Diyala Journal of Medicine

ORIGINAL RESEARCH
Published: 25 December 2024
DOI: 10.26505/DJM.27028380327

Correlation Between Lipid Profile and Liver Function in Patients With Non-Alcoholic Fatty Liver

A'laa H. Juwad [1], Ammar L. Hussein [2]

[1] Department of medical Lab technology, Al-Qalam university college, Kirkuk, Iraq.

[2] Department of Biochemistry, College of Medicine, University of Tikrit, Tikrit, Iraq.

Correspondence: A'laa H. Juwad
Email: alaaalbayati95@gmail.com

Website: https://djm.uodiyala.edu.iq/index.php/djm

Received: 27 March 2024
Accepted: 22 May 2024
Published: 25 December 2024

Abstract

Background: Non-alcoholic fatty liver disease (NAFLD) is a metabolic liver disease characterized by a broad range of liver pathology, including simple steatosis, steatohepatitis (NASH), fibrosis, cirrhosis, and hepatocarcinoma. NAFLD has emerged as a public health concern in the world within the last 20 years, it is linked to metabolic syndrome (MetS), type 2 diabetes mellitus (T2DM), obesity, and dyslipidemia. Increased visceral adipose tissue in obese people can cause insulin resistance (IR) and hyperinsulinemia, which will speed up the lipolysis of adipose tissue, Lipo-toxicity-related chronic low-grade inflammation is involved in the development of NAFLD.

Objective: Determine the correlation between lipid profile and liver function in patients with NAFLD.

Patients and Methods: A study was conducted at Tikrit Teaching Hospital from 28 November to 28 December 2023. The study involved 90 participants, 60 with NAFLD and 30 healthy subjects. The study used a spectrophotometer (Model NO. HV-2800EX**)** and a colorimetric kit from Spain linear chemicals to determine various parameters, such as Aspartate aminotransferase (AST), Alanine aminotransferase(ALT), Gamma-glutamyl transferase(GGT), High-density lipoprotein(HDL), Low-density lipoprotein(LDL), Very low-density lipoprotein(VLDL), Triglyceride(TG), and cholesterol.

Results: The mean age of patients in the group was 40.93 years, with ages ranging from 20 to 50 years. Serum levels of liver function enzymes (GGT, AST, ALT) and lipid profile (TG, HDL, LDL, VLDL, cholesterol) were measured and compared to the control groups. Patients with NAFLD had significantly higher serum liver function enzymes and increased serum lipid profile (TG, VLDL, LDL, and cholesterol) while showing a significant decrease in HDL concentration when compared to the control group.

Conclusion: The patients showed an increase in liver function enzymes (AST, ALT, GGT) and lipid profile (LDL, VLDL, TG, cholesterol) with reduced HDL as compared to healthy individuals.

Keywords: Nonalcoholic fatty liver, liver function enzymes, lipid profile.

ORIGINAL RESEARCH
Published: 25 December 2024
DOI: 10.26505/DJM.27028380327

Introduction

The metabolic liver disorder known as non-alcoholic fatty liver disease (NAFLD) is characterized by a wide spectrum of liver pathology, from simple steatosis to steatohepatitis (NASH) and fibrosis. Hepatocarcinoma and cirrhosis may result in the end. Since NAFLD is currently the most common cause of chronic liver disease worldwide, it carries a heavy socioeconomic cost. Its prevalence is rising, and it is rising at the same time that obesity and metabolic syndrome are rising. There is a high correlation between obesity and NAFLD, with over 80% of individuals having obesity. In particular, a greater incidence of fibrosis and cirrhosis is thought to be associated with morbid obesity (1). NAFLD has emerged as a significant public health concern in industrialized nations within the last 20 years. It has been connected to metabolic syndrome (MetS), type 2 diabetes mellitus (T2DM), and obesity, however, it can also occur in people who are not obese. Rather than dying from chronic liver disease, people with NAFLD typically have an increased chance of dying from cardiovascular illness. Hepatic iron overload has surfaced as a potential novel component involved in both NAFLD and insulin resistance, and it is well-known that there is a close correlation between the two conditions (2). Evidence of hepatic steatosis, as determined by imaging or histology, is necessary for the diagnosis of NAFLD. Additionally, secondary causes of hepatic fat accumulation, such as the use of steatogenic medications (corticosteroids, amiodarone, methotrexate), hereditary disorders (Wilson's disease, alpha-1 antitrypsin deficiency), or viral infections (hepatitis C infection), must be ruled out. Furthermore, the daily limit for alcohol consumption for males and women is 20 g and 30 g, respectively (3). Most NAFLD patients don't have any symptoms, and the condition may go unnoticed until it develops into cirrhosis. Right upper quadrant pain and fatigue are the most often reported symptoms among people with NAFLD at the time of first diagnosis. Affected individuals may exhibit liver fat based on an incident imaging test or as part of a diagnosis for right upper quadrant pain, or they may have an echogenic liver on ultrasonography. Serum tests relating to the liver usually show an elevation of hepatocellular enzymes, with serum alanine aminotransferase (ALT) being greater than serum aspartate aminotransferase (AST) (4).

Within days after consuming a high-fat diet (HFD), hepatic steatosis occurs (5). A recent study found that eating a diet full of saturated fat was a greater risk than eating a diet filled with free sugars for raising intrahepatic TG levels in overweight people (6). These findings support the notion that lipo-toxicity is a key factor in NAFLD. Studies on marker levels in obese individuals have demonstrated that around 60% of the total triglyceride (TG) content in the liver is made up of free fatty acids (FFAs) from adipose tissue (7). Obese individuals who have more visceral adipose tissue may experience insulin resistance (IR) and hyperinsulinemia, which will accelerate the adipose tissue's lipolysis (8). The development of NAFLD is associated with persistent low-grade inflammation caused by lipid toxicity. The level of inflammation as determined by IL-6 and TNF-α in obese individuals has been demonstrated to be significantly and dose-dependently correlated

ORIGINAL RESEARCH
Published: 25 December 2024
DOI: 10.26505/DJM.27028380327

with the severity of NAFLD (9). The term "metabolic dyslipidemia" has been used recently to describe the type of dyslipidemia that results from the combined effects of obesity and insulin resistance. The modern world's obesity pandemic is closely linked to dyslipidemia, mostly caused by pro-inflammatory adipokines and insulin resistance. However according to new research, obesity-induced dyslipidemia is not a single pathophysiological entity; rather, it has a variety of characteristics that depend on a wide range of individual circumstances. Accordingly, dyslipidemia is either less pronounced or nonexistent in a subset of metabolically healthy obese (MHO) persons (10). Increased levels of triglycerides (TGs), low-density lipoprotein (LDL), total cholesterol, and low-density lipoprotein (HDL) concentrations, either separately or in combination, are considered dyslipidemia (11). Hepatocytes store excess fat as lipid droplets covered with a variety of structural proteins, which may have a role in the pathogenesis of liver disorders. Intrahepatic lipid buildup in non-alcoholic fatty liver disease (NAFLD) is caused by anomalies in lipid metabolism, including reduced triglyceride (TG) export, increased liver free fatty acid (FFA) intake, increased whole-body lipolysis, and increased synthesis of very low-density lipoprotein (VLDL). These changes in lipid metabolism are associated with abnormal synthesis of adipokines (such as leptin, adiponectin, resisting, vastatin, and retinol-binding protein-4) that impact signaling pathways, as well as an elevation of inflammation and oxidative stress (12). It has been demonstrated that liver dysfunction markers like ALT, AST, and γ-glutamyl-transferase (GGT) are useful measures of liver function and are connected to hepatic insulin resistance. ALT is thought to be a particular marker for liver injury and is mostly seen in the liver, While GGT is present on various cell surfaces and is highly active in the liver, pancreas, and kidney. GGT is involved in the uptake of glutathione and is also assumed to have a role in oxidative stress and chronic inflammation, the two main processes that lead to the development of type 2 diabetes. Thus, the biological markers that underlie the association between liver illness and type 2 diabetes may be hepatic enzymes (13). Reducing aggravating variables and changing one's lifestyle are the goals of early NAFLD treatment, which has received substantial support from the literature. Reducing body weight, performing physical activity, and preventing excessive alcohol consumption have been demonstrated time and time again to greatly improve disease signs and in certain instances, even undo early fibrosis. Only NASH patients with severe fibrosis are often eligible for pharmacologic therapy in NAFLD. For NAFLD patients who are unable to reach their weight loss objectives, bariatric surgery is a viable option. Research has demonstrated that this procedure can effectively reverse NAFLD and lower the risk of HCC (14) .

Aim and objectives: estimate serum levels of lipid profile (cholesterol, HDL, VLDL, LDL, TG) and liver enzyme (AST, ALT, GGT) and compare with healthy subjects in nonalcoholic fatty liver disease.

Patients and Methods

patients: Ninety people, ages ranging from 20 to 50, were examined in this study: 60 patients and 30 controls. A liver examination by

ORIGINAL RESEARCH
Published: 25 December 2024
DOI: 10.26505/DJM.27028380327

ultrasound that was acquired for both groups was used to make the diagnosis of NAFLD. The patients were referred to the Tikrit Teaching Hospital's major facilities between November 28, 2023, and December 28, 2023. Using a brief questionnaire, clinical history data, demographics (age, height, weight, and weight), smoking, chronic illnesses, and the treatment plan were gathered. For each case, 5 ml of venous blood was drawn using a sterile disposable syringe, put into gel tubes, and allowed to clot at room temperature for 20 minutes. The serum was removed from each sample and split into two Eppendorf tubes after centrifuging in a centrifuge (Hettich, Germany) for 15 minutes at 3000 rpm. After that, the tubes were kept at -30 C until the biochemical analysis, which included AST, ALT, GGT, TG, VLDL, LDL, HDL, and Cholesterol.

All parameters were measured by spectrophotometric (Double beam Microprocessor ultraviolet-visible spectroscopy Spectrophotometer with software (Model NO. HV-2800EX**)**, and Liner Chemicals Spain kit.

Statistical Analysis

Using SPSS, the data analysis was carried out. P-values < 0.05 are regarded as significant. A P-value > 0.05 was considered non-significant.

Results

90 participants, 30 of whom were aberrantly healthy controls and 60 of whom were\ NAFLD patients, were involved in this study. The study groups were divided into smaller groups according to age, gender, and BMI, and these subgroups are displayed in Table (1). (21.66%) The age range of precipitant was (21-30) years old, (45%) of the patients were within (31-40) years, while (33.34%) of the patients were within the age range (41-50) years.

Table (1): Descriptive of the demographic characteristics of the study population (N=90).

Variable	Groups	Patient N=60	Control N=30
Age. Groups	21-30 Years	8	10
	31-40 Years	15	10
	41-50 Years	37	10
BMI. Groups	Normal weight	5	13
	Overweight	15	12
	Obesity	40	5
Gender	Male	30	15
	Female	30	15

Lipid Profile

Lipids and lipoproteins metabolisms are altered in nonalcoholic fatty liver. In particular, the plasma concentrations of cholesterol, TG, VLDL, and LDL were all elevated in NAFLD but HDL decreased. Results indicated a significant difference (P < 0.01) in all Lipid profiles in the fatty liver group compared to the control as shown in Table (2).

ORIGINAL RESEARCH
Published: 25 December 2024
DOI: 10.26505/DJM.27028380327

Table (2): The mean difference in lipid profile for fatty liver disease to the patients and control groups.

Lipid profile	Patient Mean±SD N=60	Control Mean±SD N=30	P value
TG (mg\dL)	215.99±15.44	189.75±9.16	<0.001[S]
Cholesterol (mg\dL)	198.34±25.09	162.74±6.50	<0.001[S]
HDL(mg\dL)	31.43±7.22	41.58±6.15	<0.001[S]
LDL(mg\dL)	125.47±24.53	83.99±9.58	<0.001[S]
VLDL (mg\dL)	43.19±3.09	37.95±1.83	<0.001[S]
T-test was *: significant at p ≤ 0.05, SD: standard deviation; S: significant; NS= Non-significant.			

Liver Function Enzymes

Comparing patients with NAFLD to healthy control groups, it was seen that the range levels of (GGT, AST, and ALT) U\L increased. The mean and standard deviation values of GGT, AST, and ALT in the patient group were (296.28±75.60), (45.52±7.01), and (45.13±6.63) U\L, respectively. These values were significantly higher than those in the control group (175.93±47.06), (33.01±2.53), and (35.86±1.47) U\L, respectively (p ≤ 0.001). Table (3) displays the distribution of GGT, AST, and ALT serum values in the patients vs the healthy control group.

Table (3): The mean difference in Liver function for fatty liver disease in the patients and control groups.

Liver function	Patient Mean±SD N=60	Control Mean±SD N=30	P value
GGT (U\L)	296.28±75.60	175.93±47.06	<0.001[S]
AST (U\L)	45.52±7.01	33.01±2.53	<0.001[S]
ALT(U\L)	45.13±6.63	35.86±1.47	<0.001[S]
T-test was *: significant at p ≤ 0.05 SD: standard deviation; S: significant; NS= Non-significant.			

The effect of gender on the measured lipid profile according to the patient and control groups. Dyslipidaemia might have a greater influence on fatty liver in males than in females, Therefore, this study also examined the lipid profile panel based on gender groups. Results indicated that cholesterol, TG, and LDL were increased significantly in both male and female adults compared to healthy

ORIGINAL RESEARCH
Published: 25 December 2024
DOI: 10.26505/DJM.27028380327

control, p-value <0.001. as presented in Figure (1)

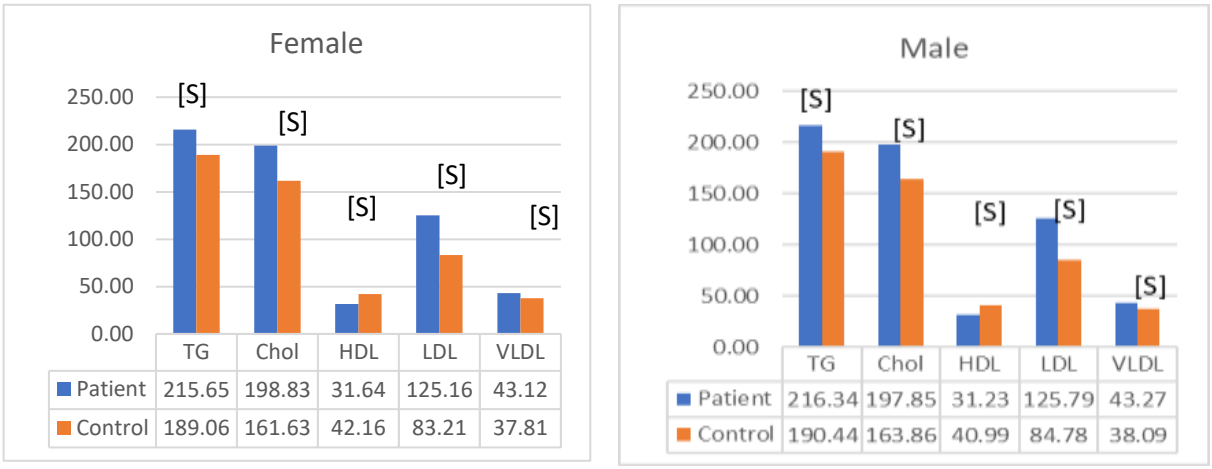

Figure (1): The effect of gender on the lipid profile parameters according to the patients and control groups.

The effect of gender on the measured liver function according to the patient and control groups.

Figure (2) illustrates the mean level of the biochemical in the patients and control groups according to gender. Results showed that male and female patients showed a highly statistically significant increase in the mean levels of GGT, AST, and ALT compared to the control, with p-values were <0.05

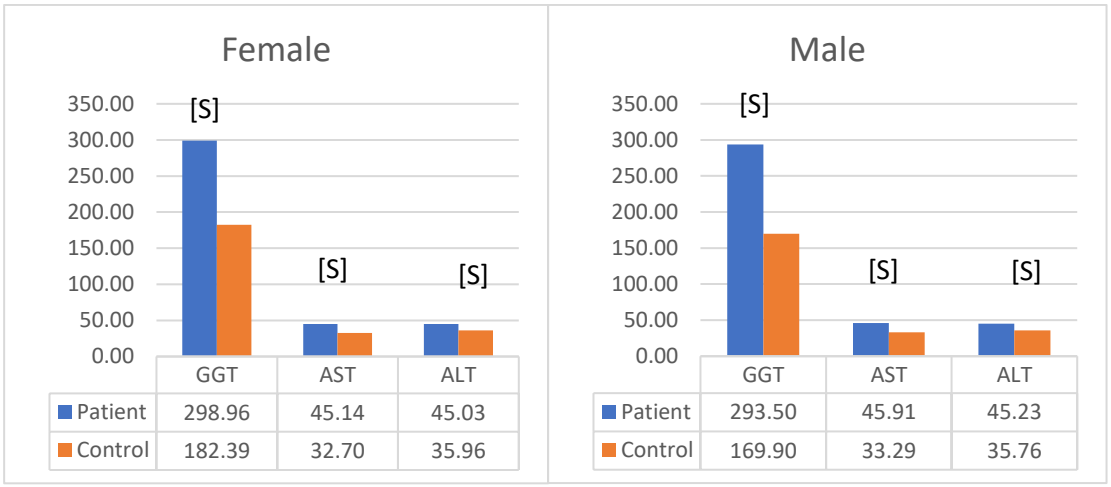

Figure (2): The effect of gender on the liver function parameters according to the patients and control groups.

Results of the receiver operating curve (ROC) and area under curve (AUC) analysis for the GGT, AST, and ALT as diagnostic parameters were done. GGT, AST, and ALT showed good performance for predicting fatty liver disease compared to the control group GGT levels:

ORIGINAL RESEARCH
Published: 25 December 2024
DOI: 10.26505/DJM.27028380327

(sensitivity = 98.3 %, specificity 96.6%) at a level = 92.1, For AST levels: (sensitivity = 98.3 %, specificity 96.6%) at a level = 99.7, For ALT levels: (sensitivity = 98.3 %, specificity 96.6%) at a level = 99.5, the p-values of the AUC were <0.05 and highly statistically significant. The p-values of the AUC were <0.05 and statistically significant. Youden's J statistics of the. parameters in Figure 3 confirm these results for GGT, AST, and ALT.

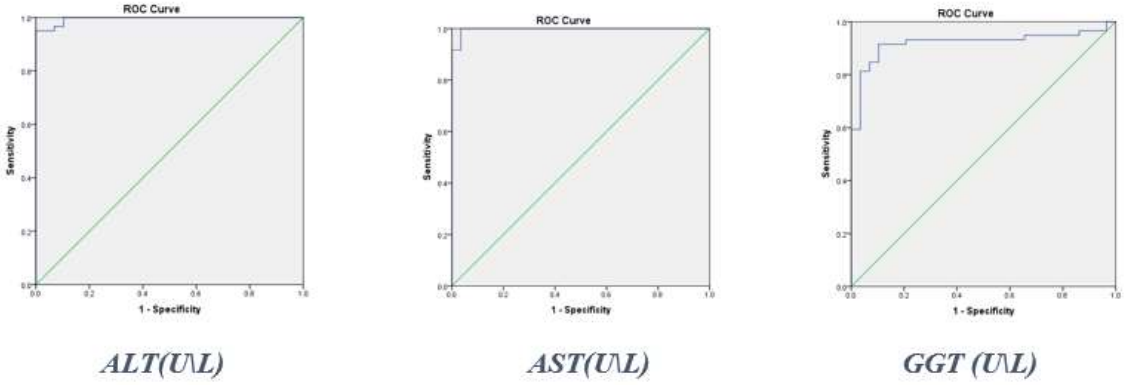

Figure 3: ROC curves for GGT, AST, and ALT in patients.

Discussion

Worldwide, the most prevalent cause of chronic liver disease in developed nations is NAFLD. It's thought that NAFLD represents the metabolic syndrome's hepatic expression. It can progress from simple fatty liver to steatohepatitis (15).

This present study shows a significant increase in serum concentration of lipid profile (LDL, VLDL, TG, cholesterol) and decreased serum concentration of HDL as compared with healthy individuals. This study supports the findings of Han and Ji Men's population study, which found that patients with NAFLD had lower concentrations of HDL-C and higher levels of TG, TC, and LDL-C in their serum compared to normal individuals. The study also found that the associations between fatty liver and dyslipidemia varied depending on the degree of hepatic steatosis. The degree of hepatic fat accumulation influenced the connection between dyslipidemia and fatty liver, with those who had fatty livers having more probability than those who did not. Fatty liver is a hallmark of the metabolic syndrome's hepatic presentation. In addition to other metabolic risk factors such as diabetes, obesity, and hypertension, dyslipidemia is commonly observed in patients with fatty liver disease (16). As a result of the fact that a larger proportion of our patients are obese, Khan and Reenam reported that obese NAFLD patients had more lipolysis in their adipose tissue, which may account for 60–70% of the fat that accumulates in the liver. Together with the excessive lipolysis of adipose tissue, adipocytes produce hormones abnormally (e.g., decreased adiponectin production),

ORIGINAL RESEARCH
Published: 25 December 2024
DOI: 10.26505/DJM.27028380327

which exacerbates adipose tissue inflammation (e.g., pro-inflammatory cytokines are released). The enhancement of insulin resistance (IR), which results in ectopic fat deposition, is facilitated by all of these variables (17).

From another point of view, Méndez-Sánchez and Nahum reported in fundamental studies that cholesterol crystals, which are lipid droplets possessing a significant birefringence under polarized light, are exclusively found in NASH models and not in cases of simple steatosis. These circumstances cause activated Kupffer cells (KCs) to gather around cholesterol crystals to form "crown-like structures," which are closely linked to the growth of foam cells and, consequently, atherosclerosis (18). Reduced plasma HDL levels are typically associated with insulin-resistant states. This relationship can be explained by the following mechanism: In the presence of normal cholesteryl ester transfer protein activity and increased plasma VLDL concentrations, VLDL TG can be substituted for HDL cholesterol. In this mechanism, an HDL cholesteryl ester molecule is exchanged for a TG molecule by a VLDL particle with an HDL particle. An atherogenic, cholesterol-rich VLDL remnant particle and an HDL particle with a high TG content but a low cholesterol content are produced by this process (19). The TG-rich HDL particle will subsequently undergo additional changes, such as the hydrolysis of its TG, which will cause the apoA-1 protein to disassociate. As a result, because free apoA-1 leaves the plasma more quickly than apoA-1 bound to HDL particles, there will be a decrease in the amount of circulating apoA-1, HDL cholesterol, and HDL particle count (20).

The present study shows elevated levels of liver function enzymes (AST, ALT, GGT) as compared to control groups this agrees with Xie, Ruijie, and Mingjiang Liu (21) and Fontes-Cal and Tereza who reported AST, ALT, and GGT values in liver enzyme tests were considerably higher in NAFL and NASH patients than in the control group. A 64,5% prevalence of individuals with altered liver enzyme profiles were classified as NAFL or NASH, with the NASH category having a higher prevalence (80%) (22).

Conclusions

Compared to healthy individuals, the patient group in this study showed abnormally high liver enzyme and lipid profiles and significantly lower HDL. Moreover, AST, ALT, and GGT were highly sensitive and specific in predicting NAFLD.

Recommendations

It is recommended to increase the number of participants to obtain accurate results, and assess insulin resistance and evaluate the relationship between it and the incidence of NAFLD. In addition, it is important to determine the role of the drugs on the advancement of NFLAD.

Source of Funding: The current study was funded by our charges with no any other funding sources elsewhere.

Ethical Clearance: Official approval has been obtained to use data and data were analyzed without the names to protect privacy. This study was conducted according to the approval of College of Medicine/ University of Diyala and in accordance with the ethical guidelines of the Declaration of ethical committee of the College (Document no. 2024AHJ838).

Conflict of Interest: Non

ORIGINAL RESEARCH
Published: 25 December 2024
DOI: 10.26505/DJM.27028380327

References

1. Borges-Canha and Marta. Thyroid function and the risk of non-alcoholic fatty liver disease in morbid obesity. Frontiers in endocrinology, 2020, 11: 572128. https://doi.org/10.3389/fendo.2020.572128

2. Martin-Rodriguez. Insulin resistance and NAFLD: relationship with intrahepatic iron and serum TNF-α using 1H MR spectroscopy and MRI. Diabetes & Metabolism, 2019, 45(5): 473-479. https://doi.org/10.1016/j.diabet.2019.01.005

3. Kasper and Philipp. NAFLD and cardiovascular diseases: a clinical review. Clinical research in cardiology, 2021, 110: 921-937. https://doi.org/10.1007/s00392-020-01709-7

4. Loomba, Rohit, Scott L. Friedman, and Gerald I. Shulman. "Mechanisms and disease consequences of nonalcoholic fatty liver disease." Cell 2021, 184(10): 2537-2564. https://doi.org/10.1016/j.cell.2021.04.015

5. Gomez-Zorita and Saioa. Relationship between changes in microbiota and liver steatosis induced by high-fat feeding—A review of rodent models. Nutrients, 2019, 11(9): 2156. https://doi.org/10.3390/nu11092156

6. Parry and Siôn. Intrahepatic fat and postprandial glycemia increase after consumption of a diet enriched in saturated fat compared with free sugars. Diabetes care, 2020, 43(5): 1134-1141. https://doi.org/10.2337/dc19-2331

7. Cordeiro and Adryana. Does adipose tissue inflammation drive the development of non-alcoholic fatty liver disease in obesity? Clinics and research in hepatology and gastroenterology, 2020, 44(4): 394-402. https://doi.org/10.1016/j.clinre.2019.10.001

8. Kojta, Iwona, Marta Chacińska, and Agnieszka Błachnio-Zabielska. Obesity, bioactive lipids, and adipose tissue inflammation in insulin resistance. Nutrients 2020,12(5):1305. https://doi.org/10.3390/nu12051305

9. Zatterale and Federica. Chronic adipose tissue inflammation linking obesity to insulin resistance and type 2 diabetes. Frontiers in physiology, 2020, 10: 1607. https://doi.org/10.3389/fphys.2019.01607

10. Mahmood, Zana Mustafa, and Karwan Hawez Sulaiman. "The association between obesity and irritable bowel syndrome among a sample of patients attending the primary health care centers in Erbil city." *Diyala* Journal of Medicine ,2022,22(2): 86-95. Doi: https://doi:10.26505/DJM.22026390223

11. Jebr, Mehdi Shemkhi, Baraa Najm Abed, and Huda Adnan Hussein. "Dyslipidemia in insulin dependent diabetic children." *Diyala* Journal of Medicine ,2024,26(2): 125-140. Doi: 10.26505/DJM.26028530425

12. Katsiki, Niki, Dimitri P. Mikhailidis, and Christos S. Mantzoros. "Non-alcoholic fatty liver disease and dyslipidemia: an update." Metabolism, 2016, 65(8): 1109-1123. https://doi.org/10.1016/j.metabol.2016.05.003

13. Islam and Shiful. "Prevalence of elevated liver enzymes and its association with type 2 diabetes: A cross-sectional study in Bangladeshi adults." Endocrinology, diabetes & metabolism 2020 3(2): e00116. https://doi.org/10.1002/edm2.116

14. Orabi, Danny, Nathan A. Berger, and J. Mark Brown.Abnormal metabolism in the progression of nonalcoholic fatty liver disease to hepatocellular carcinoma: mechanistic

ORIGINAL RESEARCH
Published: 25 December 2024
DOI: 10.26505/DJM.27028380327

insights to chemoprevention. Cancers 2021 13(14): 3473.
https://doi.org/10.3390/cancers13143473
15. Gottlieb, Aline, and Ali Canbay. Why bile acids are so important in non-alcoholic fatty liver disease (NAFLD) progression. Cells 2019 8(11): 1358.
https://doi.org/10.3390/cells8111358
16. Han and Ji Min. Differing associations between fatty liver and dyslipidemia according to the degree of hepatic steatosis in Korea. Journal of Lipid and Atherosclerosis, 2019, 8(2): 258-266.
doi:10.12997/jla.2019.8.2.258
17. Khan and Reenam. Modulation of insulin resistance in nonalcoholic fatty liver disease. Hepatology, 2019, 70(2): 711-724.
DOI 10.1002/hep.30429
18. Méndez-Sánchez and Nahum. Dyslipidemia as a risk factor for liver fibrosis progression in a multicentric population with non-alcoholic steatohepatitis. F1000Research 2020, 9.
doi: 10.12688/f1000research.21918.1
19. Mavromati, Maria, and François R. Jornayvaz. "Hypothyroidism-associated dyslipidemia: potential molecular mechanisms leading to NAFLD." International Journal of Molecular Sciences 2021,22(23): 12797.
https://doi.org/10.3390/ijms222312797.
20. Ginsberg and Henry N. "Insulin resistance and cardiovascular disease." The Journal of Clinical Investigation 2000,106(4): 453-458.
https://doi.org/10.1172/JCI10762
21. Xie, Ruijie, and Mingjiang Liu. "Relationship between non-alcoholic fatty liver disease and degree of hepatic steatosis and bone mineral density." Frontiers in Endocrinology 2022,13: 857110.
https://doi.org/10.3389/fendo.2022.857110
22. Fontes-Cal and Tereza. "Crosstalk between plasma cytokines, inflammation, and liver damage as a new strategy to monitoring NAFLD progression." Frontiers in Immunology
2021,12:708959. https://doi.org/10.3389/fimmu.2021.708959

Diyala Journal of Medicine

ORIGINAL RESEARCH
Published: 25 December 2024
DOI: 10.26505/DJM.27028380327

العلاقة بين مستوى الدهون وانزيمات وظائف الكبد لدى مرضى الكبد الدهني الغير كحولي

الاء حيدر جواد [١], عمار لطيف حسين [٢]

الملخص

خلفية الدراسة: مرض الكبد الدهني غير الكحولي هو مرض الكبد الأيضي الذي يتميز بمجموعة واسعة من أمراض الكبد، بما في ذلك التليف والتهاب الكبد الدهني والتنكس الدهني البسيط ويمكن أن يؤدي إلى تليف الكبد وسرطان الكبد. لقد برز مرض الكبد الدهني غير الكحولي باعتباره مصدر قلق للصحة العامة في الدول خلال العشرين عامًا الماضية، وقد تم ربطه بمتلازمة التمثيل الغذائي، داء السكري من النوع ٢، والسمنة واضطراب شحميات الدم. زيادة الأنسجة الدهنية الحشوية لدى الأشخاص الذين يعانون من السمنة المفرطة يمكن أن تسبب مقاومة الأنسولين وفرط أنسولين الدم، مما يؤدي إلى تسريع تحلل الدهون في الأنسجة الدهنية. ويشارك الالتهاب المزمن منخفض الدرجة المرتبط بالسمية الدهنية في تطور المرض الكبد الدهني الغير الكحولي.

اهداف الدراسة: تحديد العلاقة بين مستوى الدهون وانزيمات وضائف الكبد لدى المرضى الذين يعانون من مرض الكبد الدهني الغير الكحولي.

المرضى والطرائق: أجريت دراسة في مستشفى تكريت التعليمي في الفترة ما بين ٢٨ تشرين الثاني (نوفمبر) ٢٠٢٣ و٢٨ كانون الأول (ديسمبر) ٢٠٢٣. وشملت الدراسة ٩٠ مشاركاً، تم تشخيص ٦٠ منهم بمرض الكبد الهني لبغير الكحولي وكان ٣٠ منهم بصحة جيدة. استخدمت الدراسة مقياس الطيف الضوئي ومجموعة قياس الألوان من المواد الكيميائية الخطية الإسبانية لتحديد المعلمات المختلفة، مثل AST وALT وGGT وHDL وLDL وVLDL وTG والكوليسترول.

النتائج: كان متوسط عمر المرضى في المجموعة ٤٠,٩٣ سنة، وتتراوح أعمارهم بين ٢٠ إلى ٥٠ سنة. تم قياس ومقارنة مستويات إنزيمات وظائف الكبد (GGT، AST، ALT) ومستويات الدهون (TG. HDL، LDL. VLDL، الكوليسترول) مع أفراد السيطرة. كان لدى المرضى الذين يعانون من الكبد الدهني الغير كحولي ارتفاع ملحوظ في إنزيمات وظائف الكبد في الدم وزيادة في مستوى الدهون في الدم (TG، VLDL، LDL، والكوليسترول) بينما أظهروا انخفاضًا ملحوظًا في تركيز HDL لدى الأفراد الأصحاء. عند مقارنتها بالمجموعة الضابطة.

الاستنتاجات: أظهر المرضى زيادة غير طبيعية في مستويات إنزيمات الكبد ومستوى الدهون (LDL، VLDL، TG، الكوليسترول) مع انخفاض HDL مقارنة بالأصحاء.

الكلمات المفتاحية: الكبد الدهني الغير الكحولي، انزيمات وضائف الكبد، الملف الدهني.

البريد الالكتروني: alaaalbayati95@gmail.com

تاريخ استلام البحث: ٢٧ اذار ٢٠٢٤
تاريخ قبول البحث: ٢٢ أيار ٢٠٢٤

---

[١] قسم تقنيات المختبرات الطبية/ كلية القلم الجامعة/ كركوك/ العراق.
[٢] جامعة تكريت/ كلية الطب/ قسم الكيمياء الحياتية السريرية/ تكريت/ العراق.