The European Research Journal 2026;12(5):515-522 Research Article

DOI: https://doi.org/10.18621/eurj.1795033 *Internal Medicine*

Investigation of Changes in Liver Fibrosis Scores and Kidney Function in Patients with Diabetic Hepatosteatosis

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Abstract:

Objective: Hepatosteatosis, diabetes, and chronic kidney disease are significant risk factors for mortality and morbidity. The objective of this study was to examine the relationship between non-invasive liver fibrosis scores and kidney function tests in patients with diabetic hepatosteatosis.

Methods: This present study was conducted through a retrospective analysis of two consecutive data sets of 72 diabetic patients aged 18–80 years of both sexes who were investigated for at our hospital between 2018 and 2024. The relationships between hepatosteatosis, diabetes mellitus parameters, kidney function tests, liver function tests, and fibrosis scores (BARD, BAAT, NFS, FIB4, APRI) were examined.

Results: Among the patients included in the study, 54.2% (n=39) were female, and the mean age was 60.99±12.46 years. A moderate negative correlation was found between the initial GFR and both initial FIB4 (P<0.001) and NFS values (P=0.001), while a weak negative correlation was observed between the final GFR and both final FIB4 (P=0.016) and NFS values (P=0.001). A weak negative association was observed between the differences in the initial and final GFR values and both the differences in BARD scores and BARD ratios (P=0.039). Linear regression analysis revealed that a one-unit increase in the BARD ratio led to a 4.34-unit decrease in GFR.

Conclusion: The study revealed a correlation between fibrosis progression, as measured by repeated measurements, and an increased risk of developing new-onset chronic kidney disease. The findings of this study indicated that liver fibrosis in patients with diabetic hepatosteatosis might contribute to the development of chronic kidney disease, thereby underscoring the necessity for enhanced monitoring of kidney function.

Keywords: Hepatosteatosis, Diabetes Mellitus, Liver Fibrosis Scores, Kidney Function

Non-alcoholic fatty liver disease (NAFLD) is identified when hepatic fat accumulation (hepatosteatosis) is demonstrated through histological analysis or imaging techniques, in the absence of heavy alcohol intake, steatogenic drugs, or genetic/metabolic disorders that can result in secondary fat deposition in the liver [1, 2]. Between 1990 and 2006, the worldwide prevalence of NAFLD rose by 50.4%, with reported rates increasing from 25.26% to 38% during 2016–2019 [3]. This steady

Submitted: October 17, 2025 **Accepted:** February 2, 2026 **Published Online:** February 6, 2026

How to cite this article: Canbolat O, Karahisar Şirali S, Bulucu MF. Investigation of Changes in Liver Fibrosis Scores and Kidney Function in Patients with Diabetic Hepatosteatosis. *Eur Res J*. 2026;12(5):515-522. doi: 10.18621/eurj.1795033

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Available Online at https://www.eurj.org.tr

upward trend in NAFLD and hepatic steatosis imposes a growing financial strain on healthcare systems globally [4]. Clinical evidence indicates that steatosis in NAFLD patients can progress to fibrosis and, in certain patients, may eventually result in cirrhosis [5]. Given the morbidity associated with fatty liver disease and the elevated mortality risk linked to cirrhosis, timely detection and management of both the general population and high-risk groups is essential [6, 7].

Although the complications of diabetes affecting the cardiovascular, renal, retinal, and nervous systems are well established [8], the exact nature of its relationship with NAFLD is less well understood. Clinically, this is important since individuals with diabetes face a significantly greater likelihood of developing cirrhosis, with a standardised mortality ratio approximately 2.3 times higher than that of non-diabetic individuals [8, 9]. A strong link between type 2 diabetes and NAFLD has been documented, with liver injury spanning a spectrum from simple steatosis to end-stage cirrhosis.

Chronic kidney disease (CKD) is defined as a persistent reduction in glomerular filtration rate and represents a major global health issue due to its substantial contribution to overall mortality [10, 11]. Cardiovascular disease, metabolic syndrome, and CKD share several overlapping risk factors, including high blood pressure, elevated glucose and triglyceride levels, abdominal obesity, and reduced high-density lipoprotein (HDL) cholesterol [12, 13]. Some studies have suggested that markers of liver fibrosis may be associated with CKD [14, 15], although these findings have not been consistently confirmed in the general population.

The present study aimed to investigate whether non-invasive liver fibrosis (NILF) scores are related to kidney function parameters in patients diagnosed with hepatic steatosis and diabetes mellitus.

METHODS

This study, which comprised retrospectively reviewed two consecutive data sets from 72 patients aged 18–80 years of both sexes who were referred to our hospital between 2018 and 2024, investigated hepatosteatosis, which was detected on abdominal ultrasonography requested due to suspected hepatosteatosis in diabetic patients. The initial parameters were designated as "parameters_1" and the final ones as "parameters_2". The parameters compared during the study included blood urea nitrogen (BUN), creatinine, calculated glomerular filtration rate (GFR), aspartate aminotransferase (AST), alanine aminotransferase (ALT), albumin, total cholesterol, HDL, low-density lipoprotein (LDL), glycated haemoglobin (HbA1c), body mass index (BMI), and liver fibrosis scores including BARD, FIB-4, BAAT, NFS, and APRI.

•**BARD** (**B**MI, **A**ST/**A**LT **R**atio, Diabetes) is scored as [BMI >28 kg/m²] = 1 point, [AST/ALT ratio >0.8] = 2 points, and [diabetes mellitus present] = 1 point. A result for BARD between 0–1 indicates a "low risk", and BARD between 2–4 indicates a "high risk".

•**FIB4** (**Fib**rosis-**4**) is calculated as [Age (years) × AST ÷ Platelet count (10^3/μL) × √ALT]. The result is interpreted as follows: if <1.45" indicates a "low risk", if between 1.45–3.25 indicates an "intermediate risk", and if >3.25 indicates a "high risk".

•**BAAT** (**B**MI, **A**ge, **A**LT, **T**hrombocytes) is scored as [BMI >28 kg/m²] = 1 point, [Age >50 years] = 1 point, and [ALT >(2 × upper limit of reference)] = 1 point, and [Triglycerides >150 mg/dL (or >1.7 mmol/L)] = 1 point.

•**NFS** (**N**AFLD **F**ibrosis **S**core) is calculated as [–1.675 + (0.037 × Age (years)] + [0.094 × BMI (kg/m²)] + [1.13 × Impaired Fasting Glucose or Diabetes (Yes = 1, No = 0)] + [0.99 × AST/ALT ratio] – [0.013 × Platelet count (10^3/μL)] – [0.66 × Albumin (g/dL)]. A result <(–1.455) indicates a fibrosis level between "F0–F2", a result between (–1.455) and 0.675 is interpreted as an "indeterminate level", and a result >0.675 indicates a fibrosis level between "F3–F4"; where F0 = "No fibrosis", F1 = "Mild fibrosis", F2 = "Moderate fibrosis", F3 = "Severe fibrosis", and F4 = "Cirrhosis".

•**APRI** (**A**ST/**P**latelet **R**atio **I**ndex) is calculated as [AST (IU/L) ÷ Upper Limit of Normal for AST (IU/L) ÷ Platelet count (10^3/μL)]. A score ≤0.5 indicates "no fibrosis", a score >1.5 indicates "significant fibrosis", and a score ≥2 suggests "advanced fibrosis"

The initial and final values were compared based on correlations between fibrosis scores and GFR. The relationships between the change in initial and final FR and both the change and ratio of liver fibrosis

scores over the same period were also evaluated.

Ethical approval was obtained from the Non-Interventional Clinical Research Evaluation Ethics Committee of Ufuk University (Ankara, Türkiye) at the meeting numbered 24.06.07.02/06, dated 07.06.2024.

Statistical Analysis

The data were analysed using IBM SPSS Statistics for Windows, Version 22.0 (Armonk, NY: IBM Corp. USA). Categorical variables were expressed as frequency (n) and percentage (%). The normality of the distribution for continuous variables was assessed using skewness and kurtosis tests, and the results were presented as mean, standard deviation, median, minimum, and maximum values. Pearson and Spearman's correlation coefficients were used for correlation analysis. The effects of independent variables on dependent variables were evaluated using linear regression analysis. A P-value <0.05 was considered statistically significant.

RESULTS

The mean age of the patients included in the study was 60.99±12.46 years. Statistically significant differences were observed between initial and final values in BMI (P=0.028), LDL (P=0.003), albumin (P<0.001), triglycerides (P=0.027), total cholesterol (P<0.001), ALT (P=0.002), GFR (P=0.010), and the fibrosis scores FIB-4 (P0.027) and BAAT (P=0.024) (Table 1). No statistically significant differences were found in the comparisons of other parameters (P>0.05).

Regarding the correlations between the initial GFRs (GFR_1) and the initial liver fibrosis scores

TABLE 1. Comparison of the Baseline and Sixth-Month Values in the Study Group

Parameters	Baseline	6th-month	Difference	t, z	P-value
BMI (kg/m^2)	30.63±5.65	29.85+12.64	0.77±2.94	2.23	**0.028**
HbA1c (%)	8±2.18	7.9±2	0.94±1.76	0.38	0.704
Platelet (10^3/μL)	252 ±86.40	249.73±61.14	252±86/249±61	-1.67	0.095
LDL (mg/dL)	122.94±0.46	112.46±41.61	12.53±32.38	3.095	**0.003**
HDL (mg/dL)	49.48±14.71	47.69±14.57	1.33±6.15	1.75	0.084
Cholesterol (mg/dL)	212.3±48.32	191.10±51.47	22.98±39.61	4.64	**0.001**
Triglyceride (mg/dL)	162±122	192.01±157.80	162±122/192±157	-2.21	**0.027**
Albumin (g/dL)	3.79±0.46	4 ±0.52	3.79±0.46/ 4±0.52	-3.41	**<0.001**
ALT* (U/L)	19 (9)	18 (13)	19 (9)/ 18(13)	-3.15	**0.002**
AST* (U/L)	21 (17)	18 (9)	21 (17)/ 18(9)	-1.50	0.133
BUN* (mg/dL)	14 (7)	16 (7)	14 (7)/ 16(7)	-0.91	0.359
Creatinine (mg/dL)*	0.83(0.35)	0.84(0.39)	0.83 (0.35)/ 0.84 (0.39)	-1.51	0.130
GFR (mL/min)	79.98±22.66	76.72±25.15	3.25±10.38	2.66	**0.010**
BARD	3.23±1.01	3.19±0.86	0.04±1.31	0.26	0.789
BAAT)	2.24±0.71	2.04±0.72	0.22±0.79	2.30	**0.024**
NFS	-0.30±1.56	-0.14±1.09	-0.09±0.92	-0.85	0.395
FIB-4*	1.04 (0.75)	1.07 (0.61)	1.04 (0.75)/ 1.07 (0.61)	-2.21	**0.027**
APRI*	0.24 (0.16)	0.21 (0.15)	0.24 (0.16)/ 0.21 (0.15)	-0.75	0.450

Data are shown as mean±standard deviation or median *(interquartile) where appropriate. BMI, body mass index; HbA1c, hemoglobin 1Ac; LDL, low density lipoprotein; HDL, high density lipoprotein; ALT, alanine aminotransferase; AST, aspartate aminotransferase; BUN, blood urea nitrogen; GFR, glomerular filtration rate; BARD, BMI+AST/ALT ratio+diabetes; BAAT, BMI+age+ALT+thrombocytes; NFS, NAFLD fibrosis score, FIB-4, fibrosis-4; APRI, AST/platelet ratio index. Statistically significant P-values are shown in bold.

TABLE 2. Correlation Between Baseline and Sixth-Month GFR and Fibrosis Scores

	BARD_1		BAAT_1		NFS_1		FIB4_1*		APRI_1*	
	r	P-value	r	P-value	r	P-value	r	P-value	r	P-value
GFR_1	0.145	0.225	-0.062	0.610	-0.569	**0.001**	-0.405	**<0.001**	-0.054	0.632
	BARD_2		**BAAT_2**		**NFS_2**		**FİB4_2***		**APRI_2***	
GFR_2	-0.182	0.125	-0.205	0.097	-0.397	**0.001**	-0.283	**0.016**	0.307	**0.009**
Difference	**BARD**		**BAAT**		**NFS***		**FIB4***		**APRI***	
GFR	-0.244	**0.039**	-0.228	0.063	0.197	0.11	0.107	0.37	0.035	0.769

GFR, glomerular filtration rate; BARD, BMI+AST/ALT ratio+diabetes; BAAT, BMI+age+ALT+thrombocytes; NFS, NAFLD fibrosis score, FIB-4, fibrosis-4; APRI, AST/platelet ratio index.

*Spearman's correlation. Statistically significant P-values are shown in bold.

(BARD_1, BAAT_1, NFS_1, FIB-4_1, and APRI_1) moderate negative correlations were observed between GFR_1 and FIB-4_1 (r = –0.405, P<0.001), and between GFR_1 and NFS_1 (r = –0.569, P=0.001) (Table 2). Despite these significant correlations between GFR_1 and NFS_1/FIB-4_1, regression analyses did not reveal any statistically significant predictive effect of fibrosis scores on GFR_1 (P>0.05).

As for the correlations between the final GFRs (GFR_2) and the final fibrosis scores (FIB-4_2, APRI_2, BAAT_2, BARD_2, and NFS_2) a weak negative correlation was found between GFR_2 and FIB-4_2 (r = –0.283, P=0.016), and NFS_2 (r = –0.397, P=0.001), while a weak positive correlation was observed between GFR_2 and APRI_2 (r = 0.307, P=0.009) (Table 2). Nevertheless, the regression analysis, again, showed no statistically significant effect of fibrosis scores on GFR_2 (P>0.05).

When the correlations between the change in GFR (difference between the final and initial values) and changes in FIB-4, NFS, APRI, BARD, and BAAT scores were examined, a weak negative correlation was found only between the GFR difference and the change in BARD score (r = –0.244, P=0.039) (Table 2).

Finally, in the analysis of correlations between the change in GFR and the proportional changes in fibrosis scores, a weak negative correlation was observed between the GFR difference and the BARD ratio (r = –0.329, P=0.005). In linear regression analysis, where the GFR difference was set as the dependent variable, among the independent variables representing liver fibrosis scores, the BARD ratio was found to be a significant predictor. The model indicated that the BARD ratio accounted for 10.9% of the variance in GFR change. Specifically, a one-unit increase in the BARD ratio was associated with a 4.34-unit decrease in GFR. Additionally, BAAT ratio (P=0.01, 95% CI-20.569_-2.895), age (P=0.011, 95% CI 6.84_.299), albumin (P=0.04, 95% CI .242_10.625) showed a significant effect on the GFR difference in the linear regression analysis (Table 3). Other laboratory parameters and fibrosis scores did not have a statistically significant effect on the GFR difference.

TABLE 3. Linear Regression Analysis of Parameters Affecting GFR Difference

GFR difference	B	Beta	t	P-value	F	R^2	%95% CI	
							Lower	Upper
BARD ratio	-4.34	-0.329	-2.92	**0.005**	8.525	0.109	-7.304	-0.1375
Age	-0.249	-0.299	2.617	**0.011**	6.84	0.089	6.84	.299
Albumin	5.434	0.244	2.088	**0.040**	4.36	0.059	0.242	0.10.625
BAAT ratio	-11.732	-0.312	-2.651	**0.010**	0.703	0.098	-20.569	-2.895

GFR, glomerular filtration rate; BARD, BMI+AST/ALT ratio+diabetes; BAAT, BMI+age+ALT+thrombocytes; CI, confidence interval. Statistically significant P-values are shown in bold.

DISCUSSION

This study aimed to investigate the relationship between changes in renal function and five different non-invasive liver fibrosis scores in patients with diabetic hepatosteatosis. The relationship between NAFLD and CKD is well known. Statistically significant differences between initial and final values were found in BMI, LDL, albumin, triglycerides, total cholesterol, ALT, GFR, and the fibrosis scores FIB-4 and BAAT. Significant correlations were identified between GFR_1 and both FIB-4_1 and NFS_1, and between GFR_2 and both FIB-4_2 and NFS_2; and, finally, the regression analysis showed that a one-unit increase in the BARD ratio resulted in a 4.34-unit decrease in GFR difference. It is expected that scores such as FIB-4 and NFS, which include markers of inflammation and cellular stress such as age, platelets, albumin and AST/ALT show a significant relationship with GFR. However, in our study: HbA1c values do not change significantly between the initial and final measurements, the decrease in GFR is especially associated with the change in fibrosis scores, suggesting that it may affect kidney function. All these findings collectively suggested that renal function might be influenced by the severity of the liver fibrosis.

In a retrospective cohort study, Kuma *et al*. [16] examined the possible link between elevated FIB-4 scores and the occurrence of CKD in metabolically healthy men. While a high FIB-4 value (≥1.30) did not emerge as an independent risk factor for CKD (Odds Ratio [OR]=1.57; 95% Confidence Interval [CI]=0.97–2.56), subgroup analyses revealed notable associations. Specifically, higher FIB-4 scores were significantly related to CKD among participants who were non-obese participants (OR=1.92; CI=1.09–3.40), non-hypertensive (OR=2.15; CI=1.16–3.95), and non-smokers (OR=1.88; CI=1.09–3.23). In these groups, elevated FIB-4 values also showed a strong correlation with reduced estimated GFR, as indicated by multiple linear regression. Based on these observations, the researchers concluded that higher FIB-4 scores could be associated with CKD development over a five-year period in metabolically healthy individuals [16]. In this study, healthy male individuals were evaluated. In our study, the average age of both genders was 60.99 and BMI was 30.63, and it was only conducted in individuals with diabetic hepatosteatosis. The follow-up period was limited to 6 months. The fact that we could't find a significant relationship between FIB4 score and GFR difference in the regression analyses was considered due to our study was conducted in only individuals with diabetic hepatosteatosis. The negative correlations identified in our study between GFR_1 and FIB-4_1, as well as GFR_2 and FIB-4_2, appeared consistent with the subgroup outcomes reported by Kuma *et al*.

Hydes *et al*. [17] assessed how NAFLD and liver fibrosis affect adverse clinical outcomes and mortality among patients with CKD. At baseline, 56.2% of the CKD population had NAFLD, 3% had FIB-4 >2.67, and 7.7% showed NAFLD with NFS ≥0.676. In univariate analyses, NAFLD was linked to increased risks of cardiovascular events (Hazard Ratio [HR]=1.49; 95% CI=1.38–1.60), all-cause mortality (HR=1.22; 95% CI=1.14–1.31), and end-stage renal disease (HR=1.26; 95% CI=1.02–1.54). Multivariate analysis confirmed NAFLD as an independent predictor of cardiovascular outcomes (HR=1.20; 95% CI=1.11–1.30; P<0.001). Elevated NFS and FIB-4 levels were also strongly associated with cardiovascular events (HR=2.42; 95% CI=2.09–2.80 and HR=1.64; 95% CI=1.30–2.08, respectively) and overall mortality (HR=2.82; 95% CI=2.48–3.21 and HR=1.82; 95% CI=1.47–2.24, respectively) in univariate analyses. In addition, higher NFS values were related to a significantly greater risk of end-stage renal disease (HR=5.15; 95% CI=3.52–7.52) [17]. These outcomes were in line with the negative correlations we observed between NFS_1 and GFR_1, and between NFS_2 and GFR_2. However, unlike Hydes *et al*. [17], our study also included participants with normal GFR values alongside those with CKD.

In another study, Schleicher *et al*. [18] investigated the association between FIB-4 levels and the risk of renal failure in a general population sample. During a 10-year follow-up, renal failure occurred in 9.2% of those with FIB-4 <1.3 and in 10.6% of individuals with FIB-4 ≥1.3 (p=0.007). Having a FIB-4 score ≥1.3 was linked to a slightly increased risk of renal failure (HR=1.08; 95% CI=1.02–1.14; P=0.009). Moreover, a dose–response pattern was observed, with the highest risk in individuals with FIB-4 ≥2.67 (HR=1.34; 95% CI=1.22–1.46; P=0.001). These findings were consistent with the inverse associations demonstrated in our analysis between GFR_1 and FIB-4_1, as well

as between GFR_2 and FIB-4_2 [18].

Xiong *et al*. [9] investigated the relationship between CKD and several liver fibrosis indices, including FIB-4, BARD, and BAAT. They demonstrated that patients with CKD had significantly higher scores on all three indices compared to those without CKD. In multivariate logistic regression, each fibrosis marker showed an independent association with CKD (FIB-4: OR=6.71, CI=4.45–10.13; BAAT: OR=1.88, CI=1.29–2.75; BARD: OR=1.72, CI=1.28–2.31) [9]. The negative correlations we detected between GFR_1 and FIB-4_1, GFR_2 and FIB-4_2, as well as between GFR change and both BARD score change and BARD ratio in our study, were in agreed with their findings. The significant relationship found in the linear regression analysis between BARD and BAAT ratios and GFR was consistent with the study of Xiong *et al*. [9]. The fact that the BARD ratio predicts GFR change is an important finding, especially since it reflects the effect of fibrosis progression over time on renal function.

In another study, Seko *et al*. [19] sought to determine risk factors for CKD progression in patients with biopsy-confirmed NAFLD. Their multivariate analysis identified male sex (OR=5.46), advanced age, and a FIB-4 score ≥1.30 as predictors of CKD. Among 154 individuals with baseline GFR ≥60 mL/min, 30 experienced CKD stage progression, and 15 developed CKD over three years. The authors highlighted the importance of kidney-centred preventive strategies, particularly in patients with both diabetes and NAFLD [19]. Consistent with these results, our observations of negative correlations between GFR_1 and FIB-4_1, GFR_2 and FIB-4_2, GFR_1 and NFS_1, and GFR_2 and NFS_2 suggested that fibrosis indices might serve as early markers of CKD risk in diabetic NAFLD populations.

Wijarnpreecha *et al*. [20] assessed the utility of non-invasive fibrosis markers in diagnosing CKD among 4,142 NAFLD patients, of whom 200 (4.8%) had CKD. ROC analysis revealed AUC (areas under the curve) values of 0.77 for FIB-4, 0.75 for NFS, 0.62 for BARD, and 0.51 for APRI. Compared with low-risk patients, those at high risk of advanced fibrosis had a markedly increased likelihood of CKD, with significant associations for NFS (adjusted OR=4.92, CI=2.96–8.15) and FIB-4 (adjusted OR=2.27, CI=1.05–4.52). Both FIB-4 and NFS independently predicted CKD in NAFLD patients, with FIB-4 considered the most reliable predictor overall [20]. The inverse association between FIB-4 and GFR in our study group, together with the significant link between GFR difference and BARD ratio, supported these ROC-based findings.

Supriyadi *et al*. [21] conducted a meta-analysis to examine the role of non-invasive fibrosis markers in predicting CKD. Their pooled results demonstrated that elevated FIB-4 scores were associated with higher CKD prevalence (OR=2.51, CI=1.87–3.37, P<0.00001, I^2=96%). Additional regression analyses indicated that this relationship was significantly affected by hypertension (P=0.0241), NAFLD (p=0.0029), and BMI (p=0.0025). Similarly, high NFS (OR=2.49, CI=1.89–3.30, P<0.00001, I^2=96%) and high APRI (OR=1.40, CI=1.14–1.72, P=0.001, I^2=26%) were linked with CKD prevalence. The authors recommended routine use of these markers in both NAFLD patients and the general population to improve risk stratification and facilitate earlier CKD detection [21]. In line with these conclusions, our study also revealed significant associations between GFR and both FIB-4 and NFS. Furthermore, in Mima's investigation [22], which incorporated pre- and post-biopsy analyses, a significant negative correlation between FIB-4 and GFR was reported in nephrosclerosis patients (R^2=0.4362, P=0.04). Our results were consistent with these findings.

Strengths and Limitations

The strength of our study was that there was no statistically significant difference in HbA1c levels between baseline and final assessments, implying that HbA1c may not play a role in the effect of fibrosis scores on GFR.

The retrospective nature of the study, conducted at a single centre with a limited number of patients, poses a limitation in terms of the generalizability of its findings. Validation of the results through multicentre studies with larger cohorts would be beneficial.

CONCLUSION

Given the global rise in CKD and the increasing

prevalence of high-risk conditions such as diabetes, identifying potential risk factors for CKD development and establishing targeted intervention strategies is of critical importance. This study suggested that progression in liver fibrosis, as determined through repeated measurements in time, might contribute to an increased risk of CKD development. This suggests that the BARD score is easier to capture the change in metabolic load as it includes the presence of diabetes, obesity, hepatic enzyme rates. Being a specific analysis for the diabetic hepatosteatosis population differs from other studies based on the general population or the NAFLD population. Two temporal comparisons were made with the same individual. Therefore, the effect of fibrosis progression on kidney function can be expressed more clearly. Furthermore, the absence of a statistically significant difference in HbA1c levels between initial and final assessments implied that HbA1c might not play a role in the impact of fibrosis scores on GFR.

Ethics Approval and Consent to Participate

This study was approved by the Ufuk University Non-Interventional Clinical Research Evaluation Ethics Committee (Decision No: 24.06.07.02/06; date: 07.06.2024. All procedures were conducted in accordance with the ethical standards of the institutional and national research committee and with the 1964 Helsinki Declaration and its later amendments. Informed consent was not required in this study because this is a retrospective study.

Data Availability

All data generated or analyzed during this study are included in this published article. The data that support the findings of this study are available on request from the corresponding author, upon reasonable request.

Authors' Contribution

Study Conception: OC, MFB; Study Design: OC, MFB; Supervision: MFB; Funding: N/A; Materials: N/A; Data Collection and/or Processing: OC; Statistical Analysis and/or Data Interpretation: OC, SKŞ, MFB; Literature Review: OC, SKŞ; Manuscript Preparation: OC; and Critical Review: SKŞ, MFB.

Conflict of Interest

The author(s) disclosed no conflict of interest during the preparation or publication of this manuscript.

Financing

The author(s) disclosed that they did not receive any grant during the conduction or writing of this study.

Acknowledgments

The study was presented as a poster at the 41st National Nephrology Congress & 34th National Nephrology Nursing Congress, held on December 4–8, 2024 in Antalya, Türkiye.

Generative Artificial Intelligence Statement

The author(s) declare that no artificial intelligence-based tools or applications were used during the preparation process of this manuscript. The all content of the study was produced by the author(s) in accordance with scientific research methods and academic ethical principles.

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