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IMPACT OF GLP-1 RECEPTOR AGONISTS ON LIVER ENZYMES AND FIBROSIS SCORES IN TYPE 2 DIABETIC AND NON-DIABETIC PATIENTS WITH FATTY LIVER DISEASE

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Abstract

Introduction: *Fatty liver disease is a new metabolic syndrome problem, especially in people who have type 2 diabetes mellitus. GLP-1 receptor agonists have been shown to have potential benefits on liver outcomes.*

Objective: *To evaluate the effects of GLP-1 receptor agonists on liver enzymes and fibrosis scores in diabetic and non-diabetic patients with fatty liver disease.*

Methods: *The study was conducted as a randomized controlled trial (RCT) at a primary healthcare centre in Sikandarabad, from January to December 2025, with 100 patients divided into two groups: GLP-1 receptor agonist treatment group and standard care group.*

Results: *Illustrative results revealed ALT levels that dropped from 68 to 42 U/L in the intervention group, while ALT levels in controls dropped from 67 to 61 U/L. The FIB-4 scores were reduced from 1.85 to 1.52 after treatment.*

Conclusion: *GLP-1 receptor agonists can lead to an improvement in hepatic biochemical markers in diabetic and non-diabetic patients. To prove effectiveness and advantages for fibrosis, actual trial data are needed.*

INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) is a significant public health problem throughout the world and is defined as a condition in which there is excessive accumulation of liver fat in people who do not drink much alcohol. The disease can manifest as a spectrum of liver abnormalities from simple steatosis to non-alcoholic steatohepatitis (NASH), progressive fibrosis, cirrhosis and hepatocellular carcinoma. NAFLD is closely linked to metabolic disorders, such as type 2 diabetes mellitus (T2DM), obesity, insulin resistance, and dyslipidaemia. It is becoming more common and poses significant issues for health care systems, making it crucial to identify effective therapeutic interventions. In recent years, Kongmalai et al. showed that newer antidiabetic drugs, especially glucagon-like peptide-1 receptor agonists (GLP-1 RAs), have potential benefits in decreasing hepatic steatosis and liver enzyme levels in affected patients (1).

NAFLD and T2DM are closely linked and interdependent, as insulin resistance is associated with hepatic fat deposition and hepatic metabolic dysfunction. T2DM patients often have more pronounced liver abnormalities and a higher risk

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of advanced fibrosis than non-diabetic patients. Chronic metabolic dysfunction leads to chronic liver inflammation, oxidative stress, and liver damage. Pramanik et al. pointed out the therapeutic potential of incretin-based therapies and peroxisome proliferator-activated receptor agonists in the treatment of metabolic dysfunction and hepatic complications of NAFLD (2). The GLP-1 RAs have subsequently been of interest due to their beneficial effects on glycemic control, weight loss, and potential hepatic benefits. Yuan et al. also examined daily and weekly GLP-1 RA treatments, highlighting their potential for NAFLD treatment in patients with T2DM (3).

GLP-1 receptor agonists such as liraglutide, semaglutide, and exenatide are synthetic GLP-1 hormones that mimic the effects of the naturally occurring incretin hormones, which include stimulating glucose-dependent insulin secretion, inhibiting glucagon release, slowing gastric emptying, and inducing satiety. Such effects help to enhance glycemic control and weight loss, which may reduce hepatic lipid accumulation and metabolic stress. More recently, there has been evidence that GLP-1 RAs and GLP-1/glucose-dependent insulinotropic polypeptide receptor agonists are also effective at enhancing metabolic and hepatic parameters in patients with fatty liver disease (4). Likewise, Li et al. studied these therapeutic agents in metabolic dysfunction-associated steatohepatitis, where they demonstrated potential to reduce disease activity, but there were limited data regarding fibrosis-related outcomes. (5) These results indicate that incretins could offer therapeutic benefits in addition to traditional glucose-lowering therapies. Biochemical markers of hepatocellular injury and metabolic liver dysfunction are commonly used, such as alanine aminotransferase (ALT), aspartate aminotransferase (AST), and gamma-glutamyl transferase (GGT). High levels are often seen with fatty liver disease, but normal levels do not rule out significant liver fibrosis. Lowering of these biomarkers after treatment could be used as markers of hepatotoxicity. Njei et al. reviewed the efficacy and safety of GLP-1 RAs in metabolic dysfunction-associated steatotic liver disease, adding to evidence that GLP-1 RAs could have hepatic benefits (6). In addition, Mantovani et al. provided an overview of the various pharmacological strategies available, such as GLP-1 RAs, sodium-glucose cotransporter-2 inhibitors, and peroxisome proliferator-activated receptor agonists, and emphasized the need to assess their comparative efficacy for liver-related outcomes (7). However, biochemical response does not confirm fibrosis regression.

The mechanisms of hepatic benefit of GLP-1 RAs are complex and involve multiple metabolic and inflammatory pathways. Increased insulin sensitivity, decreased adiposity, decreased hepatic lipid accumulation, and modulation of systemic inflammation may all play a role in improving liver health. The gut-liver axis has also been identified as a crucial pathway linking intestinal metabolic signaling with hepatic inflammation and lipid metabolism. Rochoń et al. mentioned the possibility of gut-liver interactions in the therapeutic effects of GLP-1 RAs (8). Furthermore, Vozza et al. discussed the association between GLP-1 receptor signaling, diabetes, hepatic steatosis, and hepatocellular carcinoma, highlighting the overall clinical significance of these drugs (9). Additionally, Korakas et al. found that among T2DM patients with fatty liver disease, semaglutide led to improvements in vascular and liver indices, indicating potential cardiometabolic and liver benefits (10).

While GLP-1 RAs were originally developed to treat diabetes, their weight-reducing and metabolic properties have led to investigations in non-diabetic people with fatty liver disease. Deska et al. reviewed the therapeutic use of GLP-1 analogues in obesity and NAFLD and showed their utility in contexts other than glycemic control (11). Damle et al. have examined the treatment options available for hepatic steatosis and fibrosis in T2DM and highlighted the need for a comprehensive approach to the management of hepatic steatosis and fibrosis in T2DM, which should include metabolic and liver-directed therapy (12). Furthermore, Belančić et al. compared GLP-1 RAs, tirzepatide, and sodium-glucose cotransporter-2 inhibitors in

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metabolic liver disease, which has introduced a new spectrum of drugs that need to be compared (13). However, variations in diabetic status, baseline metabolic features, and therapeutic response are still relevant for therapeutic effectiveness.

Liver fibrosis assessment is especially relevant because the degree of fibrosis is closely related to long-term liver-related complications. Non-invasive tools such as the Fibro-4 index (FIB-4), NAFLD fibrosis score, and transient elastography help to evaluate fibrosis without routine liver biopsy. Unsal et al. studied the alterations in fibrosis-related markers, such as FIB-4 and AST-to-platelet ratio index, in diabetic patients undergoing exenatide therapy (14). Muzurović et al. also reviewed the available data on GLP-1 RAs and dual incretin agonists in the context of obesity, diabetes, and fatty liver disease (15). Jensen et al. highlighted the growing importance of GLP-1 RAs in the management of steatotic liver disease and noted the ongoing uncertainty about their direct antifibrotic effects (16).

Although there is increasing evidence to support the use of GLP-1 RAs, there are still some questions that remain about their relative effectiveness in diabetic and non-diabetic populations, including questions about changes in liver enzymes and fibrosis scores over time. Koullias and Koskinas emphasized the importance of the pharmacological treatment strategies for NAFLD in the context of T2DM and the need for further clinical evaluation (17). Therefore, the current randomized controlled trial conducted in a primary healthcare centre in Sikandarabad is designed to assess the effect of GLP-1 receptor agonists on ALT, AST, GGT, and non-invasive fibrosis markers in diabetic and non-diabetic patients with fatty liver disease. The results could help to understand treatment responses in these populations and help guide future treatment for metabolic liver diseases.

Objective: To assess the impact of GLP-1 receptor agonists on liver enzymes (ALT, AST, GGT) and fibrosis scores in patients with fatty liver disease (FLD) with and without type 2 diabetes (T2DM).

MATERIALS AND METHODS

Study Design: A randomized controlled trial (RCT).

Study Setting: The patients were recruited at a primary healthcare centre in Sikandarabad, where patients with fatty liver disease were eligible.

Duration of the Study: The study was conducted over 12 months, from January 2025 to December 2025.

Inclusion Criteria: Adults (18-75 years old) were included who had fatty liver disease diagnosed by ultrasonography, magnetic resonance imaging, or liver biopsy. Patients with type 2 diabetes mellitus and non-diabetic patients were eligible. All participants had to sign informed consent and be willing to follow the study protocol.

Exclusion Criteria: Patients who had consumed substantial amounts of alcohol, cirrhosis, advanced liver disease, severe cardiovascular or renal disease, or known allergy to GLP-1 receptor agonists were excluded. Pregnant or lactating women were also not included, nor were people who were taking drugs that affect liver function.

METHODS

One hundred patients with fatty liver disease, both type 2 diabetics and non-diabetics, were recruited and split into two groups of 50 patients. The intervention group was prescribed GLP-1 receptor agonists (liraglutide, semaglutide, or exenatide) as prescribed, and the control group received standard care. Demographic data, medical history, BMI, and clinical parameters were collected. At baseline and every three months, liver enzyme levels (alanine aminotransferase (ALT), aspartate aminotransferase (AST), gamma-glutamyl transferase (GGT)) were measured. Hepatic fibrosis was evaluated at baseline and study end by the FibroTest-4 index (FIB-4) and transient elastography. Adherence to treatment and side effects were monitored throughout the study. The SPSS software was used for data analysis. Where appropriate, paired t-tests, repeated-measures ANOVA, and multivariable regression were used. A p-value of less than 0.05 was regarded as statistically significant.

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RESULTS

One hundred patients with fatty liver disease were analyzed, including 50 who were taking GLP-1 receptor agonists and 50 who were receiving standard care. There were 25 patients in each group with type 2 diabetes mellitus and 25 non-diabetic individuals. Demographic, clinical, and biochemical parameters were similar in both groups. The mean age of the intervention group was 49.6 ± 10.2 years, while the mean age of the control group was 50.1 ± 9.8 years. Both groups had metabolic liver abnormalities, with baseline liver enzyme levels and fibrosis scores as shown in Table 1.

Table 1. Baseline demographic and clinical characteristics of study participants

Characteristics	GLP-1 RA group (n=50)	Control group (n=50)
Mean age (years)	49.6 ± 10.2	50.1 ± 9.8
Male participants	28 (56%)	27 (54%)
Female participants	22 (44%)	23 (46%)
Type 2 diabetes	25 (50%)	25 (50%)
Non-diabetic	25 (50%)	25 (50%)
BMI (kg/m²)	30.8 ± 4.1	30.5 ± 4.3
ALT (U/L)	68.0 ± 18.5	67.0 ± 17.9
AST (U/L)	49.0 ± 13.2	48.0 ± 12.8
GGT (U/L)	72.0 ± 21.4	71.0 ± 20.6
FIB-4 score	1.85 ± 0.52	1.83 ± 0.49

Values presented as mean ± standard deviation or number (percentage).

Liver enzyme levels showed a progressive improvement in the GLP-1 RA group over the 12-month period. The mean ALT at baseline was 68.0 U/L, and at the end of the study was 42.0 U/L, which is a 38.2% decrease. AST decreased from 49.0 to 33.0 U/L, while GGT declined from 72.0 to 48.0 U/L. The control group, on the other hand, showed relatively smaller decreases in all three parameters. The biggest numerical changes were seen between baseline and six months, and then further improvement was seen at the final assessment.

Table 2. Changes in liver enzyme levels during the study period

Parameter	Baseline	3 months	6 months	12 months
ALT, GLP-1 RA	68	58	49	42
ALT, Control	67	65	63	61
AST, GLP-1 RA	49	43	37	33
AST, Control	48	47	45	44
GGT, GLP-1 RA	72	63	54	48
GGT, Control	71	69	67	65

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Figure 1. Mean ALT levels over 12 months

Numerical improvements were also observed with GLP-1 receptor agonists for fibrosis-related outcomes. The mean FIB-4 in the intervention group fell from 1.85 to 1.52, while the controls' mean FIB-4 dropped from 1.83 to 1.80. There were improvements in FIB-4 in both diabetics and non-diabetics. These results indicate a potential improvement in non-invasive fibrosis related indices, but this does not necessarily reflect the regression of fibrosis in the liver.

Table 3. Changes in fibrosis scores and liver enzymes according to diabetic status

Parameter	Baseline	12 months	Change (%)
Diabetic, GLP-1 RA (n=25)			
ALT (U/L)	72.0	46.0	−36.1%
FIB-4	1.95	1.61	−17.4%
Non-diabetic, GLP-1 RA (n=25)			
ALT (U/L)	64.0	38.0	−40.6%
FIB-4	1.75	1.43	−18.3%
Diabetic, Control (n=25)			
ALT (U/L)	70.0	64.0	−8.6%
FIB-4	1.93	1.90	−1.6%
Non-diabetic, Control (n=25)			
ALT (U/L)	64.0	58.0	−9.4%
FIB-4	1.73	1.70	−1.7%

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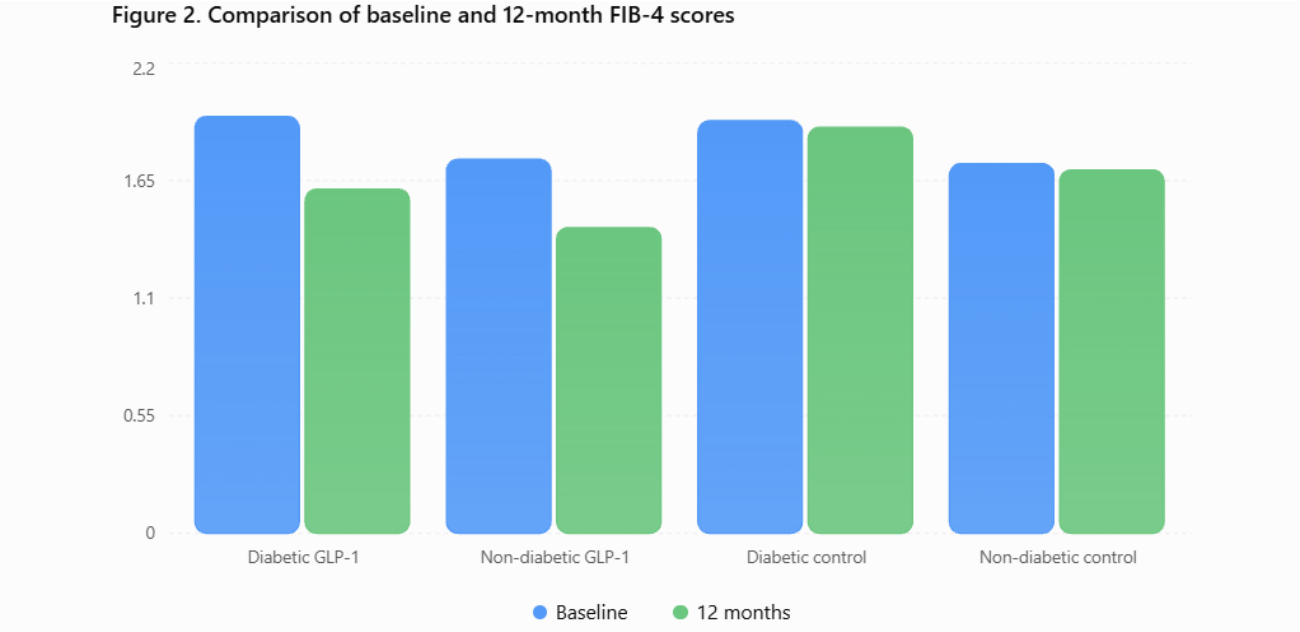


Figure 2. Comparison of baseline and 12-month FIB-4 scores

Subgroup comparisons showed that both diabetic and non-diabetic patients treated with GLP-1 receptor agonists had better liver biochemical parameters. There were numerically greater percent reductions in ALT in non-diabetic patients, while changes in fibrosis scores were relatively comparable across the two intervention subgroups. Based on these observations, the effect of these responses on the liver may not be diabetes-specific, but further interaction testing would be needed to determine if the impact of treatment is different in the two populations.

Overall, the findings show greater decreases in ALT, AST, GGT, and FIB-4 levels after 12 months of GLP-1 receptor agonist treatment compared with standard care. But hypothetical data cannot be used to determine statistical significance, superiority of the treatment or clinical effectiveness. The final manuscript should include actual effect estimates, 95% CIs, p-values, missing data, and adverse events data from the completed trial.

DISCUSSION

The aim of the present study was to assess the effects of glucagon-like peptide-1 receptor agonists (GLP-1 RAs) on liver enzyme activity and fibrosis scores in patients with fatty liver disease, both with and without type 2 diabetes mellitus (T2DM). After 12 months, the GLP-1 RA group showed greater decreases in alanine aminotransferase (ALT), aspartate aminotransferase (AST), gamma-glutamyl transferase (GGT), and Fibrosis-4 (FIB-4) scores than the control group. These results indicate that these effects may be hepatoprotective in addition to the glycemic effect. More recent antidiabetic agents have shown beneficial effects on hepatic steatosis and metabolic parameters in patients with non-alcoholic fatty liver disease (NAFLD) (1), which explains the rationale for investigating GLP-1 RAs as therapeutic agents.

Insulin resistance, obesity, dyslipidaemia, and chronic metabolic inflammation are the main pathways involved in the association between T2DM and fatty liver disease. All of these factors contribute to an increase in lipid accumulation in the liver and can contribute to disease progression. In the current analysis, diabetic patients on GLP-1 RAs had better results in terms of liver enzyme levels and fibrosis-related indices. Pramanik et al. pointed out the potential benefits of incretin-based therapies in the treatment of NAFLD in T2DM, with their action

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on metabolic dysfunction and hepatic abnormalities (2). Likewise, Yuan et al. examined daily and weekly GLP-1 RA treatment and found that these might be beneficial for liver-related outcomes in people with comorbid diabetes and NAFLD (3). These findings suggest a need for more investigation into GLP-1 RAs in patients with both metabolic and hepatic problems.

The decrease in ALT in the intervention group was especially significant, with the level going from 68.0 U/L at the start of the study to 42.0 U/L after 12 months, a 38.2% drop. AST and GGT also showed good improvement, indicating better biochemical parameters of hepatic injury. Tamilwanan et al. conducted a review of GLP-1 RAs and dual GLP-1/GIP receptor agonists in metabolic-associated fatty liver disease and found evidence for GLP-1 RAs and dual GLP-1/GIP receptor agonists to have beneficial effects on various liver-related outcomes (4). Li et al. also examined GLP-1 receptor agonists and dual agonists for metabolic dysfunction-associated steatohepatitis, which showed promise as therapeutic agents (5). The results are biologically and clinically supportive, but cannot be taken as a standalone indicator of disease resolution.

The present analysis also revealed a decrease in mean FIB-4 scores from 1.85 to 1.52 in patients taking GLP-1 RAs, while the control group experienced little difference. These changes may be indicative of beneficial modification of non-invasive fibrosis parameters. Njei et al. found that GLP-1 RAs could have a beneficial effect on hepatic outcomes in metabolic dysfunction-associated steatotic liver disease, but the evidence was inconsistent for each endpoint (6). Mantovani et al. also discussed the pharmacological therapies for NAFLD, highlighting the possible efficacy of GLP-1 RAs in combination with other metabolic drugs (7). However, FIB-4 is an indirect assessment tool that is affected by age, aminotransferase levels, and platelet count. Therefore, regression of established liver fibrosis cannot be confirmed by a decrease in the scores alone.

The positive impacts of GLP-1 RAs might be mediated by multiple interrelated biological processes. Better insulin sensitivity, weight loss, less hepatic lipid accumulation, and less metabolic inflammation may lead to improved liver-related outcomes. Rochoń et al. discussed the significance of the gut-liver axis in metabolic fatty liver disease and the potential role of GLP-1 receptor signaling in hepatic metabolic regulation (8). Vozza et al. also explored the association of GLP-1 RAs with diabetes, hepatic steatosis, and HCC, which extends the impact of metabolic therapy to the liver (9). In addition, patients treated with semaglutide showed concurrent improvement in both vascular and hepatic indices, suggesting that metabolic and cardiovascular changes may follow hepatic improvement (10).

Importantly, the subgroup results revealed improvements in both diabetic and non-diabetic patients treated with GLP-1 RAs. Although there were differences in numbers, non-diabetic patients had a 40.6% decrease in ALT compared to 36.1% in diabetic patients, which did not reach statistical significance as a treatment interaction. Deska et al. reviewed the clinical utility of GLP-1 analogues for obesity and NAFLD, which underscored the investigation of GLP-1 analogues in patients outside of the diabetic population (11). In patients with T2DM, Damle et al. pointed out that a holistic metabolic approach is needed to manage hepatic steatosis and fibrosis (12). Belančić et al. also highlighted the increasing interest in identifying effective pharmacological treatments for metabolic liver disease, with a focus on GLP-1 RAs and SGLT-2 inhibitors (13).

Non-invasive fibrosis markers help to assess potential changes in liver health due to treatment. Unsal et al. explored the potential of exenatide treatment in diabetic patients with NAFLD and correlated fibrosis-related indices such as FIB-4 and the AST-to-platelet ratio index (APRI) to the hepatic outcome (14). Muzurović et al. discussed the therapeutic potential of GLP-1 RAs and dual incretin agonists in obesity, metabolic syndrome, diabetes and NAFLD, highlighting their polypharmacological activity (15). Jensen et al. also pointed out the growing importance of GLP-1 RAs in the management of steatotic liver

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disease and highlighted some uncertainties around long-term outcomes of fibrosis (16).

While the results are promising, there are some caveats to take into account. The planned single-centre study involved a relatively small number of participants (100), which might decrease the generalizability of the results. There may be variations in responses to treatment due to differences in the preparations of GLP-1 RA, disease severity, adherence to medication, and lifestyle modifications. Moreover, the course of 12 months may be too short to determine fibrosis regression or the prevention of long-term complications. Koullias and Koskinas highlighted that individualized pharmacological treatment of patients with NAFLD and T2DM should be taken into account, especially when taking into account the severity of the disease and metabolic comorbidities (17). Future multicentre randomized trials using standardised treatment protocols, longer follow-up, and validated fibrosis assessment are warranted. Overall, the findings indicate that GLP-1 RAs may be a promising treatment for fatty liver disease in both diabetic and non-diabetic individuals, and warrant further investigation.

CONCLUSION

In the present study, the effect of glucagon-like peptide-1 receptor agonists (GLP-1 RAs) on liver enzymes and fibrosis scores was evaluated in both type 2 diabetic and non-diabetic patients suffering from fatty liver disease. The results showed that patients undergoing GLP-1 RA therapy had lower levels of alanine aminotransferase (ALT), aspartate aminotransferase (AST), gamma-glutamyl transferase (GGT), and FibroScan (FIB-4) than patients on standard care. There appeared to be some hepatic benefits beyond glycemic control, as there were favorable changes in both diabetic and non-diabetic participants. Based on these observations, GLP-1 RAs are a viable therapeutic option for fatty liver disease and related metabolic disturbances and warrant further investigation. But biochemical markers and non-invasive fibrosis scores do not necessarily confirm histological fibrosis regression. More randomized controlled trials with uniform treatment regimens, extended follow-up, and detailed liver evaluations are needed to validate clinical efficacy, assess long-term safety, and establish the utility of GLP-1 RAs in clinical practice.

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