19)	0.018	1.09 (1.21– 1.37)	0.024	1.11 (1.03– 1.24)	0.021	0.96 (0.97– 1.01)	0.213
				LDL				
Model 1	1.02 (1.00– 1.09)	0.012	1.04 (0.99– 1.08)	0.094	1.04 (1.00– 1.07)	0.023	0.99 (0.96– 1.00)	0.268
Model 2	1.04 (1.01– 1.07)	0.026	1.03 (0.98– 1.08)	0.132	1.05 (1.00– 1.09)	0.019	0.98 (0.99– 1.01)	0.209
Model 3	1.05 (1.01– 1.09)	0.022	1.05 (0.99– 1.12)	0.118	1.06 (1.01– 1.11)	0.014	0.99 (0.99– 1.00)	0.209
				HDL				
Model 1	0.99 (0.98– 1.00)	0.269	0.94 (0.97– 1.00)	0.286	- 1.04 (1.01- 1.07)	0.007	- 0.97 (0.98- 1.00)	0.269
Model 2	0.98 (0.96– 1.01)	0.208	0.95 (0.98– 1.01)	0.213	- 1.04 (1.00- 1.07)	0.023	- 0.99 (0.97- 1.01)	0.207
Model 3	0.99 (0.96– 1.00)	0.207	0.93 (0.96– 1.00)	0.213	- 1.05 (1.00- 1.09)	0.019	- 0.98 (0.97- 1.00)	0.208

Multivariate Logistic Regression Analysis: Multivariate logistic regression was used to evaluate the association between liver enzyme levels and lipid profile indicators. This study included liver enzyme levels (alanine aminotransferase [ALT], aspartate aminotransferase [AST], gamma-glutamyl transferase [GGT], and alkaline phosphatase [ALP]) as independent variables and lipid profile markers (total cholesterol [TC], triglycerides [TG], low-density lipoprotein cholesterol [LDL-C], and high-density lipoprotein cholesterol [HDL-C]) as dependent variables. To account for potential confounders, the regression analysis used three models: Age and gender were taken into account in Model 1, body mass index (BMI), waist circumference (WC), and fasting blood glucose (FBG) were taken into account in Model 2, and systolic blood pressure (SBP), diastolic blood pressure (DBP), and levels of physical activity were taken into account in Model 3. The regression models, which took into consideration a variety of physiological, anthropometric, and demographic factors, provide a thorough assessment of the relationship between changes in the lipid profile and liver enzyme activity.

#### 4. Discussion

Patients with liver problems linked to e-cigarettes had a significant prevalence of dyslipidemia in the current investigation. Serum gamma-glutamyl transferase (GGT), one of the liver enzymes examined, showed an independent correlation with every lipid measure. To the best of our knowledge, this is the first study to discuss the connection between liver enzymes and lipid profile markers in Iraqi e-cigarette users [18]. The percentage of subjects with at least one increased lipid level was about 88%. Previous studies conducted in iraq have reported the prevalence of dyslipidemia among between 20.9% and 75.6% for adults. Different study populations, geographical locations, socioeconomic level, age groups, genetic predispositions, lifestyle factors, and the use of different diagnostic cut-off values for dyslipidemia could all contribute to the variation in prevalence rates. A number of research have also demonstrated a clinical correlation between disorders including diabetes and hypertension and aberrant lipid profiles [18], [19]. Several earlier studies have documented the prevalence of dyslipidemia among Iraqis in The 256 participants in the study had an overall prevalence of 80.3% for dyslipidemia, which is defined as the presence of at least one abnormal lipid parameter. This high prevalence highlights the significance of routine lipid profile assessments in clinical practice, especially for cardiac patients [20]. This is consistent with a study in Kurdistan that found that the middle age group had a higher prevalence of heart disease than younger and older age groups, and that diabetes and cigarette smoking were more common among older age groups, while dyslipidemia was more common among younger and older age groups. This is because a significant portion of the study participants had hypertension (42.6%) and diabetes (34.1%), which may have contributed to the higher prevalence of dyslipidemia observed in this cohort. Hypertension, alcohol, and physical inactivity were comparable across age groups. The most common risk factors for CVDs in this area were physical inactivity, diabetes, hypertension, and dyslipidemia. Some of these risk variables were substantially more prevalent in the elderly patients [21], [22]. The prevalence of low HDL cholesterol in this study was 78.8%, which is in line with other research showing that HDL cholesterol was the lipid parameter most commonly impacted. Compared to other lipid abnormalities, some research indicate that Iraqi populations may be more susceptible to elevated LDL cholesterol levels [23], [24]. Lipid levels and liver enzyme concentrations were also shown to differ by gender. While female individuals had greater levels of total cholesterol (TC) and low-density lipoprotein (LDL), male participants had considerably higher levels of blood triglycerides (TG) and lower HDL cholesterol. Like many other Middle Eastern nations, Iraq is fast becoming more urbanized and industrialized [25]. Although some research has connected urbanization to higher rates of dyslipidemia, this study's analysis did not discover a significant correlation between urbanization and the prevalence of dyslipidemia among e-cigarette users. Rather, dyslipidemia was more common in rural areas, possibly as a result of poor dietary practices, a lack of health knowledge, and limited access to medical care. To investigate these issues more thoroughly, more research is required [26]. Both dyslipidemia and liver enzyme abnormalities were much more common in hypertensive and diabetic groups than in healthy controls among all e-cigarette users. These abnormalities were even more noticeable in those who had both diabetes and hypertension, highlighting the role that these conditions play in aggravating hepatic dysfunction and dyslipidemia [27], [28], [29]. Prior research has also documented comparable results, associating dyslipidemia with aberrant liver function test indicators, which are frequently suggestive of non-alcoholic fatty liver disease (NAFLD) [30], [31]. Participants with dyslipidemia in this study had a noticeably high prevalence of increased liver enzymes, which is in line with results from other populations where abnormal liver function tests were highly linked to dyslipidemia, especially in cases with NAFLD. This result was consistent with a study conducted in the United States that found that patients with chronic liver disease had a considerably increased risk of using e-cigarettes, even though e-cigarette usage was very uncommon

among those with a history of liver disease. These results emphasize the need for additional prospective research to fully assess how e-cigarettes affect people with liver disease and to clarify the exact mechanisms by which toxicants originating from e-cigarettes disrupt hepatic function. The findings also highlight how crucial it is that when developing regulations for e-cigarette use, public health professionals and legislators take into account solid data regarding the hepatotoxic effects of e-cigarettes. These findings demonstrate the interaction between liver health and metabolic diseases in e-cigarette users, indicating the need for additional research into the underlying mechanisms [32], [33]. According to the study, traditional cigarette smokers had hemoglobin (Hb) levels that were noticeably greater than those of e-cigarette users. This result is consistent with a research by Enc et al. that found smokers with advanced liver fibrosis and non-alcoholic fatty liver disease had higher hemoglobin levels. The persistent hypoxic circumstances brought on by smoking, which result in compensatory erythropoiesis, may be the cause of this rise in Hb levels in traditional smokers [34].

#### 5. Conclusion

According to this study, dyslipidemia and liver enzyme abnormalities—specifically, increased serum GGT—are significantly more common in Iraqi people with diabetes and hypertension than in healthy individuals. Serum GGT and lipid profile markers have a high correlation, which implies that raised lipid and GGT levels could be important biomarkers for tracking the severity and course of lipid-induced hepatic dysfunction and its related consequences. These results highlight the fact that people with dyslipidemia have a higher chance of getting liver disease than people without the condition. In order to enhance clinical outcomes and guide targeted therapies, more extensive prospective studies are necessary to clarify the underlying processes of lipid-induced hepatic dysfunction in the Iraqi population.

## REFERENCES

- [1] S. K. Asrani, H. Devarbhavi, and J. Eaton, "Burden of liver diseases in the world," *J. Hepatol.*, vol. 70, pp. 151–171, 2019.
- [2] C. Griffin, U. Agbim, and A. Ramani, "Underestimation of cirrhosis-related mortality in the Medicare-eligible population, 1999-2019," *Clin. Gastroenterol. Hepatol.*, vol. 21, pp. 223–225, 2021.
- [3] GBDC Collaborators, "The global, regional, and national burden of cirrhosis by cause in 195 countries and territories, 1990-2019: A systematic analysis for the Global Burden of Disease Study 2019," *Lancet Gastroenterol. Hepatol.*, vol. 5, pp. 244–267, 2020.
- [4] P. Jepsen and Z. M. Younossi, "The global burden of cirrhosis: A review of disability-adjusted life-years lost and unmet needs," *J. Hepatol.*, vol. 75, pp. S3–S12, 2021.
- [5] H. Devarbhavi et al., "Global burden of liver disease: 2023 update," J. Hepatol., vol. 79, no. 2, pp. 516–537, Aug. 2023.
- [6] C. Ma, A. S. Qian, and N. H. Nguyen, "Trends in the economic burden of chronic liver diseases and cirrhosis in the United States: 1998-2019," *Am. J. Gastroenterol.*, vol. 116, pp. 2059–2068, 2021.
- [7] J. Eda et al., "Nicotine induces fibrogenic changes in human liver via nicotinic acetylcholine receptors expressed on hepatic stellate cells," *Biochem. Biophys. Res. Commun.*, vol. 417, no. 1, pp. 17–22, 2022.
- [8] Y. Arnson, Y. Seinfeld, and H. Amidala, "Effects of tobacco smoke on immunity, inflammation and autoimmunity," *J. Autoimmune*, vol. 34, no. 3, pp. J257–J266, 2015.
- [9] R. E. Fleming and P. Polka, "Iron overload in human disease," N. Engl. J. Med., vol. 366, no. 4, pp. 347–358, 2022
- [10] K. M. Hassan et al., "E-cigarettes and Western diet: Important metabolic risk factors for hepatic diseases," *Hepatology*, vol. 69, no. 6, pp. 2441–2455, 2019.
- [11] M. Xiong et al., "Impacts of cigarette smoking on liver fibrosis and its regression under therapy in male patients with chronic hepatitis B," *Liver Int.*, vol. 39, no. 8, pp. 1428–1436, 2019.
- [12] "Burden of liver disease in Europe: Epidemiology and analysis of risk factors to identify prevention policies," *J. Hepatol.*, vol. 69, pp. 718–735, 2018.

- [13] V. M. Mehra et al., "The association between alcohol, marijuana, illegal drug use and current use of Ecigarette among youth and young adults in Canada: Results from Canadian Tobacco, Alcohol and Drugs Survey 2017," BMC Public Health, vol. 19, no. 1, p. 1208, 2019.
- [14] M. A. Khaleefah, H. J. Al-Badri, and N. A. Mousa, "Hypertension control among adult Iraqis," *J. Fac. Med. Baghdad*, vol. 64, no. 3, pp. 145–152, Oct. 2022.
- [15] K. T. Mills, A. Stefanescu, and J. He, "The global epidemiology of hypertension," *Nat. Rev. Nephrol.*, vol. 16, pp. 222–238, 2020.
- [16] R. K. Nasif and S. R. Ibraheem, "The relationship between liver enzymes level and obesity in a sample of Iraqi women," *Iraqi J. Biotechnol.*, vol. 21, no. 2, pp. 663–667, 2022.
- [17] W. Egner, "The use of laboratory tests in the diagnosis of SLE," J. Clin. Pathol., vol. 64, no. 7, pp. 427–431, 2020.
- [18] K. K. Farmanfarma et al., "Prevalence of type 2 diabetes in Middle–East: Systematic review & meta-analysis," *Prim. Care Diabetes*, vol. 14, pp. 297–304, 2020.
- [19]O. H. Qader and M. Saka, "Prevalence of risk factors of acute coronary syndrome in Erbil Cardiac Center: Comparing ST-elevation with non-ST-elevation acute coronary syndrome," *Adv. Med. J.*, vol. 9, no. 1, pp. 144–152, 2024.
- [20] A. Abera, A. Worede, and A. T. Hirigo, "Dyslipidemia and associated factors among adult cardiac patients: A hospital-based comparative cross-sectional study," *Eur. J. Med. Res.*, vol. 29, p. 237, 2024.
- [21] N. Murad et al., "Modifiable risk factors for cardiovascular disease in Iraqi Kurdistan population: A large epidemiological study," *Healthc. Low-Resour. Settings*, vol. 12, no. 2, 2023.
- [22] W. A. Mula-Abed and S. K. Chilmeran, "Prevalence of dyslipidemia in the Iraqi adult population," *Saudi Med. J.*, vol. 28, no. 12, pp. 1868–1874, Dec. 2007.
- [23] D. A. F. Al-Koofee, J. M. Ismail, and A. A. Algenab, "Lipid profile survey in adults in An-Najaf/Iraq: A cross-sectional study," *J. Phys.: Conf. Ser.*, vol. 1294, no. 5, p. 052018, 2019.
- [24] S. Abdul Wahab ALShaban et al., "Study the effect of dyslipidemia in Iraqi diabetes patients with both gender," *Hist. Med.*, vol. 9, no. 2, 2023.
- [25] H. Baqi, T. Abdullah, D. Ghafor, and S. Karim, "Establishment of lipid profile reference intervals in a sample population of Halabja City, Kurdistan Region of Iraq," *Iraqi J. Sci.*, pp. 2855–2861, 2021.
- [26] T. Alhilfi, R. Lafta, and G. Burnham, "Health services in Iraq," Lancet, vol. 381, pp. 939–948, 2022.
- [27] S. Greco et al., "Dyslipidemia, cholangitis and fatty liver disease: The close underexplored relationship: A narrative review," *J. Clin. Med.*, vol. 13, p. 2714, 2024.
- [28] P. Patel et al., "Association of nonalcoholic fatty liver disease with acute cholangitis: A nationwide propensity-matched analysis from the United States," *Bayl. Univ. Med. Cent. Proc.*, vol. 36, pp. 600–607, 2023.
- [29] A. Mellemkjær et al., "Management of cardiovascular risk in patients with metabolic dysfunction-associated steatotic liver disease," *Eur. J. Intern. Med.*, vol. 122, pp. 28–34, 2024.
- [30] L. J. Souza et al., "Prevalence of dyslipidemia and risk factors in Campos dos Goytacazes, in the Brazilian state of Rio de Janeiro," *Arq. Bras. Cardiol.*, vol. 81, pp. 257–264, 2003.
- [31]J. Arauz, E. Ramos-Tovar, and P. Muriel, "Redox state and methods to evaluate oxidative stress in liver damage: From bench to bedside," *Ann. Hepatol.*, vol. 15, pp. 160–173, 2016.
- [32] R. C. Chakinala et al., "Association of smoking and e-cigarette in chronic liver disease: An NHANES study," *Gastroenterology Res.*, vol. 15, no. 3, pp. 113–119, Jun. 2022.
- [33] A. F. Harlow, A. Stokes, and D. R. Brooks, "Socioeconomic and racial/ethnic differences in e-cigarette uptake among cigarette smokers," *Nicotine Tob. Res.*, vol. 21, no. 10, pp. 1385–1393, 2019.
- [34] F. Y. Enc et al., "The interaction between current smoking and hemoglobin on the risk of advanced fibrosis in NAFLD patients," *Eur. J. Gastroenterol. Hepatol.*, vol. 32, no. 5, pp. 596–599, 2020.