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Research Article

Association between Non-alcoholic Fatty Liver Disease and Selected Physiological and Biochemical Factors among Diabetic Individuals

[1], IMAN FALIH HADI
[2,] Ibtisam A. Al-ALI [3,] Hameed Hussein Al-jameel [4,] Ali Rubaie
[1,2,] Department of Biology, Collage of Science, University of Kerbala, Kerbala, Iraq
[3,] College of Medicine, University of Karbala, Karbala, Iraq [4,] Consultant Esoteric, Al-Hindiya General Hospital

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Abstract

Background: The primary function of the liver is to keep glucose and cholesterol levels at a normal level. Non-alcoholic fatty liver disease (NAFLD) is a chronic disorder that affects the liver and is characterized by insulin resistance, type 2 diabetes, and the accumulation of fat in the liver. It is possible for this to result in inflammation and scarring in the liver, which can then progress to non-alcoholic steatohepatitis (NASH) and other types of liver disease. That cannot be rectified in any way. cirrhosis of the liver.

Objective: The primary aim of the current study was to identify clinically applicable biomarkers for diabetes and fatty liver disease.

Materials and Methods:seventy women and men with nonalcoholic fatty liver disease and type 2 diabetes were among the 108 participants whose blood samples were taken for the research. The participants' ages ranged from 35 to 77. In addition, thirty-eight blood samples were obtained from individuals in good health.

Result: ALT and asprosin have a slightly negative relationship, but the connection is not statistically significant. In contrast, adiponectin and liver enzymes demonstrate a modest association. Also, with p values less than 0.05, a significant association was found between Asprosin and LDL, Insulin, and HOMA-IR. Cholesterol, TG, VLDL, HDL, and Asprosin all had modest to moderate positive associations (r between 0.3 and 0.46) with one another. Only LDL has demonstrated a negative relationship with asprosin.

Conclusion: Type two diabetes is closely associated with the development of non-alcoholic fatty liver disease, primarily due to the underlying mechanism of insulin resistanceby facilitating the liver's accumulation of free fatty acids .Adiponectin and asprosin can serve as non-invasive markers for metabolic fatty liver disease.

Corresponding Author E-mail : eman.falih@s.uokerbala.edu.iq , i.alali@uokerbala.edu.iq, hamid.toufan@uokerbala.edu.iq, Arubaie87@gmail.com
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Introduction

Most cardiovascular diseases manifest in the liver, the principal organ for maintaining average glucose and cholesterol levels. In 1986, Schaner and Thaler were the first to propose the concept of nonalcoholic fatty liver disease[1]. Non-alcoholic fatty liver disease (NAFLD) is a prevalent, progressive metabolic disorder of the liver.It is characterized by an abnormal buildup of fat in the liver, unrelated to high alcohol intake[2]. The research indicates that the global incidence of NAFLD is around 38%, making it the most common chronic liver disease globally[3]. In 2020, there was a proposal to reclassify non-alcoholic fatty liver disease (NAFLD) as metabolic-associated fatty liver disease (MAFLD) to gain a better understanding of the underlying pathophysiology and metabolic abnormalities [4].

The spectrum of NAFLD extends from basic fatty liver to non-alcoholic steatohepatitis (NASH), which may advance to fibrosis, cirrhosis, and ultimately hepatocellular carcinoma, a primary liver malignancy often arising in the setting of chronic liver disease [5].

Arterial hypertension, central obesity, dyslipidemia, insulin resistance (IR), and type 2 diabetes are all components of the metabolic syndrome, and there is evidence that NAFLD is linked to many of these. There is a direct correlation between the number of components and the likelihood of developing NAFLD and, later, advanced fibrosis [6].

Prior to imaging and liver biopsy, a diagnosis of nonalcoholic fatty liver disease (NAFLD) is often made using a patient's medical history in conjunction with serum diagnostic panels that integrate clinical factors with standard laboratory testing [7].

Studies employing controlled attenuation parameter [8] or magnetic resonance-based techniques (the gold standard) have shown an even greater incidence of steatosis in DM patients, although these imaging methods are expensive and scarce. Fibrosis, not steatosis, increases the risk of end-stage liver disease and CVD mortality, making it the actual screening target [9] . The FIB-4 index and NAFLD fibrosis score (NFS) are the most extensively used blood diagnostic panels because they may predict liver-related mortality in NASH patients [10].

The interplay between inflammatory processes and dysregulated lipid metabolism plays a vital part in the onset and development of NAFLD. Insulin resistance is a well-established contributor to NAFLD. The coexistence of NAFLD and type 2 diabetes poses a significant threat to public health[5]. Multiple large-scale studies conducted across different countries have demonstrated that NAFLD is linked to an elevated risk of developing type 2 diabetes.[6]. These studies demonstrate that NAFLD is a warning factor for type 2 diabetes and that an increased risk of NAFLD may be associated with raised blood glucose levels as well as a higher risk of more severe hypertension, atherogenic dyslipidemia, cardiac arrythmias, and cardiovascular events. people with type 2 diabetes at such high risk of steatohepatitis and the eventual development of cirrhosis multiple factors appear to be at play,these

includes genetic factors that modulate insulin action or hepatocyte lipid metabolism and a web of acquired factors driven by insulin resistance, such as glucotoxicity and lipotoxicity linked to dysfunctional adipose tissue and ectopic fat accumulation in the liver in obese and diabetic people[11]. Insulin resistance alters glucose and lipid metabolism, intracellular inflammatory pathways, mitochondrial malfunction, and endothelial reticulum stress... Higher hyperinsulinemia, atherogenic dyslipidemia, and adipose tissue, hepatic, and muscle insulin resistance are frequent causes.

The therapy for NAFLD and NASH is unknown. Weight reduction and low-fat diet are advised. The multiple diseases of

NAFLD and NASH make it difficult to determine the best pharmacological treatments. The ultimate therapy aim is histologic steatosis,

inflammation, and fibrosis improvement. Lifestyle change, weight reduction surgery, and medication are now treatment options [13].

MATERIALS AND METHODS

Patients

This case control study was conducted at Al-Hindiya General Hospital and Imam Hassan Hospital in Karbala Governorate between October 2023 and April 2024. 108 blood samples were collected from participants aged 35 - 77. Of these, seventy samples were obtained from male and female visitors to the hospital's advisory clinics who were diagnosed with type 2 diabetes and non-alcoholic fatty liver disease. The comparison group consisted of thirty-eight blood samples collected from healthy individuals of similar age and gender distribution.

The questionnaire data collected for all participants included measurements of their height, weight, and body mass index, calculated by dividing weight by the square of height in meters. Physician assistants took these measurements. Additionally, comprehensive medical histories and physical examinations were conducted on all patients who consented to gather information on the duration of their diabetes, medication use, and any chronic conditions. The patients were also confirmed to have non-alcoholic fatty liver disease using imaging techniques such as ultrasoundb by (Siemens SONY sonar) to examine their livers.

The exclusion criteria were:

(a) history of alcohol ingestion, (b) malignancy, (c) previous gastrointestinal tract surgery, (d) presence of any liver

disease that can cause fatty liver, such as chronic hepatitis C, autoimmune liver disease, and Wilson’s disease.

Clinical and laboratory information

Participants fasted for 8 to 12 hours before having their blood drawn. The blood samples were analyzed for glucose, triglycerides, serum ALT, AST, cholesterol, vitamin D, albumin, uric acid, and insulin. The LDL-cholesterol ratio was also measured. The VLDL ratio was calculated using the formula VLDL=TG/5. The HOMA IR was determined by multiplying the fasting insulin and fasting plasma glucose and dividing by 405. The tests were conducted using a Monaech 240 device from Biorex to measure the chemical components. A SYSMEX XP-300 device from CBC Company was utilized to examine the complete blood and platelet counts. The sandwich-ELISA method was employed to assess the hormones asprosin and adiponectin levels.

Statistical Analysis

All statistical analyses were conducted with GraphPad Prism software. Visual and analytical methods were utilized to ascertain the distribution of variables. Correlations among variables were examined utilizing Pearson and Spearman tests. A p-value below 0.05 was deemed statistically significant.

Results

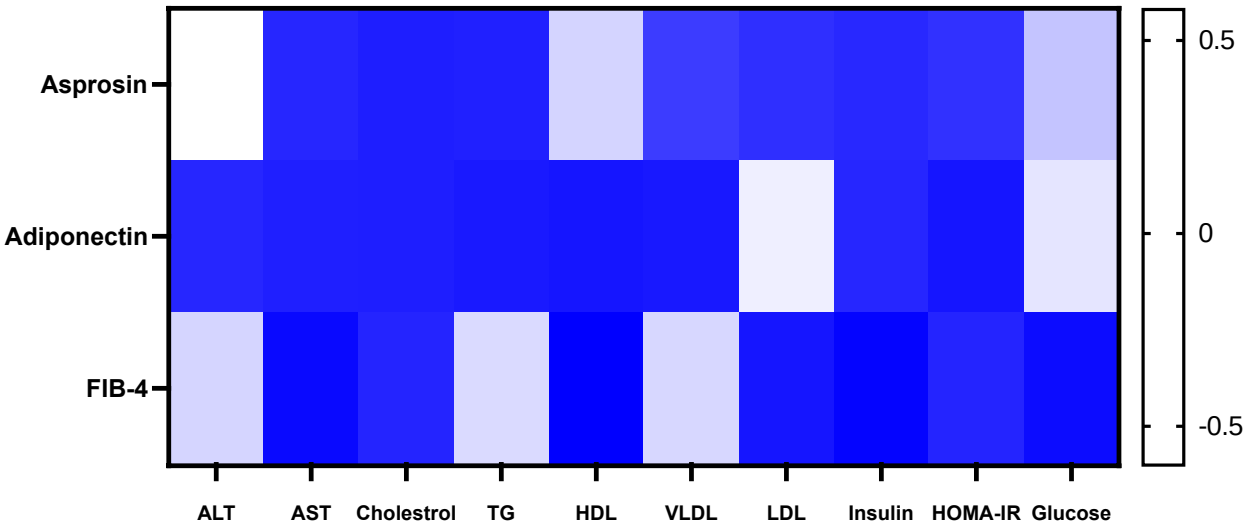

Figure (1) The correlation coefficient (r) between biomarkers among patients with diabetes mellitus with fatty liver compared to the control group

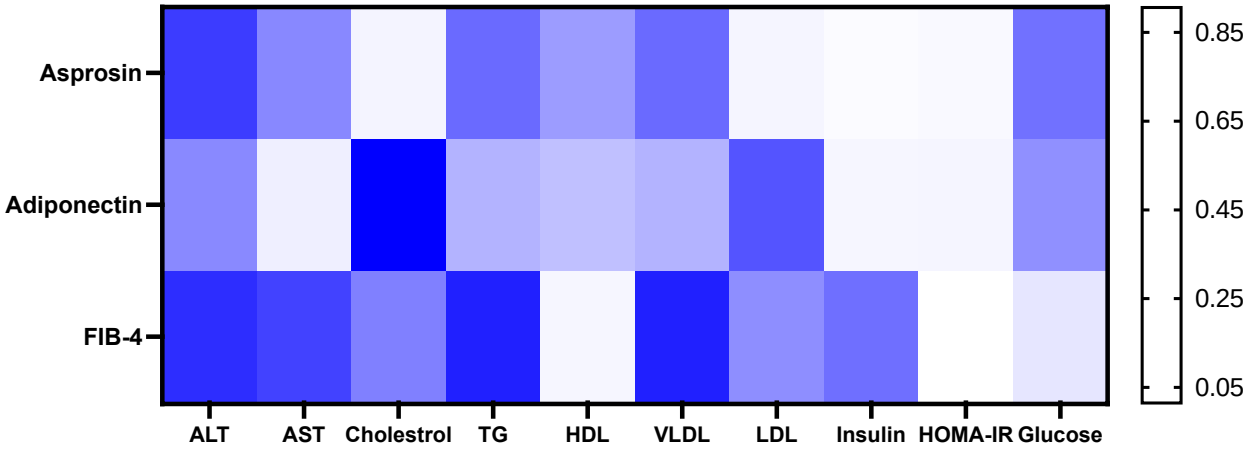

Figure (2) The significant *P* value between biomarkers among patients' diabetes mellitus group with fatty liver compared to the control group.

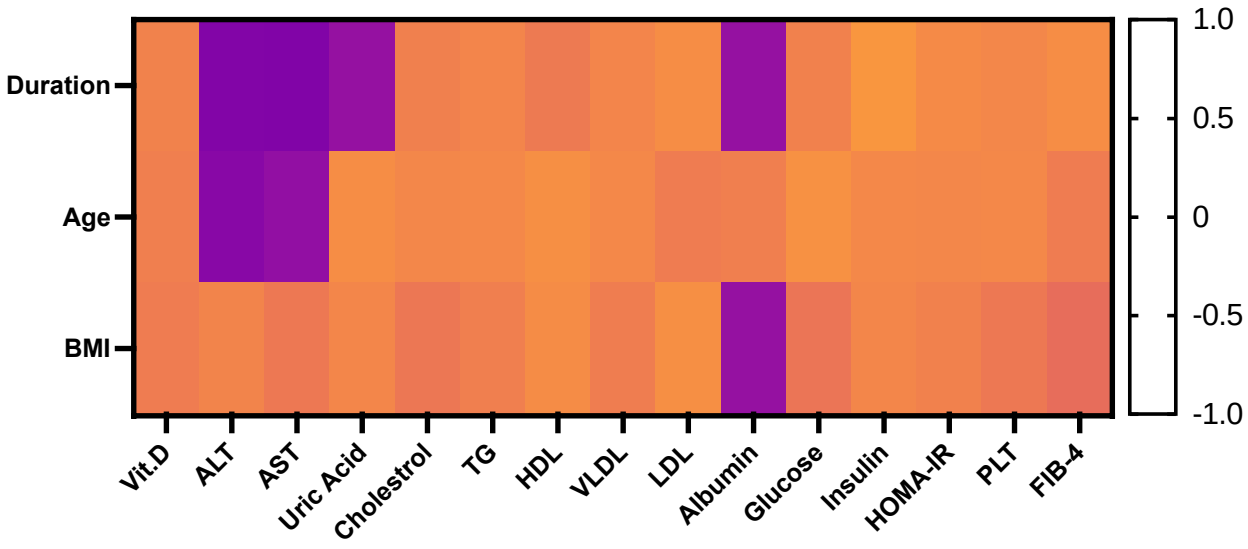

Figure (3) The correlation coefficient (r) between biomarkers among patients with diabetes mellitus with fatty liver based on the Duration of disease, Age and BMI

The Pearson correlation analysis was conducted on patients with Diabetes Mellitus and Fatty Liver to investigate the relationships among the critical measured parameters. The study revealed numerous significant correlations between the parameters and reported the corresponding p-values. Liver Enzymes, namely ALT, show a non-significant moderate negative correlation (r = -0.602) with Asprosin and a weak correlation with adiponectin. Results also showed a significant correlation between Asprosin and LDL, Insulin and HOMA-IR; p values were <0.05, as presented in Figure (1)

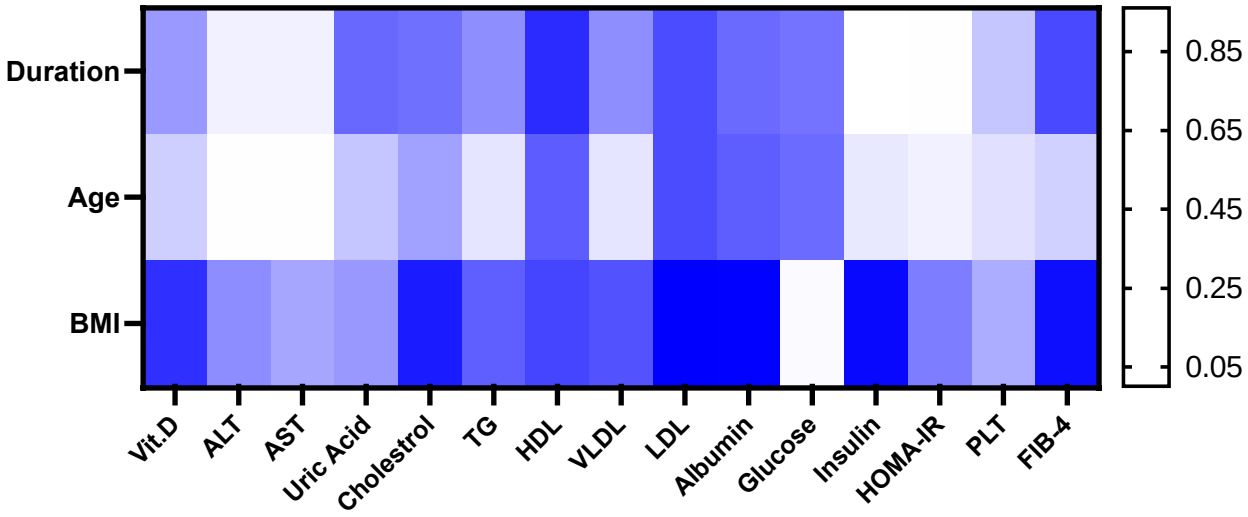

Figure (4) The significance of the *P* value for biomarkers among patients with diabetes mellitus with fatty liver based on the Duration of disease, Age and BMI

Figures (3) & (4) summarized the correlation coefficients (r) and *P* values between various biomarkers in a group of patients with diabetes mellitus and fatty liver

Discussion

The lipid profile markers showed positive correlations for cholesterol, triglycerides, VLDL, and HDL but a negative correlation for LDLwith Adiponectin. Adiponectin had weak positive correlations with most markers, except LDL and glucose, and a significant correlation with insulin. The liver fibrosis marker FIB-4 had negative correlations with ALT and HDL, suggesting better liver function with lower FIB-4 scores. FIB-4 also showed a significant correlation with Homo-IR at $p<0.005$. Data is shown in Figure 2.

Similar findings have been observed, with elevated levels of circulating asprosin being found in individuals with fatty liver disease [14].

These levels were found to be associated with HOMA-IR, which indicates that there is a strong connection between asprosin and insulin resistance in non-alcoholic fatty liver disease. The results of paired analysis suggest that the variations in asprosin levels that were detected could be predominantly related to fatty liver disease rather than to changes in metabolic processes. As an additional point of interest, the findings demonstrated a favorable association between asprosin and HOMA-IR This is consistent with previous studies[15].

According to the findings of the multivariate linear regression analysis, HOMA-IR was found to have an independent correlation with asprosin in the group of individuals with fatty liver. It has been proven in previous studies that the HOMA-IR has an excellent indicative value in fatty liver disease[16], with the level of triglycerides and body mass index .These findings suggest that odd HOMA-IR may indicate alterations within the liver, and the elevated asprosin, which is intimately associated with HOMA-IR, may primarily be due to liver-specific alterations.

The findings suggest that FLD and T2DM share standard features associated with insulin resistance. FLD was associated with insulin resistance in both hepatic and extrahepatic tissues, including muscle and adipose tissue. Numerous investigations have shown a diminished ability of insulin to inhibit endogenous glucose synthesis, signifying hepatic insulin resistance and an approximate 50% reduction in glucose clearance, indicative of decreased systemic insulin sensitivity [17]. Recent studies have suggested that FLD may be an independent contributor to the risk of developing T2DM [18]. Changes in asprosin levels may occur across the FLD-T2DM spectrum and be associated with insulin resistance. However, the specific pathological mechanism by which asprosin contributes to insulin resistance in FLD patients is unclear. Recent research by Lee et al. shows that recombinant asprosin can exacerbate inflammation, impair glucose-stimulated insulin secretion, induce apoptosis through TLR4/JNK signaling, and participate in a self-perpetuating cycle of hyperlipidemia-driven β-cell dysfunction and inflammation in the context of type 2 diabetes [19]. The inflammatory response that was mediated by TLR4 and JNK was found to have a strong correlation with both FLD and insulin resistance [20]. There is a possibility that asprosin dysregulation is also operational through this pathway. There was a positive correlation between the levels of asprosin and the concentrations of triglycerides, but there was no independent link between the levels of triglycerides and the levels of asprosin in patients who had fatty liver disease. Despite this, there was a significant independent association between TG levels and adiponectin [21].

. The correlations are assessed based on three factors: Duration of disease, Age, and BMI. The strength of the correlations can be categorized as weak (0.3-0.5), moderate (0.5-0.7), or firm (0.7-1.0). In this dataset, most correlations are weak, suggesting limited linear relations between the variables. Results indicated that there was a significant negative correlation ($r < -0.4$) between Age and both ALT (alanine aminotransferase) and AST (aspartate aminotransferase) levels. This suggests that ALT and AST levels tend to decrease with increasing age in this patient group This is consistent with previous studies [22].

Declining liver enzyme levels with age may be attributed to a reduction in the size or functioning of the normal liver.

Comparative studies in rats have demonstrated that older livers exhibit a slower and less robust regenerative ability, diminished homeostatic capacity, and a lower inflammatory response rate.[23]. In rat liver transplantation models, older livers demonstrated reduced size, elevated hepatocyte degeneration, and increased scarring [24]. Likewise, in humans, aging livers exhibit a gradual reduction in size and blood supply, along with changes associated with the buildup of oxidative damage[25].

The present study found that lipid biomarkers, including triglycerides, VLDL, LDL, total cholesterol, and HDL, were positively correlated with all three factors examined: disease duration, age, and body mass index. This suggests that these lipid levels rise with longer disease progression, older age, and increased adiposity. These findings align with prior research that has established the close connections between obesity, dyslipidemia, and fatty liver disease[26]. An imbalance between the processes of acquiring and disposing of lipids, which are controlled by four key mechanisms, is the cause of the accumulation of fat in the liver. These mechanisms include the absorption of lipids that are circulating in the blood, the synthesis of new lipids, the breakdown of fatty acids, and the export of lipids in very low-density lipoproteins [27].

The study found that Vitamin D, Uric Acid, Glucose, Insulin, and HOMA-IR had weak positive associations with age and BMI. HOMA-IR also showed a weak positive correlation with disease duration This is consistent with previous study [28].

non-alcoholic fatty liver disease by facilitating the liver's accumulation of free fatty acids[29]. One of the primary drivers of the increasing prevalence of NAFLD is the rise in insulin resistance observed in developed nations[30]. Most studies demonstrate that insulin resistance has predictive value for developing liver fibrosis [31, 32]. Even though studies express contrary views[33]. Insulin resistance is an independent risk factor that can be used to predict liver fibrosis, according to recently published research that lends credence to this viewpoint [34].

high serum uric acid levels were also found to be associated with an increased likelihood of being obese. The connection between serum uric acid and obesity can be explained by a number of different mechanisms. It is possible that obesity or extra body fat is linked to increased serum uric acid synthesis and insufficient excretion as a result of insulin resistance. This can result in impaired uric acid metabolism and hyperuricemia. While this is going on, serum uric acid can also play a role in obesity by encouraging the creation of fat in the liver and the peripheral tissues. This can lead to a vicious cycle of hyperuricemia and obesity. It is also possible that dysfunctions in the metabolism of glycolipids and uric acid could exacerbate the presence of these two components. Being aware of the tight biological connection that exists between serum uric acid and body mass index, it is of the utmost importance for preventive medicine to conduct a thorough analysis of the interaction that exists between the two [35, 36].

Notably, metabolic syndrome has been linked to lower serum albumin concentrations, especially in the presence of abdominal obesity, high triglyceride levels, and elevated blood sugar[37]. Albumin, which is produced by the liver, is involved in both the process of anti-inflammatory and anti-oxidative defense [38]. Individuals who suffer from liver cirrhosis have abnormalities in the structure and function of albumin in the serum, as well as a decreased albumin synthesis [39]. Multiple studies have demonstrated that patients with non-alcoholic fatty liver disease (NAFLD) have a worse prognosis when they have hypoalbuminemia [40, 41,42].

Conclusion

Type two diabetes is closely associated with the development of non-alcoholic fatty liver disease, primarily due to the underlying mechanism of insulin resistance.Adiponectin

and asprosin can serve as non-invasive markers for metabolic fatty liver disease.

Disease duration, age, and BMI are strongly linked with lipid levels, whereas asprosin levels relate to insulin resistance.

physiological markers had weak linear relationships with illness duration, age, and BMI .

Adiponectin demonstrates greater efficacy than asprosin in differentiating between individuals who are ill and those who are healthy

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Conflict of interest disclosure

The authors state they have no conflict of interest related to this work.

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