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Effects of Weekly Semaglutide and Tirzepatide Therapy on APRI and FIB-4 Scores in Adults with Obesity: A Real-World 12-Month Study

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Abstract

Objective

Obesity is a major driver of metabolic dysfunction-associated steatotic liver disease (MASLD), a condition that may progress to advanced fibrosis and liver-related complications. Simple non-invasive indices such as the Aspartate Aminotransferase-to-Platelet Ratio Index (APRI) and Fibrosis-4 (FIB-4) score are commonly used to estimate fibrosis risk in clinical practice. Weekly incretin-based therapies, including semaglutide and tirzepatide, have emerged as highly effective treatments for obesity; however, data regarding their impact on non-invasive hepatic fibrosis markers in routine clinical settings remain limited. This study investigated changes in APRI and FIB-4 scores during one year of treatment with weekly semaglutide or tirzepatide in adults with obesity.

Methods

In this retrospective real-world study, 56 adults with obesity treated with once-weekly semaglutide or tirzepatide were evaluated. Clinical, anthropometric, and biochemical parameters were recorded at baseline, 6 months, and 12 months. APRI and FIB-4 scores were calculated using validated formulas. Longitudinal changes in body mass index (BMI) and fibrosis indices were assessed over the study period.

Results

The study population had a mean age of 42.5 ± 11.4 years, and 76.8% were women. Treatment with weekly semaglutide or tirzepatide resulted in a significant reduction in BMI, which decreased from 34.5 ± 6.3 kg/m² at baseline to 31.5 ± 6.3 kg/m² after 12 months ($p < 0.001$). APRI values declined significantly from 0.46 ± 0.47 to 0.25 ± 0.29 ($p < 0.001$). Likewise, FIB-4 scores decreased from 1.03 ± 0.87 at baseline to 0.77 ± 0.67 at month 12 ($p < 0.001$). Improvements in both fibrosis indices were accompanied by sustained reductions in body weight and BMI throughout follow-up.

Conclusion

One year of treatment with weekly semaglutide or tirzepatide was associated with significant improvements in APRI and FIB-4 scores in adults with obesity. These findings suggest that incretin-based therapies may confer benefits extending beyond weight reduction and glycemic control, potentially contributing to improvement in liver fibrosis risk. Larger prospective studies incorporating imaging and histological assessment are warranted to further clarify the hepatic effects of these agents.

Introduction

Obesity is a chronic, relapsing disease associated with substantial metabolic, cardiovascular, and hepatic complications. The worldwide prevalence of obesity has increased dramatically over recent decades, leading to a parallel rise in obesity-related disorders, including metabolic dysfunction-associated steatotic liver disease (MASLD). Currently, MASLD is recognized as the hepatic manifestation of metabolic dysfunction and affects a considerable proportion of individuals with obesity, contributing significantly to healthcare burden and long-term morbidity [1–4].

The development and progression of MASLD are closely linked to excess adiposity, insulin resistance, chronic low-grade inflammation, and altered lipid metabolism. While hepatic steatosis is often asymptomatic, a subset of patients develops progressive liver fibrosis, which is the strongest determinant of liver-related outcomes and overall mortality. Consequently, identifying individuals at risk for fibrosis has become a major focus in the management of obesity-related liver disease [1, 2, 5].

Although liver biopsy remains the reference standard for fibrosis assessment, its invasive nature limits its routine use. In everyday clinical practice, non-invasive fibrosis markers have gained increasing importance. Among these, the Fibrosis-4 (FIB-4) index and Aspartate Aminotransferase-to-Platelet Ratio Index (APRI) are simple, inexpensive, and widely available tools that can be calculated using routinely obtained laboratory parameters. These scores have demonstrated acceptable performance for risk stratification in patients with MASLD and are increasingly incorporated into clinical decision-making algorithms [6–8].

The treatment of obesity has undergone a major transformation with the introduction of incretin-based therapies. Semaglutide, a glucagon-like peptide-1 receptor agonist (GLP-1 RA), and tirzepatide, a dual glucose-dependent insulinotropic polypeptide (GIP) and GLP-1 receptor agonist, produce substantial and sustained weight reduction while simultaneously improving glycemic control and cardiometabolic risk factors [9–12]. Beyond their established metabolic benefits, emerging evidence suggests that these agents may exert favorable effects on the liver through mechanisms extending beyond weight loss alone, including reductions in hepatic fat accumulation, inflammation, and metabolic stress [13–18].

Recent studies evaluating anti-obesity medications have reported improvements in non-invasive markers of liver injury and fibrosis. In particular, pharmacological weight-loss interventions such as orlistat have been associated with reductions in APRI scores in patients with obesity [19, 20]. However, despite the rapidly expanding use of semaglutide and tirzepatide in routine obesity management, evidence regarding their effects on simple fibrosis indices such as APRI and FIB-4 remains limited, especially in real-world clinical settings.

Therefore, this study aimed to investigate the longitudinal effects of weekly semaglutide and tirzepatide treatment on APRI and FIB-4 scores in adults with obesity during a 12-month follow-up period.

Materials and Methods

Study design and patient selection

This retrospective observational study was conducted at the Department of Endocrinology and Metabolism of a tertiary referral center. Medical records of adults with obesity who received once-weekly semaglutide or tirzepatide treatment and completed 12 months of follow-up were reviewed retrospectively.

Patients aged ≥ 18 years with a body mass index (BMI) ≥ 30 kg/m² and available clinical and laboratory data at baseline, 6 months, and 12 months were eligible for inclusion. Patients with chronic viral hepatitis, autoimmune liver disease, significant alcohol consumption, decompensated liver disease, active malignancy, pregnancy, severe renal impairment, or missing laboratory parameters required for APRI and FIB-4 calculations were excluded.

The study included patients who were followed between January 2025 and January 2026, and for whom complete 12-month treatment data were available. A total of 56 patients met the inclusion criteria and were included in the final analysis.

Clinical and laboratory measurements

Demographic characteristics, comorbidities, anthropometric measurements, and laboratory parameters were obtained from electronic medical records.

Body weight and BMI were recorded at treatment initiation (baseline) and at the 6th and 12th months of follow-up. Laboratory evaluations included fasting plasma glucose, glycated hemoglobin (HbA1c), alanine aminotransferase (ALT), aspartate aminotransferase (AST), platelet count, and other routinely measured biochemical parameters.

All laboratory analyses were performed in the same hospital laboratory using standardized methods.

Calculation of fibrosis scores

Non-invasive liver fibrosis indices were calculated using established formulas.

The Fibrosis-4 (FIB-4) index was calculated as:

FIB-4 = (Age [years] × AST [U/L]) ÷ (Platelet count [10^9/L] × √ALT [U/L])

The Aspartate Aminotransferase-to-Platelet Ratio Index (APRI) was calculated as:

APRI = [(AST ÷ Upper Limit of Normal AST) × 100] ÷ Platelet count (10^9/L)

For APRI calculations, the upper limit of normal for AST was accepted as 40 U/L.

Outcomes

The primary outcomes were changes in APRI and FIB-4 scores between baseline, month 6 and month 12.

Secondary outcomes included changes in body weight and BMI during follow-up.

Statistical analysis

Statistical analyses were performed using IBM SPSS Statistics version XX (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation (SD) or median (interquartile range [IQR]) according to data distribution. Categorical variables were presented as frequencies and percentages. The normality of data distribution was assessed using the Shapiro–Wilk test and graphical methods. Comparisons of repeated measurements obtained at baseline, 6 months, and 12 months were performed using repeated-measures analysis of variance (ANOVA) for normally distributed variables and the Friedman test for non-normally distributed variables. Post hoc pairwise comparisons were conducted using Bonferroni correction when appropriate. A two-sided p-value < 0.05 was considered statistically significant.

Ethics approval

The study protocol was approved by the İnönü University Non-Interventional Clinical Research Ethics Committee. The study was conducted in accordance with the Declaration of Helsinki. Owing to the retrospective design of the study, the requirement for written informed consent was waived by the Ethics Committee.

Results

A total of 56 adults with obesity were included in the study. The mean age of the participants was 40.3 ± 13.1 years, and 43 (76.8%) were female. At baseline, the mean body weight was 97.1 ± 17.1 kg and the mean BMI was 34.8 ± 6.0 kg/m². Thirty-four patients (60.7%) received semaglutide, while 22 patients (39.3%) received tirzepatide. Baseline demographic, clinical, and laboratory characteristics are summarized in Table 1.

Changes in anthropometric, metabolic, and fibrosis-related parameters during follow-up are presented in Table 2. Significant reductions in body weight and BMI were observed throughout the 12-month follow-up period. Mean body weight decreased from 97.1 ± 17.1 kg at baseline to 88.5 ± 17.6 kg at month 6 and 87.9 ± 17.8 kg at month 12 ($p < 0.001$). Similarly, mean BMI decreased from 34.8 ± 6.0 kg/m² to 31.8 ± 6.2 kg/m² and 31.5 ± 6.3 kg/m² at months 6 and 12, respectively ($p < 0.001$). The mean weight reduction from baseline was 8.6 ± 5.0 kg at month 6 and 9.2 ± 6.1 kg at month 12, corresponding to percentage weight losses of 9.1 ± 5.2% and 9.7 ± 6.3%, respectively (both $p < 0.001$). At the end of follow-up, 24 patients (42.9%) achieved weight loss greater than 10%, whereas 18 patients (32.1%) experienced weight loss between 5% and 10%.

Liver-related biochemical parameters improved significantly during treatment. Mean AST levels decreased from 35.6 ± 26.6 U/L at baseline to 27.2 ± 22.4 U/L at month 6 and 22.8 ± 16.1 U/L at month 12 ($p < 0.001$). Likewise, mean ALT levels declined from 44.4 ± 45.8 U/L to 29.4 ± 31.2 U/L and 23.3 ± 15.0 U/L at months 6 and 12, respectively ($p < 0.001$). Significant improvements were also observed in lipid parameters. Mean LDL cholesterol decreased from 122.5 ± 34.4 mg/dL at baseline to 105.6 ± 22.8 mg/dL at month 12 ($p < 0.001$), while triglyceride levels decreased from 149.6 ± 64.3 mg/dL to 119.9 ± 51.0 mg/dL during follow-up ($p < 0.001$).

Non-invasive fibrosis markers demonstrated progressive improvement throughout the study period (Table 2, Fig. 1). The mean APRI score decreased significantly from 0.46 ± 0.47 at baseline to 0.32 ± 0.40 at month 6 and 0.25 ± 0.29 at month 12 ($p < 0.001$). Compared with baseline values, APRI scores decreased by approximately 46% at month 12. Similarly, the mean FIB-4 score declined from 1.03 ± 0.87 at baseline to 0.88 ± 0.76 at month 6 and 0.77 ± 0.67 at month 12 ($p < 0.001$), corresponding to an approximate 25% reduction from baseline. The improvements observed in APRI and FIB-4 scores paralleled the reductions in body weight, BMI, AST, and ALT levels over the course of treatment.

Table 1
Baseline demographic, clinical, and laboratory characteristics of the study population

Parameter	Value (n = 56)
Female sex, n (%)	43 (76.8)
Male sex, n (%)	13 (23.2)
Age, years	40.3 ± 13.1
Follow-up duration, months	12
Body weight, kg	97.1 ± 17.1
BMI, kg/m²	34.8 ± 6.0
Treatment, n (%)	
Semaglutide	34 (60.7)
Tirzepatide	22 (39.3)
Obesity class, n (%)	
Class I obesity (BMI 30.0–34.9 kg/m²)	37 (66.1)
Class II obesity (BMI 35.0–39.9 kg/m²)	11 (19.6)
Class III obesity (BMI ≥ 40 kg/m²)	8 (14.3)
Laboratory values	
AST, U/L	35.6 ± 26.6
ALT, U/L	44.4 ± 45.8
LDL cholesterol, mg/dL	122.5 ± 34.4
Triglycerides, mg/dL	149.6 ± 64.3
APRI score	0.46 ± 0.47
FIB-4 score	1.03 ± 0.87

Data are presented as mean ± standard deviation or n (%).BMI: body mass index; AST: aspartate aminotransferase; ALT: alanine aminotransferase; LDL: low-density lipoprotein; APRI: Aspartate Aminotransferase-to-Platelet Ratio Index; FIB-4: Fibrosis-4 index.

Table 2
Changes in anthropometric, metabolic, and fibrosis parameters during follow-up

Parameter	Baseline (n = 56)	6 Months (n = 56)	12 Months (n = 56)	p-value
BMI, kg/m²	34.8 ± 6.0	31.8 ± 6.2	31.5 ± 6.3	< 0.001
Weight, kg	97.1 ± 17.1	88.5 ± 17.6	87.9 ± 17.8	< 0.001
Weight change from baseline, kg	–	-8.6 ± 5.0	-9.2 ± 6.1	< 0.001
Weight change from baseline, %	–	-9.1 ± 5.2	-9.7 ± 6.3	< 0.001
Percentage of Weight Loss				
< 5%, n (%)	–	14 (25.0)	14 (25.0)	
5–10%, n (%)	–	16 (28.6)	18 (32.1)	
> 10%, n (%)	–	26 (46.4)	24 (42.9)	
LDL Cholesterol, mg/dL	122.5 ± 34.4	–	105.6 ± 22.8	< 0.001*
Triglycerides, mg/dL	149.6 ± 64.3	–	119.9 ± 51.0	< 0.001*
AST, U/L	35.6 ± 26.6	27.2 ± 22.4	22.8 ± 16.1	< 0.001
ALT, U/L	44.4 ± 45.8	29.4 ± 31.2	23.3 ± 15.0	< 0.001
FIB-4 score	1.03 ± 0.87	0.88 ± 0.76	0.77 ± 0.67	< 0.001
FIB-4 change from baseline	–	-0.15 ± 0.33	-0.26 ± 0.44	
APRI score	0.46 ± 0.47	0.32 ± 0.40	0.25 ± 0.29	< 0.001
APRI change from baseline	–	-0.14 ± 0.24	-0.21 ± 0.31	

Data are presented as mean ± standard deviation or n (%).

*Baseline and 12-month comparison.

BMI: body mass index; LDL: low-density lipoprotein; AST: aspartate aminotransferase; ALT: alanine aminotransferase; FIB-4: Fibrosis-4 index; APRI: Aspartate Aminotransferase-to-Platelet Ratio Index.

Discussion

In this retrospective real-world study, we investigated changes in non-invasive liver fibrosis markers during 12 months of treatment with weekly semaglutide or tirzepatide in adults with obesity. The main findings of our study were: (1) significant reductions in body weight and BMI, (2) substantial

improvements in liver enzyme levels, and (3) significant decreases in both APRI and FIB-4 scores over the follow-up period. These findings suggest that incretin-based therapies may provide hepatic benefits in addition to their well-established effects on weight reduction and metabolic control.

Obesity is a major risk factor for MASLD and contributes to the progression of hepatic steatosis, inflammation, and fibrosis [1–5]. Liver fibrosis is considered the strongest predictor of liver-related and all-cause mortality in patients with MASLD, highlighting the importance of early identification and monitoring of fibrosis risk [2, 5]. Although liver biopsy remains the gold standard for fibrosis assessment, non-invasive fibrosis scores such as APRI and FIB-4 have become increasingly important because they are inexpensive, easily obtainable, and suitable for routine clinical practice [6–8].

Weight loss remains the cornerstone of treatment for both obesity and MASLD. Previous studies have demonstrated that sustained weight reduction is associated with improvements in hepatic steatosis, necroinflammation, and fibrosis [2]. In our study, patients experienced a mean weight loss of approximately 9% during follow-up, accompanied by significant reductions in BMI. Nearly half of the study population achieved weight loss greater than 10%, supporting the effectiveness of semaglutide and tirzepatide in real-world obesity management [9–12].

The introduction of incretin-based therapies has transformed the treatment of obesity. Semaglutide, a GLP-1 receptor agonist, and tirzepatide, a dual GIP/GLP-1 receptor agonist, have demonstrated remarkable efficacy in reducing body weight and improving cardiometabolic risk factors [9–12]. Beyond their effects on body weight, growing evidence suggests that these agents may exert direct hepatic benefits through improvements in insulin sensitivity, reduction of hepatic lipid accumulation, attenuation of oxidative stress, and modulation of inflammatory pathways [13–18].

One of the most notable findings of our study was the significant reduction in APRI scores from 0.46 ± 0.47 at baseline to 0.25 ± 0.29 after 12 months of treatment. APRI is a simple and widely used fibrosis marker based on AST and platelet count and has been shown to correlate with fibrosis severity in chronic liver diseases [6]. The nearly 46% reduction observed in our cohort suggests a substantial improvement in fibrosis-related risk. Likewise, FIB-4 scores decreased significantly from 1.03 ± 0.87 to 0.77 ± 0.67, representing an approximate 25% reduction. The simultaneous improvement in both APRI and FIB-4 strengthens the likelihood that these changes reflect meaningful improvements in liver health rather than isolated laboratory fluctuations.

Our findings are consistent with previous reports demonstrating favorable hepatic effects of anti-obesity pharmacotherapy. Semaglutide has been shown to improve steatohepatitis activity and increase the likelihood of NASH resolution [13], while recent studies evaluating tirzepatide have reported significant improvements in metabolic dysfunction-associated steatohepatitis and fibrosis-related outcomes [14]. Furthermore, meta-analyses of GLP-1 receptor agonists have demonstrated beneficial effects on liver fat content and liver enzyme levels [15]. Together, these findings support the hypothesis that incretin-based therapies may contribute to improvements in fibrosis-related parameters.

Interestingly, previous studies evaluating other anti-obesity agents have reported similar findings. Durantas et al. demonstrated significant reductions in APRI scores following orlistat treatment in patients with obesity [20]. However, compared with orlistat, semaglutide and tirzepatide induce substantially greater weight loss and exert broader metabolic effects [9–12, 19]. The significant reductions observed in both APRI and FIB-4 scores in our study suggest that incretin-based therapies may have a particularly favorable impact on non-invasive fibrosis markers.

Several limitations should be acknowledged. First, the retrospective design limits causal inference and may introduce selection bias. Second, this was a single-center study with a relatively small sample size. Third, liver fibrosis was assessed using surrogate indices rather than liver biopsy, transient elastography, or magnetic resonance imaging. Finally, the absence of a control group prevents definitive attribution of the observed changes solely to semaglutide or tirzepatide treatment. Nevertheless, our study reflects routine clinical practice and provides valuable real-world data regarding longitudinal changes in APRI and FIB-4 scores during incretin-based obesity treatment.

Despite these limitations, the study has important strengths. To our knowledge, this is among the first real-world studies evaluating changes in both APRI and FIB-4 scores during one year of treatment with weekly semaglutide or tirzepatide in adults with obesity. The availability of serial measurements at baseline, 6 months, and 12 months allowed assessment of temporal changes in fibrosis-related markers and provided insight into the potential hepatic effects of these therapies.

Conclusions

Treatment with weekly semaglutide or tirzepatide for 12 months was associated with significant weight loss, improvement in liver enzyme levels, and reductions in non-invasive fibrosis markers in adults with obesity. Both APRI and FIB-4 scores demonstrated significant and progressive declines throughout follow-up, suggesting a potential beneficial effect of incretin-based therapies on fibrosis-related liver risk. Larger prospective studies incorporating imaging modalities and histopathological assessment are warranted to confirm these findings and further clarify the long-term hepatic effects of semaglutide and tirzepatide.

Declarations

Competing Interests

The authors declare that they have no competing interests.

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Author Contribution

R.K. conceived and designed the study, collected the data, performed the statistical analyses, interpreted the results, and wrote the first draft of the manuscript. B.E. supervised the study, contributed to study design and data interpretation, critically revised the manuscript for important intellectual content, and provided overall guidance throughout the research process. Both authors read and approved the final manuscript.

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Figures

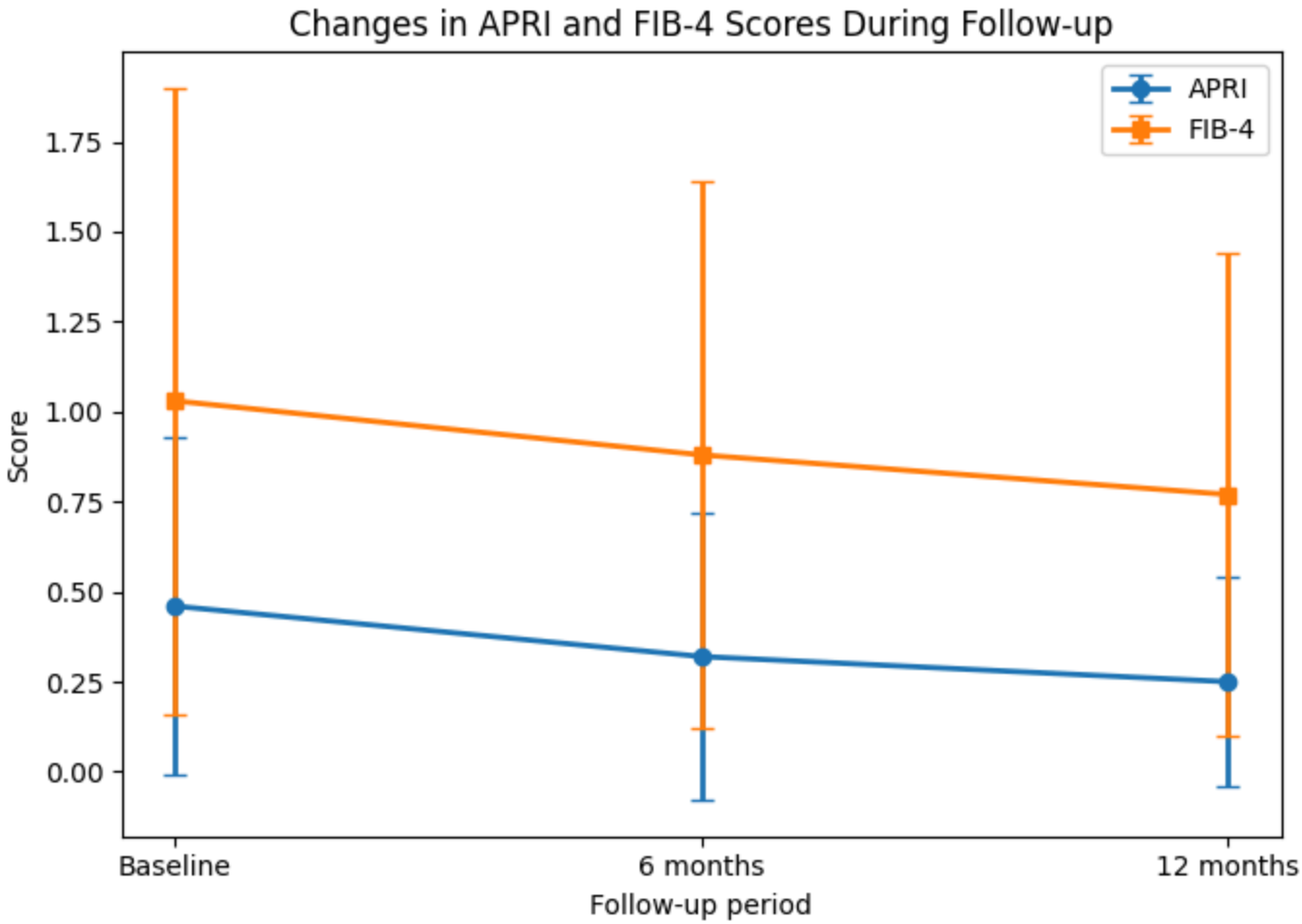


Figure 1

Changes in APRI and FIB-4 scores during 12 months of GLP-1 receptor agonist therapy.