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Type 2 Diabetes with Insulin Resistance Markers Fatty Liver Disease: A Biochemical Approach

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A b s t r a c t

This study examined the correlation between type 2 diabetes mellitus (T2DM), insulin resistance, and fatty liver disease (FLD) using biochemical assessment of 50 diabetic patients diagnosed with FLD in comparison to 40 healthy persons. The findings indicated a substantial elevation in HOMA-IR among the patients (16.25±12.75) relative to the control group (1.74±0.96; p<0.001), signifying an increase of nearly ninefold. The C-peptide level was significantly enhanced in the patients (3.88±2.70) compared to the control group (2.64±1.02; p<0.001), and insulin levels were nearly 3.5 times more (31.61±21.53 vs. 8.70±5.16; p=0.007).
Concerning hepatic functioning, patients exhibited markedly elevated levels of AST (37.00±19.34 vs. 22.53±5.11; p<0.001) and ALT (41.84±22.48 vs. 24.63±7.29; p<0.001), signifying hepatocellular injury, although ALP levels did not demonstrate a significant difference (110.70±45.97 vs. 104.20±22.08; p=0.414). A modest yet statistically significant reduction in albumin levels was noted (4.27±0.58 vs. 4.41±0.30; p=0.001).
The HbA1c level in patients was considerably elevated at 8.24±1.28 compared to the control group at 4.93±0.47 (p<0.001), reflecting inadequate blood sugar regulation and a level about 67% greater in the diabetes-FLD cohort. Clinically, 78% of individuals had non-alcoholic fatty liver disease (NAFLD), whereas 22% presented with alcoholic fatty liver disease, underscoring the prevalence of NAFLD among diabetes patients.

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1. INTRODUCTION

Insulin resistance (IR) is defined by a diminished responsiveness of insulin-sensitive organs and tissues to insulin stimulation. Insulin resistance (IR) is characterized by diminished insulin sensitivity resulting from a suboptimal response to insulin signals regulating blood glucose levels. This abnormal physiological response elevates plasma insulin levels, resulting in hyperinsulinemia (Kashyap & Defronzo, 2007; Li et al., 2022; Szablewski, 2024). The liver plays a vital role in the balance of glucose and fat metabolism in the body. Insulin resistance (IR) is considered a major causative factor for metabolic disorders in the liver, such as metabolic dysfunction-associated steatotic liver disease (MASLD), which is one of the most common chronic liver disorders and a precursor to a wide range of liver diseases, ranging from fatty liver, liver fibrosis, and cirrhosis, ultimately leading to hepatocellular carcinoma (Bo et al., 2024).

Type 2 diabetes is a metabolic disorder clinically defined by both acute and chronic hyperglycemia. This disorder is frequently related with liver problems. The spectrum of hepatic disorders extends from mild hepatitis to Metabolic associated fatty liver disease (MAFLD). Metabolic associated fatty liver disease (MAFLD) encompasses non-alcoholic fatty liver (fatty liver) characterized by the absence of inflammation (normal transaminases), metabolic dysfunction-associated steatohepatitis (MASH) without fibrosis, MASH with fibrosis that may ultimately advance to cirrhosis, hepatocellular carcinoma, and liver failure resulting in mortality(Niranjan, Phillips, & Giannoukakis, 2023). As type 2 diabetes advances to a

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chronic phase, it can inflict considerable harm on several organ systems, including the eyes, blood vessels, kidneys, and cardiovascular system, and is frequently linked with illnesses such as dyslipidemia and hypertension(Ranade et al., 2024). The global burden of diabetes is considerable, and its growing prevalence poses significant pressure on healthcare systems, exacerbating preexisting societal challenges such as food insecurity and poverty (Ogunjobi et al., 2025)

The development of IR is influenced by several factors, such as age, genetic predisposition, obesity, oxidative stress, among others. Irregularities in the insulin-signaling pathway—encompassing defects in insulin receptors, internal environmental disruptions, and metabolic changes in muscles, liver, and intracellular organelles—are central to the pathogenesis of IR (Mir et al., 2025; Sinaiko & Caprio, 2012).

In addition to that T2DM and fatty liver disease (FLD) are common and interrelated conditions that influence one another through shared mechanisms. A link between T2DM and FLD has been hypothesized. Lipid accumulation in the liver has been proposed as a contributing factor in the development of T2DM . Therefore, the early detection and intervention of FLD are essential to prevent disease progression toward fibrosis, cirrhosis, and hepatocellular carcinoma(Tanase et al., 2020).

Fatty liver disease (FLD) is highly prevalent among individuals with excessive alcohol intake as well as those with obesity, particularly in the context of insulin resistance (Habibullah et al., 2024; Mitra, De, & Chowdhury, 2020).

Non-alcoholic fatty liver disease (NAFLD) and alcoholic fatty liver disease (AFLD) are the two predominant metabolic liver disorders globally. Metabolic liver illnesses profoundly impact human health and quality of life, resulting in substantial public health challenges and considerable medical burdens. Fat buildup, insulin resistance, oxidative stress, inflammation, and dietary practices are intricately associated with non-alcoholic fatty liver disease and alcoholic fatty liver disease.(Zhang et al., 2021).

(MAFLD) and (AFLD) are linked to extrahepatic risk factors and consequences, including metabolic syndrome, characterized by obesity, hypertension, type 2 diabetes, and chronic kidney disease. Alcoholic fatty liver disease is linked to hypertension and cardiovascular illnesses, as well as harm to other organs. This narrative review examines the associations, causal variables, and diagnostic biological determinants linking (MAFLD) and (AFLD) to elevated mortality rates (Johnston, Patel, & Byrne, 2020):

1. 1. Patients and Methods

Fifty samples were collected from the Najaf Diabetes and Endocrinology Center and the Specialized Hospital for Digestive and Liver Diseases. The samples comprised male patients aged 30 to 60 years. They received a diagnosis of type 2 diabetes and fatty liver disease upon confirmation of an 8-12 hour fasting period before to the test. The Research Ethics Committee of the College of Education, University of Karbala, in conjunction with the Diabetes and Endocrinology Center and the Gastroenterology and Liver Diseases Hospital in Najaf Al-Ashraf, sanctioned the study protocol. The investigation occurred from November 2024 to May 2025 .

The blood samples underwent centrifugation for 5 minutes at 5000 rpm to separate the serum. Biochemical analyses were conducted with a COBAS analyzer that employs electrochemiluminescence immunoassay (ECLIA) technology to quantify serum insulin and C-peptide levels. The results were juxtaposed with those derived from 40 healthy subjects in the control group. Individuals with any type of viral hepatitis and those who had recently had gastrointestinal surgery were excluded. The statistical analysis was conducted using (SPSS), and (t-test) was used to compare the groups and find (Means ± SD) and (p- value < 0.05). in addition origin Lab program it used in this study and .

1. 2. Results

Table (1.1) delineates the demographic features of the study participants, consisting of 50 patients and 40 control people. The age distribution of the patient group revealed a greater prevalence in older age groups, with the most significant proportion (n=29) in the 51-60 years group, while compared to control (n=8). In contrast, the 30-40 years cohort exhibited a significantly greater number of control subjects (n=18) compared to patients (n=8). in the 41-50 years It was observed that the numbers of control individuals (n=14) compared to patients (n=13) were very similar.

All 60 patients were identified with a hepatic disease, mainly fatty liver, with 32 categorized as Grade 1 and 18 as Grade 2. Among the patients identified with fatty liver disease, 11 were classified with Alcoholic Fatty Liver and 39 with Non-alcoholic Fatty Liver.

This signifies that the predominant etiology of fatty liver in the patient cohort (39 out of 50, 78%) was non-alcoholic, whereas a lesser fraction (11 out of 50, 22%) was ascribed to alcoholic fatty liver.

The smoking status revealed a disparity, with 11 patients identifying as smokers, whilst the control group had no smokers. All 40 control subjects identified as non-

smokers, whereas 39 patients also claimed being non-smokers. Within the patient cohort, the duration of diabetes exhibited variability: 24 patients had diabetes for over 4 years, 14 patients for 3 to 4 years, and 12 patients for 1 to 2 years. The control group exhibited no documented history of diabetes.

A familial history was more common in the patient group, with 28 patients indicating a positive family history compared to 12 control subjects. In contrast, 28 control volunteers indicated no family history, but 22 cases did.

Table (1.1): Distribution of the Samples According to demographic information

Variable	**Groups**	**Patient N=50**	**Control N=40**
Age. Groups	30-40 Years	8	18
	41-50 Years	13	14
	51-60 Years	29	8
hepatic condition			
	GRADE 1	32	0
	GRADE 2	18	0
Type of FL	alcoholic fatty liver	11	0
	non-alcoholic fatty liver	39	0
smoking	YES	11	0
	NO	39	40
Duration of diabetes	1-2 Years	12	0
	3-4 Years	14	0
	More than 4 Years	24	0
History of family	Yes	28	12
	No	22	28

Table (1.2) Mean levels of serum HOMA IR, C-PEPTIDE and INSULIN between patients with fatty liver disease (FLD), and healthy control

Biomarkers	**Patient Mean±SD N=50**	**Control Mean±SD N=40**	**P value**
HOMA IR	16.25±12.75	1.74±0.96	**<0.001[S]**
C-PEPTIDE	3.88±2.70	2.64±1.02	**<0.001[S]**
INSULIN	31.61±21.53	8.70±5.16	**0.007[S]**
T -test was *: significant at p ≤ 0.05 SD: standard deviation; S: significant; NS= Non-significant.			

Table (1.2) displays the average blood concentrations of Homeostatic Model Assessment for Insulin Resistance (HOMA-IR), C-Peptide, and Insulin in patients with fatty liver disease (FLD) (N=50) in contrast to healthy control subjects (N=40). Statistical significance was evaluated by an independent samples t-test, with a p-value of < 0.05 .being significant

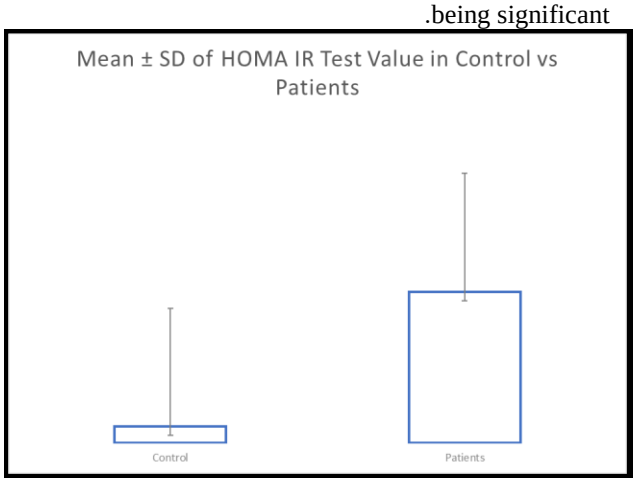

Figure 1. Patients with fatty liver disease (FLD) showed a significantly elevated average serum HOMA-IR level (16.25±12.75) compared to the healthy control group (1.74±0.96). This difference was highly statistically significant (p<0.001), indicating a profound level of insulin resistance in patients with fatty liver disease.

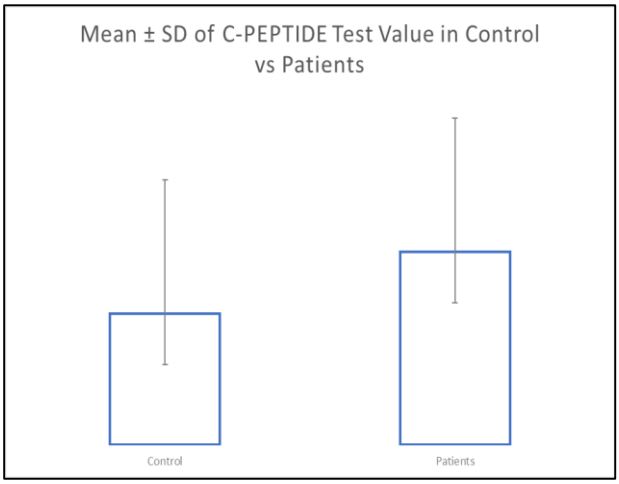

Figure 2. The average levels of C-PEPTIDE in the blood were high compared to the control group achieved a statistical significance of (P<0.001).

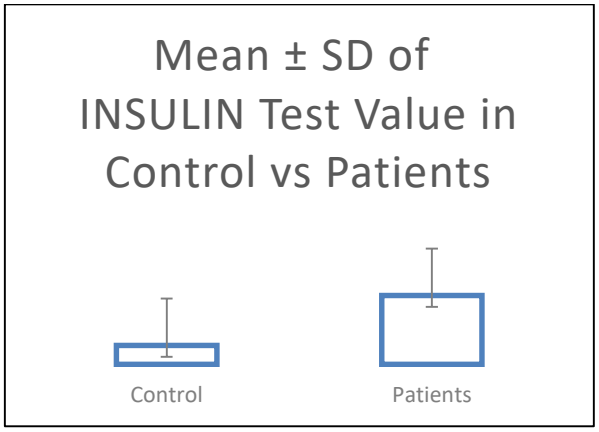

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Figure 3. Patients with FLD exhibited a markedly elevated mean blood insulin level in comparison to control controls. The observed difference was statistically significant (p=0.007).

Table (1.3) *Mean levels of serum ALBUMIN & Liver enzymes (AST, ALT and ALP) between patients with fatty liver disease (FLD), and healthy control*

Biomarkers	Patient Mean±SD N=50	Control Mean±SD N=40	P value
ALBUMIN	4.27±0.58	4.41±0.30	0.001[S]
AST	37.00±19.34	22.53±5.11	**<0.001[S]**
ALT	41.84±22.48	24.63±7.29	**<0.001[S]**
ALP	110.70±45.97	104.20±22.08	0.414[NS]
T -test was *: significant at p ≤ 0.05 SD: standard deviation; S: significant; NS= Non-significant.			

Table (1.3) displays the average serum concentrations of Albumin and liver enzymes, namely Aspartate Aminotransferase (AST), Alanine Aminotransferase (ALT), and Alkaline Phosphatase (ALP), contrasting patients with fatty liver disease (FLD) (N=50) with healthy control participants (N=40). An independent samples t-test was employed to assess statistical significance, with p≤0.05 being significant.

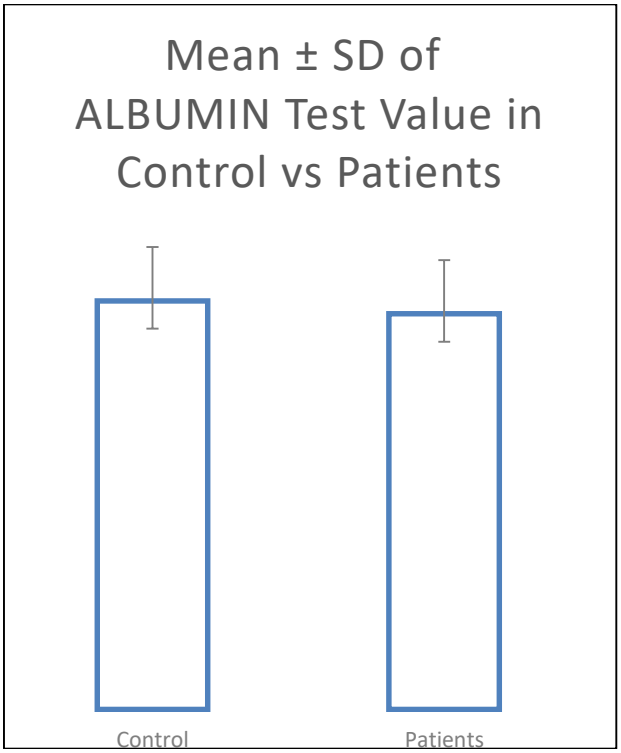

Figure 4. The average serum albumin level in FLD patients (4.27±0.58) was slightly lower than the albumin level in the control group (4.41±0.30). Despite this numerical difference, the result was statistically significant (p=0.001).

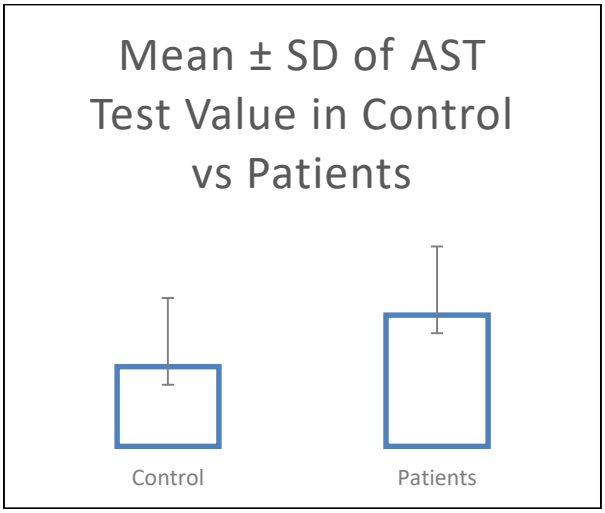

Figure 5. Patients with fatty liver disease (FLD) showed a significantly higher average serum AST enzyme level (37.00±19.34) compared to the healthy control group (22.53±5.11). The difference was statistically significant (p<0.001).

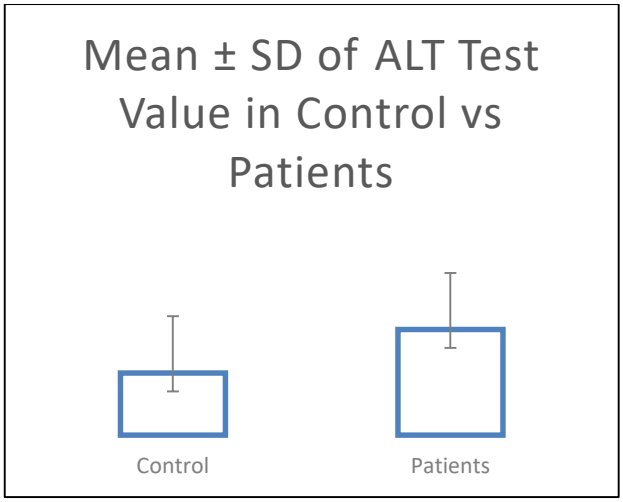

Figure 6. The ALT levels in the serum of the group of patients with fatty liver showed elevated levels compared to the control group, with a statistically significant (p <0.001).

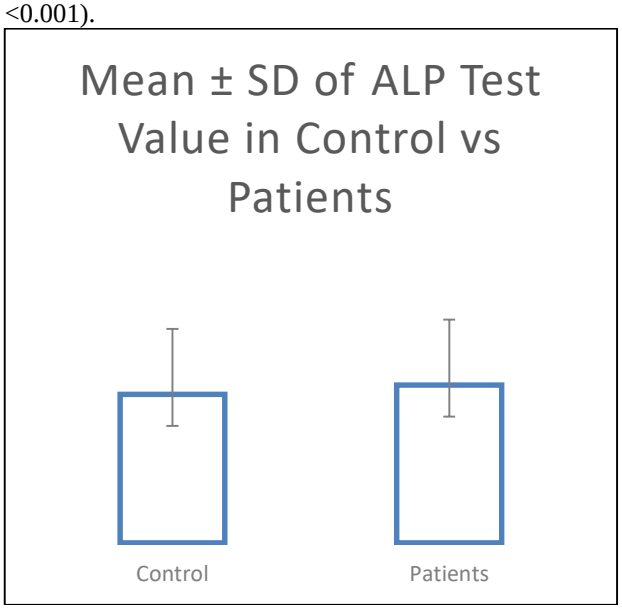

Figure 7. The mean ALP level in the serum of patients with fatty liver disease (110.70±45.97) was statistically higher than the level in the control group (104.20±22.08). However, this gap was not statistically significant (p=0.414, referred to as NS), indicating that ALP levels were similar between the two groups,

The results indicate clear differences in liver function markers between FLD patients and healthy controls

Table (1.4) *Mean levels of HbA1c Profile between patients with fatty liver disease (FLD), and healthy control*

Biomarkers	**Patient Mean±SD N=50**	**Control Mean±SD N=40**	**P value**
HbA1c	8.24±1.28	4.93±0.47	**<0.001[S]**
T -test was *: significant at p ≤ 0.05 SD: standard deviation; S: significant; NS= Non-significant.			

Table (3.5) shows that FLD patients have significantly higher HbA1c levels compared to the control group, with a p-value of <0.001, which is a clear statistical significance.

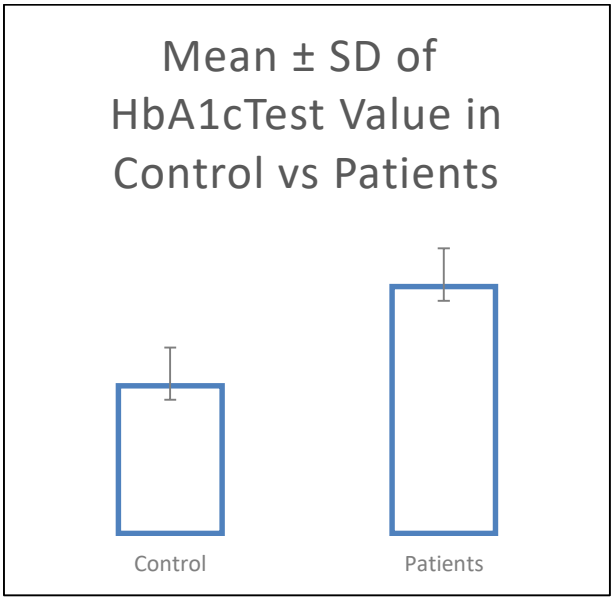

Figure 7. The HbA1c levels in the serum of the group of patients with fatty liver showed elevated levels compared to the control group, with a statistically significant (p <0.001).

1. 3. Discussion:

The results can be explained of the shared mechanisms of both diseases consequently amplify the analytical measurements, which is attributed to the role of insulin resistance and the impaired function of beta cells in responding to insulin, as well as the increased concentrations of insulin in the blood. This, in turn, affects the process of lipolysis, leading to an increased accumulation of fat in liver cells after storage, which explains the significant increase (Qasem, Al-Shami, & Al-Olofi, 2025) .

This elevation reflects the stress the liver is subjected to due to insulin resistance and the high secretion of insulin from beta cells in response to elevated blood sugar levels. C-peptide, which is secreted in equal amounts with insulin from beta cells, along with the accumulation of fat in patients and insulin resistance, supports this high blood level(Fawzi, Abd El Aziz, Mohamed, Kamal, & El Shenawy, 2025) (LIU, WANG, LIU, & WEI, 2025).

The results consistently indicate a substantial disruption in glucose homeostasis and insulin sensitivity measures in patients with fatty liver disease relative to healthy controls. Insulin levels in the blood rise in response to high sugar levels due to insulin resistance. Consequently, the pancreas reacts by secreting more insulin to lower blood sugar levels and restore glucose balance. This increase leads to the production of more fats in the liver, resulting in the accumulation of more fats within liver cells in patients with fatty liver disease, which exacerbates the condition(Bae et al., 2025; Guerra & Gastaldelli, 2020).

The significantly raised HOMA-IR in the FLD group signifies substantial insulin resistance, a characteristic of metabolic dysfunction frequently linked to fatty liver disease. These findings corroborate the notion that insulin resistance is pivotal in the development and advancement of fatty liver disease(Sung & Kim, 2011).

Significant changes occur in the reduction of albumin protein levels in fatty liver disease, but in advanced cases of liver damage and albumin leakage into the blood due to liver cell damage. However, in our study, there were few cases of fibrosis, so this did not cause a significant statistical change. But the role of albumin remains important and crucial in this disease(Takahashi et al., 2023).

Indicating an increase in AST levels in the FLD group, This increase is due to liver fibrosis and inflammation associated with fatty liver, resulting from oxidative stress, fat accumulation within liver cells, and mitochondrial damage in the liver .An elevated AST is considered a clear clinical biomarker indicating fatty liver disease. (Paulino, Cuthrell, & Tzenios, 2024; Sharma & Arora, 2020; Wang et al., 2024).

The accumulation of fat leads to inflammation and oxidative stress, resulting in damage to the cell membrane and the leakage of the AST enzyme from the cytoplasm into the blood. This enzyme is considered a reliable marker and indicator of the onset of liver injury, as well as the appearance of fibrosis and liver failure (Paulino et al., 2024; Sharma & Arora, 2020; Wang et al., 2024; Zhyzhneuskaya et al., 2025).

As it is abundantly expressed in the liver and bones. Often, when it rises due to liver conditions, it is the result of bile duct obstruction or a severe inflammatory process. Therefore, an elevated alkaline phosphatase level may indicate the severity of liver disease, such as the progression of fatty liver disease (FLD) to non-alcoholic steatohepatitis (NASH), and even cirrhosis and fibrosis. Given this possibility, alkaline phosphatase can be a useful marker for monitoring the progression of fatty liver disease (FLD) in patients with type 2 diabetes(Aransiola & Balogun, 2024).

HbA1c levels, as a primary indicator for glucose control, are usually associated with improved management of type 2 diabetes and reduced risk of complications. It reflects the glycemic state over a period of 2-3 months, resulting from the breakdown of red blood cells by the spleen, which is then released into the bloodstream(Chen, Yin, & Dou, 2023).

A study conducted in 2024 on the role of diabetes in the development of fatty liver showed that there is a strong positive correlation between its increase and the worsening of fatty liver and the increase in liver fibrosis. And a study in 2025 found that HbA1c is one of the three strongest biomarkers used in the study, which helps in the early detection of fatty liver. We should not forget that the increase in HbA1c is due to chronic high blood sugar, which is stored in the liver, increasing inflammation and oxidative stress, and activating hepatic stellate cells that lead to liver deterioration and fibrosis(Han et al., 2024; Yanmei Liu et al., 2025).

1. 4. Practical implications

This study's conclusions possess numerous significant clinical and practical implications. Pragmatic ramifications. The consistent use of HOMA-IR and C-peptide evaluations is recommended as initial screening tools for type 2 diabetes patients to identify individuals at increased risk of developing fatty liver disease before the onset of significant complications. Moreover, the integration of these indicators with regular monitoring of HbA1c levels and liver enzymes (AST, ALT) can produce a comprehensive biochemical profile for the early detection and intervention of diseases.

Moreover, the results support the development of a clinical care protocol for those demonstrating elevated HbA1c (>7.5%) or increased HOMA-IR readings. This treatment may include lifestyle modifications (dietary changes and increased physical activity), biannual liver function assessments, and the timely administration of

insulin-sensitizing drugs (such as metformin or pioglitazone) under medical supervision.

Thirdly, the results of this study may serve as a foundation for health education initiatives designed to enhance awareness among diabetic patients about the critical relationship between poor blood sugar control and the development of fatty liver, emphasizing the importance of rigorous blood sugar regulation and weight management to reduce the risk of fibrosis and hepatocellular carcinoma.

From a public health perspective, integrating HOMA-IR and HbA1c into the routine clinical evaluation of diabetes patients in specialized facilities can improve early detection rates, reduce healthcare costs associated with advanced liver diseases, and ultimately promote better long-term outcomes.

1. 5. Conclusion

The current investigation establishes that individuals with type 2 diabetes and fatty liver disease demonstrate significant insulin resistance, shown by a HOMA-IR increase of nearly ninefold compared to healthy subjects. Increased C-peptide and insulin levels signify compensatory hyperinsulinemia, although markedly elevated AST and ALT levels (up to 70% higher than controls) suggest persistent liver damage. The substantial elevation in HbA1c (8.24 vs. 4.93; $p<0.001$) underscores chronic hyperglycemia as a critical contributor to the advancement of fatty liver disease.

The results underscore the significance of early biochemical monitoring (HOMA-IR, insulin, C-peptide, HbA1c, liver enzymes) in diabetic patients. to identify high-risk individuals and avert progression to fibrosis, cirrhosis. Rigorous regulation of blood glucose levels, lifestyle alterations, and specific pharmacological interventions are crucial in mitigating the combined challenges of type 2 diabetes and fatty liver disease.

REFERENCES

Aransiola, C., & Balogun, W. (2024). NON-ALCOHOLIC FATTY LIVER DISEASE AND RELATIONSHIP WITH ADIPOSITY IN NIGERIAN PATIENTS WITH TYPE 2 DIABETES MELLITUS: THE IBADAN EXPERIENCE. *Annals of Ibadan Postgraduate Medicine*, *22*(2), 74.

Bae, J. H., Lee, M. J., Kim, S. H., Kim, J. Y., Khang, A. R., Kang, Y. H., & Yi, D. (2025). Fatty liver index and risk of type 2 diabetes of adults with normoglycemia: Insights into insulin sensitivity and beta-cell function. *PloS one*, *20*(6), e0327058.

Bo, T., Gao, L., Yao, Z., Shao, S., Wang, X., Proud, C. G., & Zhao, J. (2024). Hepatic selective insulin resistance at the intersection of insulin signaling and metabolic dysfunction-associated steatotic liver disease. *Cell Metabolism*, *36*(5), 947-968.

Chen, J., Yin, D., & Dou, K. (2023). Intensified glycemic control by HbA1c for patients with coronary heart disease and Type 2 diabetes: a review of findings and conclusions. *Cardiovascular Diabetology*, *22*(1), 146.

Fawzi, M. M., Abd El Aziz, G. R., Mohamed, R. A. E. D., Kamal, M. M., & El Shenawy, A. (2025). The level of serum C-peptide signals fibrosis in obese patients with NAFLD. *The Egyptian Journal of Internal Medicine*, *37*(1), 108.

Guerra, S., & Gastaldelli, A. (2020). The role of the liver in the modulation of glucose and insulin in non alcoholic fatty liver disease and type 2 diabetes. *Current opinion in pharmacology*, *55*, 165-174.

Habibullah, M., Jemmieh, K., Ouda, A., Haider, M. Z., Malki, M. I., & Elzouki, A.-N. (2024). Metabolic-associated fatty liver disease: A selective review of pathogenesis, diagnostic approaches, and therapeutic strategies. *Frontiers in medicine*, *11*, 1291501.

Han, X., Zhang, X., Liu, Z., Fan, H., Guo, C., Wang, H., . . . Zhang, T. (2024). Prevalence of nonalcoholic fatty liver disease and liver cirrhosis in Chinese adults with type 2 diabetes mellitus. *Journal of Diabetes*, *16*(5), e13564.

Johnston, M. P., Patel, J., & Byrne, C. D. (2020). Causes of mortality in non-alcoholic fatty liver disease (NAFLD) and alcohol related fatty liver disease (AFLD). *Current Pharmaceutical Design*, *26*(10), 1079-1092.

Kashyap, S. R., & Defronzo, R. A. (2007). The insulin resistance syndrome: physiological considerations. *Diabetes and Vascular Disease Research*, *4*(1), 13-19.

Li, M., Chi, X., Wang, Y., Setrerrahmane, S., Xie, W., & Xu, H. (2022). Trends in insulin resistance: insights into mechanisms and therapeutic strategy. *Signal transduction and targeted therapy*, *7*(1), 216.

Liu, Y., Shi, R., Cao, H., Zhang, J., Li, S., Kang, X., . . . Feng, L. (2025). AIP, fatty liver, and HbA1c as modifiers of the C-index and diabetes risk relationship. *Lipids in health and disease*, *24*(1), 129.

LIU, Y., WANG, X., LIU, Y., & WEI, L. (2025). Correlation of Fasting C-peptide to Diabetes Duration Ration and Type 2 Diabetes Mellitus Combined with Metabolism-related Fatty Liver Disease. *Chinese General Practice*, *28*(23), 2852.

Mir, M. M., Jeelani, M., Alharthi, M. H., Rizvi, S. F., Sohail, S. K., Wani, J. I., . . . Alshahrani, A. M. (2025). Unraveling the Mystery of Insulin Resistance: From Principle Mechanistic Insights and Consequences to Therapeutic Interventions. *International journal of molecular sciences*, *26*(6), 2770.

Mitra, S., De, A., & Chowdhury, A. (2020). Epidemiology of non-alcoholic and alcoholic fatty liver diseases. *Translational gastroenterology and hepatology*, *5*, 16.

Niranjan, S., Phillips, B. E., & Giannoukakis, N. (2023). Uncoupling hepatic insulin resistance–hepatic inflammation to improve insulin sensitivity and to prevent impaired metabolism-associated fatty liver disease in type 2 diabetes. *Frontiers in Endocrinology*, *14*, 1193373.

Ogunjobi, T., Adeyanju, S., Akinwande, K., Obasi, D., Aigbagenode, A., Musa, A., & Abdul, S. (2025). Improving the prevention and treatment of Lean Type 2

Diabetes in Sub-Saharan Africa: A review. *European Journal of Sustainable Development Research, 9*(2).

Paulino, P., Cuthrell, K. M., & Tzenios, N. (2024). Non Alcoholic Fatty Liver Disease; Disease Burden, Management, and Future Perspectives. *International Research Journal of Gastroenterology and Hepatology, 7*(1), 1-13.

Qasem, L., Al-Shami, A., & Al-Olofi, A. (2025). Evaluation of anthropometric parameters, Liver Enzymes, Albumin, and Lipid Profile Levels in Patients with Type Two Diabetes Mellitus, in Amran City, Yemen..

Ranade, S. D., Alegaon, S. G., Khatib, N. A., Gharge, S., Kavalapure, R. S., & Kumar, B. P. (2024). Reversal of insulin resistance to combat type 2 Diabetes mellitus by newer thiazolidinedione's in fructose induced insulin resistant rats. *European Journal of Medicinal Chemistry, 280*, 116939.

Sharma, P., & Arora, A. (2020). Clinical presentation of alcoholic liver disease and non-alcoholic fatty liver disease: spectrum and diagnosis. *Translational gastroenterology and hepatology, 5*, 19.

Sinaiko, A. R., & Caprio, S. (2012). Insulin resistance. *The Journal of pediatrics, 161*(1), 11.

Sung, K.-C., & Kim, S. H. (2011). Interrelationship between fatty liver and insulin resistance in the development of type 2 diabetes. *The Journal of Clinical Endocrinology & Metabolism, 96*(4), 1093-1097.

Szablewski, L. (2024). Changes in cells associated with insulin resistance. *International journal of molecular sciences, 25*(4), 2397.

Takahashi, H., Kawanaka, M., Fujii, H., Iwaki, M., Hayashi, H., Toyoda, H., . . . Munekage, K. (2023). Association of serum albumin levels and long-term prognosis in patients with biopsy-confirmed nonalcoholic fatty liver disease. *Nutrients, 15*(9), 2014.

Tanase, D. M., Gosav, E. M., Costea, C. F., Ciocoiu, M., Lacatusu, C. M., Maranduca, M. A., . . . Floria, M. (2020). The intricate relationship between type 2 diabetes mellitus (T2DM), insulin resistance (IR), and nonalcoholic fatty liver disease (NAFLD). *Journal of diabetes research, 2020*(1), 3920196.

Wang, S., Lin, X., Zhu, C., Dong, Y., Guo, Y., Xie, Z., . . . Chen, M. (2024). Association between nonalcoholic fatty liver disease and increased glucose-to-albumin ratio in adults without diabetes. *Frontiers in Endocrinology, 14*, 1287916.

Zhang, P., Wang, W., Mao, M., Gao, R., Shi, W., Li, D., . . . Meng, X. (2021). Similarities and differences: a comparative review of the molecular mechanisms and effectors of NAFLD and AFLD. *Frontiers in Physiology, 12*, 710285.

Zhyzhneuskaya, S., Al-Mrabeh, A., Peters, C., Barnes, A., Hollingsworth, K., Welsh, P., . . . Taylor, R. (2025). Clinical utility of liver function tests for resolution of metabolic dysfunction-associated steatotic liver disease after weight loss in the Diabetes Remission Clinical Trial. *Diabetic Medicine, 42*(3), e15462.