The Medical Journal of Tikrit University (2023) 29 (1): 131-11
DOI: http://dx.doi.org/10.25130/mjotu.31.2.3

IRAQI Academic Scientific Journals

ISSN: 1813-1638

The Medical Journal of Tikrit University

Available online at:www.mjotu.com

MJTU

The Medical Journal of Tikrit University

The correlation between Apo B lipoprotein and lipid profile in Patients with Non-Alcoholic Fatty Liver Disease

Zryan S.Omer [1]. Ammar L. Hussein .[2]

[1] Azadi Teaching Hospital, Kirkuk Health Director, Kirkuk,Iraq

[2] Department of microbiology, College of Medicine, University of Tikrit, Tikrit, Iraq

Keywords: *Nonalcoholic fatty liver disease, Apo B lipoprotein, lipid profile*

ARTICLE INFO

Article history:

Received	01 Jul 2025
Accepted	01 Sep 2025
Available online	31 Dec 2022

Citation:

ABSTRACT

Background: Non-alcoholic fatty liver disease (NAFLD) is a prevalent etiology of chronic liver disease globally. NAFLD is a spectrum of disease defined by hepatic steatosis in the absence of identifiable secondary causes of hepatic fat buildup, such as excessive alcohol intake. NAFLD encompasses a spectrum from the relatively benign non-alcoholic fatty liver (NAFL) to the more severe non-alcoholic steatohepatitis (NASH). NAFLD may advance to fibrosis and cirrhosis. The liver plays an important role in lipid metabolism, including importing Free FAs (FFAs) and manufacturing, storing, and exporting lipids, and the dysregulation of these processes in the liver can lead to the development of NAFLD.

Objective: determine the correlation between Apo B and lipid profile in Patients with NAFLD

Patients and Methods: A study was conducted at Azadi Teaching Hospital from 1 June of 2024 to 15 June 2025. The study involved 90 participants, 60 with NAFLD and 30 healthy subjects. The study used a spectrophotometer to determine various parameters, High-density lipoprotein(HDL), Triglyceride(TG), and cholesterol. ELISA washer and reader also used for measurement of Apo B.

Result: Age of patients ranging from 20 to 65 years. Serum level of Apo B lipoprotein and lipid profile were measured and compared to control group, Apo B was significantly higher in patients with NAFLD.However lipid (TRI, CHOL, VLDL, LDL) also were measured and they were significantly higher in patient group than control group, while HDL level was lower in patients than controls.

Conclusion: Positive correlation between Apo B and HDL while negative correlation with other lipid (tri,chol,VLDL,LDL)

INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) has emerged as one of the most prevalent form of chronic liver diseases due to an imbalance between lipid accumulation and removal (1). Nonalcoholic fatty liver disease (NAFLD) is a hepatic condition commonly observed in adults who do not consume alcohol. While other factors have been identified as risk contributors, obesity unequivocally stands as the primary risk factor in the onset of NAFLD. The prevalence of NAFLD in the general population reaches 30%; nonetheless, it is closely linked to obesity, insulin resistance, hypertension, and dyslipidemia (2). A liver biopsy remains the clinical standard for a definitive diagnosis of NAFL or NASH, despite imaging and clinical characteristics potentially indicating significant concerns regarding an individual (3). Over fifty percent of persons with NAFLD are asymptomatic, and the condition may have insidiously advanced to cirrhosis (4). The most apparent symptoms observed in the initial stages of NAFLD in individuals include right upper quadrant pain and fatigue (5). Metabolic syndrome and its defining characteristics, namely obesity, type 2 diabetes, hypertension, and dyslipidemia, are established risk abnormalities, such as thyroid dysfunction, adrenal insufficiency, growth hormone deficiency, and polycystic ovary syndrome, contribute to the development of non-alcoholic fatty liver disease NAFLD (6).

Hepatic steatosis occurs when the equilibrium between hepatic triglyceride synthesis and export is disrupted, leading to synthesis surpassing export capacity. The synthesis of hepatic triglycerides may be augmented by the provision of substrates, such as free fatty acids or 3-phosphoglycerate derived from glycolysis due to dietary carbohydrate surplus, to the liver. (7). Lipid buildup in the liver is the primary characteristic of NAFLD. The dyslipidemic profile associated with fatty liver, marked by elevated large very low-density lipoprotein (VLDL), reduced small dense low-density lipoprotein (LDL), and diminished large high-density lipoprotein (HDL), corresponds with intrahepatic lipid accumulation (8). Apolipoproteins, the protein components of lipoproteins, are attached to the lipoprotein surface and primarily determine the characteristics, transport, and metabolism of lipoproteins. ApoB is a crucial constituent of VLDLs, along with its metabolites IDLs and LDLs, as well as chylomicrons and their remains. The apoB particle functions as a framework and is essential for preserving the structural integrity of the lipoprotein (9).

ApoB is encoded by the APOB gene and exists in two forms: full-length apoB100, comprising 4536 amino acids, and apoB48, a truncated variant consisting of the N-terminal 2152 amino acids (10). Despite being encoded by the same gene; they fulfill disparate functions in physiology. ApoB48 is predominantly produced and expressed in the colon of humans and is found in chylomicrons and their leftovers. ApoB100 is predominantly produced and expressed in the liver, serving as a fundamental component of VLDL, IDL, and LDL (11). Consequently, among the two variants, apoB100 is more clinically significant in assessing the concentration of circulating atherogenic lipoproteins (12). ApoB100 is distinctive among plasma apolipoproteins due to its

substantial size, moderate hydrophobicity, and lack of capacity to transfer across lipoproteins (13). The bulk of apoB-containing lipoproteins in circulation are mostly taken up and eliminated by the liver in humans. Heparin sulfate proteoglycans, the LDL receptor, and scavenger receptor class B type I are the three main receptors that facilitate uptake. More than two-thirds of normal LDL is cleared by the LDL receptor, which has a half-life of approximately 25 hours (14). Familial hypercholesterolemia, caused by mutations in the LDL receptor or apoB, causes abnormally high amounts of LDL in the blood and speeds up the process of atherosclerosis (15). The aim of this study was to know the correlation between apoB level and lipid profile in patients with nonalcoholic fatty liver disease.

PATIENTS AND METHODS

The study included 60 NAFLD patients, ranging in age from 20 to 65. These individuals were diagnosed by ultrasound after being brought to the gastrointestinal unit at Azadi teaching hospital. Patients' personal information, including age, weight, height, smoking status, chronic diseases, and treatment plans, were collected through interviews using investigator-designed questionnaires. Thirty healthy individuals will also serve as controls in the study. Gel tubes were used to transmit blood samples (4-5 ml) taken from both healthy individuals and sick. Centrifugation was used for 15 minutes at 3000 rpm to extract the serum. The serum was kept at a temperature of -20 °C until the assays were conducted. The tests which were performed include Apo B using ELISA technique and lipid (Triglyceride, cholesterol, HDL) using spectrophotometer technique.

Statistical Analysis

We used the SPSS program, which is a statistical package for the social sciences, to determine the level of statistical significance. Unless the P value was less than 0.05, we did not consider the difference between the two groups to be statistically significant. We then expressed the results as (mean ± standard deviation).

RESULTS

The ages of the study participants, as indicated in Table 1, varied from participants were divided into three categories, as illustrated in figure 1, with a greater percentage of female (58.5%) compared to males (41.6%), as shown in table-1. Based on this distribution, it is plausible to infer that most NAFLD cases arise in individuals aged 36 to 50, with a much higher frequency in women compared to men with in this demographic. Both the healthy control group and the individuals with NAFLD had their Apo B levels measured in the blood. Statistical analysis was used to examine the estimation result. The results showed that the concentration of Apo B in patients increased significantly (with a probability of 1.874 ± 0.221) and decreased (with a probability of (1.287 ± 0.206) compared to the control group. Shown in Table 2. Excessive lipid buildup in the liver is the pathophysiological characteristic of NAFLD.The plasma concentrations of cholesterol, triglycerides, VLDL, and LDL were elevated significantly in NAFLD with the probability of (212.4 ± 20.9,226.2 ± 28.0, 45.27 ± 5.63, 130.4 ± 21.6) than in control (180.5 ± 12.5, 195.5 ± 17.3, 39.08

± 3.45, 87.0 ± 15.6) but HDL levels were decreased in the patient group with probability (36.82±6.76 vs. 55.80±8.51), as shown in figure 1. This study found that there was weak negative correlation between Apo B and triglyceride in patients with NAFLD(r=-0.156), figure 2. Weak negative correlation also indicated between Apo B and cholesterol in patients with NAFLD wiche mean as one variable increase other one decrease ,figure 3. Apo B correlated with both LDL and VLDL a weak negative correlation, figure 4 and figure 5. The study found that there was weak positive correlation between Apo B and cholesterol in NAFLD patient

DISCUSSSION

Nonalcoholic fatty liver disease (NAFLD) is the predominant cause of chronic liver disease worldwide. The spectrum encompasses simple hepatic steatosis to nonalcoholic steatohepatitis (NASH), potentially resulting in liver fibrosis and cirrhosis. (16,17).This study set out to compare Apo B and lipid profile levels in NAFLD patients to those in healthy people and to see whether there was any correlation between the two. The results of the study corroborate those of Nass KJ et al., who discovered that ApoB dyslipoproteinemias may enhance the risk of NAFLD, since patients with NAFLD had a higher Apo B lipoprotein level than the control group (18). The liver plays a central role in lipid metabolism, storing and exporting lipids and lipoproteins. Lipid accumulation in the liver is the major hallmark of NAFLD. Abnormalities in lipid and lipoprotein metabolism accompanied by chronic inflammation are considered to be the central pathway for the development of NAFLD (19). This study also evaluated lipid profiles and found that patients with NAFLD had significantly higher levels of TRI, CHOL, VLDL, and LDL compared to the control group, but their levels of HDL were lower. These findings are in agreement with the study by Almobarak AO et al. (20).Regarding the correlation between Apo B and lipid profile, a weak negative connection was detected between Apo B and triglycerides, cholesterol, LDL, and VLDL, whereas a weak positive association was noted between Apo B and HDL in individuals with NAFLD. Maria Dorobanțu et al were found in their study Lipides (total cholesterol, triglycerides, and LDL-cholesterol) and ApoB levels were found to be positively correlated, while HDL-cholesterol levels were found to be negatively correlated(21). Researchers have found that incident NAFLD is more likely in people with higher levels of blood oxidized low density lipoprotein (LDL) cholesterol and apolipoprotein B (ApoB). There is weak causative evidence for ApoB, LDL cholesterol, and triglycerides, however one Mendelian randomization study indicated that genetically determined lower HDL cholesterol and higher triglycerides were causal risk factors for NAFLD(22).

CONCLUSION

The patients with NAFLD in current study present with increased level of Apo B when compared to control group, they also show elevated levels of lipid (TRI, CHOL, VLDL and LDL) and decrease HDL level. Positive correlation was reported between Apo B and HDL and negative correlation with other lipid.

RECOMMENDATION

It is recommended that more study performing about the correlation between Apo B and lipid profile in NAFLD. Increase number of participants in the study to obtain more accurate result.

Source of funding: No source of funding

REFERENCES

1. Chen, Hanqing. Nutrient mTORC1 signaling contributes to hepatic lipid metabolism in the pathogenesis of non-alcoholic fatty liver disease. Liver Research, 2020, 4.1: 15-22.
2. Sanyal, M.L. Van Natta, J. Clark, et al.Prospective study of outcomes in adults with non-alcoholic fatty liver disease N Engl J Med, 2021, 385.17: 1559-1569.
3. Gunn NT, Shiffman ML. The Use of Liver Biopsy in Nonalcoholic Fatty Liver Disease. Clinics in Liver Disease. 2018 Feb;22(1):109–19.
4. Spengler EK, Loomba R. Recommendations for Diagnosis, Referral for Liver Biopsy, and Treatment of Nonalcoholic Fatty Liver Disease and Nonalcoholic Steatohepatitis. Mayo Clinic Proceedings. 2015 Sep;90(9):1233–46.
5. Chalasani N, Younossi Z, Lavine JE, Charlton M, Cusi K, Rinella M, Harrison SA, Brunt EM, Sanyal AJ. The diagnosis and management of nonalcoholic fatty liver disease: Practice guidance from the American Association for the Study of Liver Diseases. Hepatology. 2018 Jan;67(1):328-357.
6. Liu Y, Wang W, Yu X, Qi X. Thyroid Function and Risk of Non-Alcoholic Fatty Liver Disease in Euthyroid Subjects. Ann Hepatol. 2018 Aug 24;17(5):779-788.
7. Rishan S. Correlation between non-alcoholic fatty liver disease (NAFLD) and dyslipidemia in type 2 diabetes. Diabetes & Metabolic Syndrome: Clinical Research & Reviews. 2016 Apr;10(2): S77–81.
8. Klementina FT, Damjana R. Non-alcoholic fatty liver disease: focus on lipoprotein and lipid deregulation. Hindawi J Lipids 2011; 2011:1-14.
9. Behbodikhah J, Ahmed S, Elyasi A, Kasselman LJ, De Leon J, Glass AD, Reiss AB. Apolipoprotein B and Cardiovascular Disease: Biomarker and Potential Therapeutic Target. Metabolites. 2021 Oct 8;11(10):690. doi: 10.3390/metabo11100690.
10. Fisher, E.A.; Ginsberg, H.N. Complexity in the secretory pathway: The assembly and secretion of apolipoprotein B containing lipoproteins. J. Biol. Chem. 2002, 277, 17377–17380.
11. Nakajima, K.; Nakano, T.; Tokita, Y.; Nagamine, T.; Inazu, A.; Kobayashi, J. Postprandial lipoprotein metabolism; VLDL vs chylomicrons. Clin. Chim. Acta 2011, 412, 1306–1318.
12. Doonan, L.M.; Fisher, E.A.; Brodsky, J.L. Can modulators of apolipoproteinB biogenesis serve as an alternate target for cholesterollowering drugs? Biochim. Biophys. Acta Mol. Cell. Biol. Lipids 2018, 1863, 762–771.

13. Whitfield AJ, Barrett PHR, van Bockxmeer FM, Burnett JR. Lipid Disorders and Mutations in the APOB Gene. Clinical Chemistry. 2004 Oct 1;50(10):1725–32.
14. Morita, S.Y. Metabolism and Modification of Apolipoprotein B-Containing Lipoproteins Involved in Dyslipidemia and Atherosclerosis.Biol. Pharm. Bull. 2016, 39, 1–24.
15. Bruikman, C.S.; Hovingh, G.K.; Kastelein, J.J. Molecular basis of familial hypercholesterolemia. Curr. Opin. Cardiol. 2017, 32, 262–266.
16. Cheemerla S, Balakrishnan M. Global epidemiology of chronic liver disease. Clin Liver Dis (Hoboken) 2021; 17:365-370.
17. Chalasani N, Younossi Z, Lavine JE, Charlton M, Cusi K, Rinella M, et al. The diagnosis and management of nonalcoholic fatty liver disease: practice guidance from the American Association for the Study of Liver Diseases. Hepatology 2018; 67:328-357.
18. Nass KJ, van den Berg EH, Faber KN, Schreuder TCMA, Blokzijl H, Dullaart RPF. High prevalence of apolipoprotein B dyslipoproteinemias in non-alcoholic fatty liver disease: The lifelines cohort study. Metabolism. 2017 Jul; 72:37-46. doi: 10.1016/j.metabol.2017.04.004.
19. Dick C, Gerald F, Seng KG, Annette T, Esther MM, Hugh RB. Non-alcoholic fatty liver disease as the transducer of hepatic oversecretion of very low density lipoprotein apolipoprotein B-100 in obesity. Arterioscler Thromb Vasc Biol 2010; 30:1043-50.
20. Almobarak AO, Barakat S, Suliman EA, Elmadhoun WM, Mohamed NA, Abobaker IO, Noor SK, Bushara SO, Ahmed MH. Prevalence of and predictive factors for nonalcoholic fatty liver disease in Sudanese individuals with type 2 diabetes: Is metabolic syndrome the culprit? Arab J Gastroenterol. 2015 Jun;16(2):54-8. doi: 10.1016/j.ajg.2015.06.001.
21. Dorobanțu M, Halațiu VB, Gheorghe-Fronea O, Bala CG, Moldovan H, Irinel-Parepa R, Rodean IP, Benedek I, Benedek T. The Association between Apolipoprotein B, Cardiovascular Risk Factors and Subclinical Atherosclerosis-Findings from the SEPHAR National Registry on Hypertension in Romania. Int J Mol Sci. 2023 Feb 1;24(3):2813. doi: 10.3390/ijms24032813.
22. Wang, J., Zhu, W., Huang, S., Xu, L., Miao, M., Wu, C., et al. (2017) Serum apoB levels independently predict the development of non-alcoholic fatty liver disease: a 7-year prospective study. Liver Int. 37, 1202–1208.

TABLES

Table 1: Descriptive of the demographic features of the research.

Variables	Groups	Patients N=60	Control N=30
Age groups	A1 group (20-35)Years	17 (28.34%)	22 (73.33%)
	A2 group (36-50)Years	23 (38.33%)	6 (20%)
	A3 group(50-65)Years	20 (33.33%)	2 (6.67%)
BMI	B1 group(18-24)	Non	25(83.7%)
	B2 group(25-28)	8 (13.3%)	5(16.75)
	B3 group (29 above)	52 (86.7%	Non
Gender	Male	25 (41.6%)	12 (40%)
	Female	35 (58.4%)	60%)

Table 2: Apo B lipoprotein level in patients with NAFLD compared to control group.

Test	Patients mean±SD N=60	control mean±SD N=60	P value
Apo B	1.874 ± 0.221	1.287 ± 0.206	0.0006
T-test was *: significant at p ≤ 0.05, SD: standard deviation; S: significant; NS= Non-significant			

FIGURES

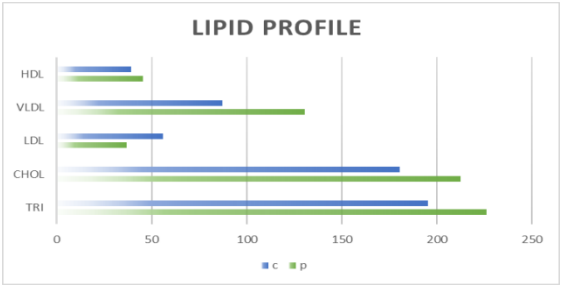

Figure 1: lipid profile levele in patients with NAFLD versus control group

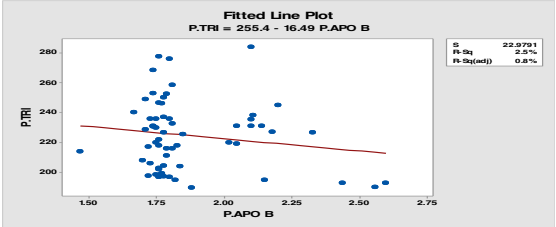

Figure 2: correlation between apo B and triglyceride.

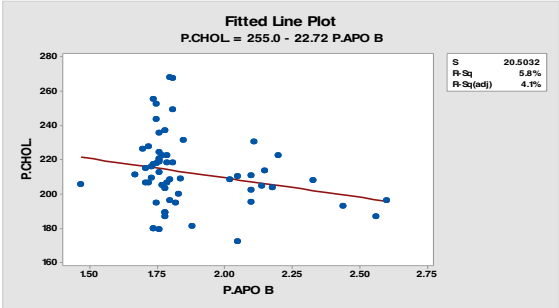

Figure 3: correlation between Apo B and cholesterol in NAFLD patients

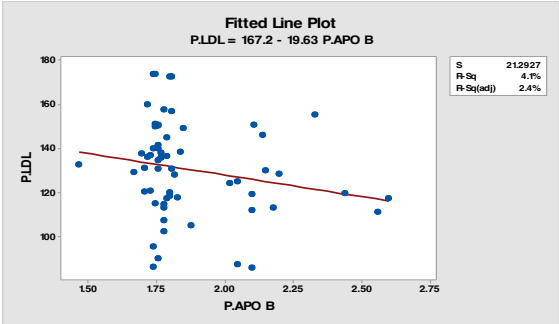

Figure 5: correlation between Apo B and VLDL

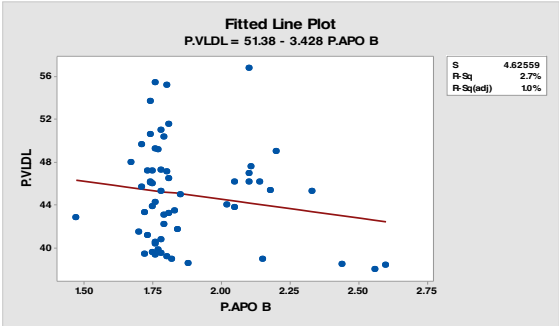

Figure 5: correlation between Apo B and VLDL

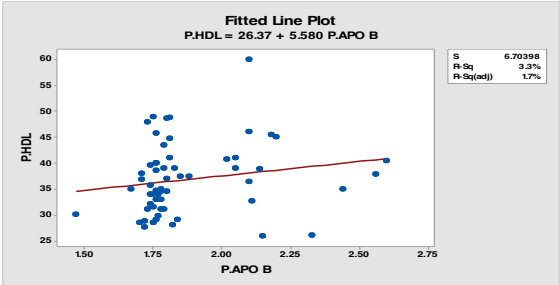

Figure 6: correlation between Apo B and HD