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PREVALENCE AND SEVERITY OF NON-ALCOHOLIC FATTY LIVER DISEASE IN PATIENTS WITH CHRONIC KIDNEY DISEASE.

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Abstract

Background: Non-alcoholic fatty liver disease (NAFLD) and chronic kidney disease (CKD) share overlapping metabolic risk factors. ***Aim:*** This study investigates the prevalence and severity of NAFLD across CKD stages using non-invasive tools. ***Methods:*** Prospective observational study of 100 CKD patients. NAFLD was diagnosed by ultrasonography; liver fibrosis was staged using FibroScan and fibrosis scores (NFS, FIB-4, APRI). Statistical analysis used Chi-square test, Student's t-test, and Mann-Whitney U test; significance set at $p<0.05$. ***Results:*** NAFLD prevalence was 68%. Advanced liver fibrosis was found in 48% by FibroScan, 59% by FIB-4, 52% by APRI, and 55% by NFS. USG-NAFLD grade correlated significantly with CKD stage ($p<0.001$). FibroScan-detected advanced fibrosis rose from 25% in Stage 1 to 82.05% in Stage 5 ($p<0.001$). AST and AST: ALT ratio increased significantly with CKD stage ($p<0.01$ and $p<0.001$ respectively). Renal markers — eGFR, creatinine, urea, and uric acid — all showed highly significant stage-wise trends ($p<0.001$). Lipid parameters showed no significant stage-wise variation ($p>0.05$). ***Conclusions:*** NAFLD is highly prevalent in CKD patients, with fibrosis severity escalating with advancing renal impairment. FibroScan combined with NFS and FIB-4 provides effective non-invasive risk stratification and should be incorporated into routine CKD management.

Keywords: NAFLD, CKD, transient elastography, FibroScan, FIB-4, APRI, NAFLD fibrosis score, liver fibrosis, p-value, metabolic syndrome.

INTRODUCTION

Chronic kidney disease (CKD) is a major global health burden affecting 10.4–13.4% of the world population, with CKD-related mortality rising by 31.7% in recent decades. In India, CKD prevalence is approximately 0.78%, driven by the rising burden of type 2 diabetes mellitus (T2DM), hypertension, obesity, and metabolic syndrome — all well-established risk factors for renal disease progression.1,2

Non-alcoholic fatty liver disease (NAFLD) represents the hepatic manifestation of metabolic dysfunction, encompassing a spectrum from simple steatosis to non-alcoholic steatohepatitis (NASH), advanced fibrosis, cirrhosis, and hepatocellular carcinoma. Globally, NAFLD affects 20–30% of the general population. Indian studies report prevalence ranging from 9% to 32%, with higher rates in metabolically at-risk cohorts.3.

A bidirectional relationship between NAFLD and CKD is increasingly recognized. Insulin resistance — central to both conditions — promotes hepatic steatosis and simultaneously induces glomerulosclerosis and podocyte injury. Systemic inflammatory mediators (IL-6, TNF-α, TGF-β) generated in NAFLD drive renal interstitial fibrosis and proteinuria, while advanced CKD amplifies oxidative stress and renin-angiotensin system dysregulation, perpetuating hepatocellular injury. NAFLD has been shown to independently predict incident CKD (hazard ratio 1.49; 95% CI: 1.1–2.2; $p<0.01$), and coexistent CKD markedly increases cardiovascular and all-cause mortality in NAFLD patients.4,5

Non-invasive assessment of liver fibrosis is critical in CKD patients, in whom liver biopsy carries heightened haemorrhagic risk. Transient elastography (FibroScan) and serum-based fibrosis scores — NAFLD Fibrosis Score (NFS), Fibrosis-4 Index (FIB-4), and AST-to-Platelet Ratio Index (APRI) — provide validated, practical alternatives for risk stratification.6-8 This study aimed to determine NAFLD prevalence and fibrosis severity across CKD stages using these tools, generating local epidemiological evidence to support early screening and multidisciplinary care.

Aim: to determine NAFLD prevalence and fibrosis severity across CKD stages.

MATERIALS AND METHODS

Study Design

Prospective observational hospital-based study conducted at the Department of General Medicine, Mahatma Gandhi Medical College and Hospital, from January 2024 to August 2025. Ethics Committee approval was obtained and written informed consent was secured from all participants.

Participants

One hundred CKD patients aged >18 years were enrolled by consecutive sampling. Exclusion criteria included: refusal of consent, pregnancy, hepatitis B or C infection, alcohol intake >20 g/day (males) or >10 g/day (females), drug-induced steatosis, prior gastrointestinal bypass surgery, FibroScan failure due to obesity or ascites, and contraindications to elastography (jaundice, right heart failure, ALT >5× ULN, AST >3× ULN).

Clinical and Radiological Assessment

Abdominal ultrasonography (USG) was performed by an experienced radiologist to diagnose and grade NAFLD (Grades 1–3) based on liver echogenicity, liver-kidney contrast, vascular wall brightness, and deep beam attenuation. Patients with USG-confirmed fatty liver underwent FibroScan for liver stiffness measurement (LSM): ≤7 kPa = no significant fibrosis; 7.0–12.9 kPa = mild-to-moderate fibrosis (F1–F2); ≥13 kPa = advanced fibrosis.

Non-Invasive Fibrosis Scores

APRI, FIB-4, and NFS were calculated using standard formulae. Cut-offs for advanced fibrosis: APRI >1.5; FIB-4 >3.25; NFS >0.675.9,10

Statistical Analysis

Data were analyzed using SPSS v29.0. Descriptive statistics expressed as mean ± SD and proportions. Chi-square test (with/without Yates correction) was used for categorical variables; Student's t-test and Mann-Whitney U test for continuous variables. One-way ANOVA (F-statistic) was used for multi-group comparisons. A p-value <0.05 was considered statistically significant.

RESULTS

Demographic and CKD Stage Distribution

The mean age was 46.06 ± 12.08 years; males comprised 54% and females 46%. CKD Stage 5 was most common (39%), followed by Stage 4 (28%), Stage 3 (19%), Stage 1 (8%), and Stage 2 (6%). Age showed a highly significant association with CKD stage ($p<0.001$), with older patients predominating in advanced stages. BMI did not vary significantly across stages ($F=1.82$, $p=0.13$).

Table 1: Demographic parameters and statistical significance.

Variable	Finding	p-value	Significance
Age vs CKD Stage	Older age → advanced CKD	**< 0.001**	Highly Significant
Gender (M:F)	54% : 46%	—	Descriptive
BMI across CKD stages	24.43–26.99 kg/m²	0.13	NS

Liver Function Tests Across CKD Stages

AST levels rose significantly from 50.13 ± 20.89 U/L (Stage 1) to 79.1 ± 22.53 U/L (Stage 5), with $F\approx4.85$ ($p<0.01$). The AST:ALT ratio increased from 0.85 ± 0.42 in Stage 1 to 1.51 ± 0.71 in Stage 5 ($F\approx5.92$, $p<0.001$), indicating worsening hepatic dysfunction with CKD progression. ALT ($p=0.79$), ALP ($p=0.64$), and serum bilirubin ($p=0.31$) showed no significant stage-wise differences.

Table 2: Liver function tests compared across CKD stages. NS = not significant.

Parameter	Stage 1 (Mean±SD)	Stage 5 (Mean±SD)	F-statistic	p-value
AST (U/L)	50.13 ± 20.89	79.1 ± 22.53	F ≈ 4.85	< 0.01
ALT (U/L)	61.13 ± 9.21	59.03 ± 18.02	—	0.79 (NS)
AST:ALT Ratio	0.85 ± 0.42	1.51 ± 0.71	F ≈ 5.92	**< 0.001**
ALP (U/L)	8.39 ± 1.89	9.58 ± 2.16	—	0.64 (NS)
Serum Bilirubin (mg/dL)	0.38 ± 0.35	0.54 ± 0.28	—	0.31 (NS)

Renal Function Parameters Across CKD Stages

All renal parameters showed highly significant deterioration with advancing CKD stage ($p<0.001$ for all). Mean eGFR declined sharply from 93.5 ± 1.58 mL/min/1.73m² (Stage 1) to 7.41 ± 3.36 mL/min/1.73m² (Stage 5), F≈950. Blood urea rose from 121.63 ± 77.11 mg/dL (Stage 1) to 199.67 ± 72.5 mg/dL (Stage 5), F≈7.85. Serum creatinine rose from 3.75 ± 2.6 mg/dL (Stage 1) to 8.04 ± 3.11 mg/dL (Stage 5), F≈16.2. Uric acid rose from 5.7 ± 2.58 mg/dL (Stage 1) to 9.27 ± 3.37 mg/dL (Stage 5), F≈6.45.

Table 3: Renal function parameters across CKD stages. All associations highly significant.

Renal Parameter	Stage 1 (Mean±SD)	Stage 5 (Mean±SD)	F-statistic	p-value
eGFR (mL/min/1.73m²)	93.5 ± 1.58	7.41 ± 3.36	F ≈ 950	**< 0.001**
Blood Urea (mg/dL)	121.63 ± 77.11	199.67 ± 72.5	F ≈ 7.85	**< 0.001**
Serum Creatinine (mg/dL)	3.75 ± 2.60	8.04 ± 3.11	F ≈ 16.2	**< 0.001**
Uric Acid (mg/dL)	5.7 ± 2.58	9.27 ± 3.37	F ≈ 6.45	**< 0.001**

Lipid Profile Across CKD Stages

No lipid parameter showed statistically significant variation across CKD stages. Serum triglycerides ranged from 124.63 ± 42.05 mg/dL (Stage 1) to 146.51 ± 39.15 mg/dL (Stage 5), p=0.54. Total cholesterol, LDL, HDL, and VLDL similarly showed no significant stage-wise differences (p=0.11, 0.21, 0.63, and 0.25 respectively).

Table 4: Lipid profile across CKD stages. NS = not significant (p>0.05 for all parameters).

Lipid Parameter	Range Across Stages	p-value	Significance
Triglycerides (mg/dL)	124.63 – 146.51	0.54	NS
Total Cholesterol (mg/dL)	169.13 – 192.29	0.11	NS
LDL (mg/dL)	121.46 – 145.89	0.21	NS
HDL (mg/dL)	48.3 – 54.75	0.63	NS
VLDL (mg/dL)	Variable	0.25	NS

Non-Invasive Fibrosis Scores Across CKD Stages

All three fibrosis scores demonstrated significant associations with CKD stage. Mean APRI values rose from 0.67 ± 0.69 (Stage 2) to 2.21 ± 0.79 (Stage 5), p<0.01. Mean FIB-4 rose from 2.25 ± 1.54 (Stage 3) to 4.64 ± 1.05 (Stage 5), p=0.04. Mean NFS rose from −1.25 ± 1.17 (Stage 2) to 1.17 ± 0.92 (Stage 5), p=0.04.

Table 5: Non-invasive fibrosis scores across selected CKD stages.

Fibrosis Score	Stage 2 Mean±SD	Stage 3 Mean±SD	Stage 5 Mean±SD	p-value
APRI	0.67 ± 0.69	0.72 ± 0.75	2.21 ± 0.79	< 0.01
FIB-4	2.50 ± 1.30	2.25 ± 1.54	4.64 ± 1.05	0.04
NFS	−1.25 ± 1.17	−0.89 ± 1.77	1.17 ± 0.92	0.04

Prevalence of NAFLD and USG Grade vs CKD Stage

NAFLD was detected in 68 of 100 patients (68%). A highly significant association was found between USG grade of NAFLD and CKD stage (p<0.001). Grade 2 NAFLD predominated in Stage 5 (73.07% of USG Grade 2 patients) and Grade 3 NAFLD in Stages 5 and 4 (59.09% and 31.8% respectively). Patients without fatty liver changes were predominantly in Stages 1–3.

FibroScan Findings vs CKD Stage

Advanced fibrosis was identified in 48 patients (48%), F1–F2 fibrosis in 24 (24%), and no fibrosis in 28 (32%). A clear progression in fibrosis severity with worsening CKD was observed (p<0.001). Among patients without fibrosis, 57.89% were in Stage 3 and none in Stage 5. Advanced fibrosis was present in 82.05% of Stage 5 and 39.39% of Stage 4 patients.

Table 6: Transient elastography findings by CKD stage (p<0.001 for all).

FibroScan Finding	n (%)	Stage 1	Stage 3	Stage 4	Stage 5	p-value
No Fibrosis	28 (28%)	37.5%	57.9%	39.4%	0%	< **0.001**
F1–F2 Fibrosis	24 (24%)	37.5%	31.6%	21.4%	18.0%	< **0.001**
Advanced Fibrosis	48 (48%)	25.0%	10.5%	39.4%	82.1%	< **0.001**

Summary of All p-values

Table 7 consolidates all statistical associations reported in this study for rapid reference.

Parameter / Association	Statistical Test	p-value	Interpretation
Age group vs CKD Stage	Chi-square	< **0.001**	Highly Significant
BMI vs CKD Stage	ANOVA (F=1.82)	0.13	Not Significant
AST vs CKD Stage	ANOVA (F≈4.85)	< 0.01	Significant
ALT vs CKD Stage	ANOVA	0.79	Not Significant
AST:ALT Ratio vs CKD Stage	ANOVA (F≈5.92)	< **0.001**	Highly Significant
ALP vs CKD Stage	ANOVA	0.64	Not Significant
Serum Bilirubin vs CKD Stage	ANOVA	0.31	Not Significant
eGFR vs CKD Stage	ANOVA (F≈950)	< **0.001**	Highly Significant
Blood Urea vs CKD Stage	ANOVA (F≈7.85)	< **0.001**	Highly Significant
Serum Creatinine vs CKD Stage	ANOVA (F≈16.2)	< **0.001**	Highly Significant
Uric Acid vs CKD Stage	ANOVA (F≈6.45)	< **0.001**	Highly Significant
Triglycerides vs CKD Stage	ANOVA	0.54	Not Significant
Total Cholesterol vs CKD Stage	ANOVA	0.11	Not Significant
LDL vs CKD Stage	ANOVA	0.21	Not Significant
HDL vs CKD Stage	ANOVA	0.63	Not Significant
VLDL vs CKD Stage	ANOVA	0.25	Not Significant
APRI vs CKD Stage	ANOVA	< 0.01	Significant
FIB-4 vs CKD Stage	ANOVA	0.04	Significant
NFS vs CKD Stage	ANOVA	0.04	Significant
USG Grade (NAFLD) vs CKD Stage	Chi-square	< **0.001**	Highly Significant
FibroScan Fibrosis vs CKD Stage	Chi-square	< **0.001**	Highly Significant

Table 7: Consolidated p-value summary for all outcome associations. Red = significant; grey = not significant.

DISCUSSION

This study found a NAFLD prevalence of 68% among CKD patients — consistent with Dahiya et al.11 (71.4%), higher than Hydes et al. (56.2%) and Shehzad et al. (54%), and substantially higher than Adrian et al.12 (7.9% in non-diabetic CKD), underscoring the impact of metabolic comorbidities on hepatic involvement in CKD. The highly significant association between USG-NAFLD grade and CKD stage (p<0.001) confirms that hepatic steatosis severity escalates with worsening renal function. FibroScan advanced fibrosis prevalence rose from 25% in Stage 1 to 82.05% in Stage 5 (p<0.001), demonstrating a robust dose-response relationship between renal and hepatic fibrosis.

These findings closely parallel Dahiya et al.11, who reported advanced fibrosis in 12.5% of Stage 3, 36.6% of Stage 4, and 71.7% of Stage 5 CKD patients (p=0.04). The mechanistic basis lies in shared fibrogenic pathways: insulin resistance drives both hepatic stellate cell activation (via TGF-β/SMAD signalling) and renal mesangial cell proliferation; angiotensin II and hyperuricaemia amplify both hepatic and renal fibrosis simultaneously.

Among fibrosis scores, FIB-4 showed the highest sensitivity for advanced fibrosis (59%), followed by NFS (55%) and APRI (52%), compared to FibroScan's 48%. Both FIB-4 and NFS reached statistical significance at p=0.04 against CKD stage, while APRI achieved p<0.01. The relatively modest concordance between score-based and FibroScan-based classification in CKD reflects a known limitation: elevated AST from uraemia, anaemia, and rhabdomyolysis, alongside low platelet counts from erythropoietin therapy or bone marrow suppression, can artificially inflate FIB-4 and APRI. Dahiya et al. demonstrated APRI's poor performance in grading fibrosis severity versus FibroScan in CKD (p=0.317), supporting the use of FibroScan as the primary modality with scores as adjunctive screening tools.

Liver function test analysis revealed a significant rise in AST across CKD stages (p<0.01; F≈4.85) and a highly significant increase in the AST:ALT ratio (p<0.001; F≈5.92), while ALT, ALP, and bilirubin remained stable (p>0.05 for all). An AST:ALT ratio progressively exceeding 1.0 in advanced CKD stages suggests hepatocellular injury, hepatic fibrosis, or mitochondrial dysfunction, consistent with the FibroScan findings of advanced fibrosis predominating in Stages 4–5.13.

Renal parameters showed the most striking associations: all four markers — eGFR, blood urea, creatinine, and uric acid — were highly significantly associated with CKD stage ($p<0.001$ for each). The near-linear inverse relationship between eGFR and advancing stage (Stage 1: 93.5 mL/min/1.73m² → Stage 5: 7.41 mL/min/1.73m²; F≈950) validates CKD staging in this cohort and provides a robust comparator for hepatic findings. Hyperuricaemia in advanced CKD (mean 9.27 mg/dL in Stage 5) independently contributes to renal tubular injury and may also promote hepatic steatosis through xanthine oxidase-mediated oxidative stress.

Lipid parameters showed no significant stage-wise variation (all $p>0.05$). This finding, corroborated by Angulo et al.9 and the CRIC study, likely reflects the combined effects of statin therapy, dietary restriction, reverse epidemiological effects of chronic inflammation on cholesterol synthesis, and variable nutritional status across stages. Poor glycaemic control (mean HbA1c 7.65%; FBS 171.32 mg/dL) and hypoalbuminaemia (mean 3.24 g/dL) were prevalent across the cohort, reflecting the metabolic burden and chronic protein-energy wasting characterizing advanced CKD-NAFLD overlap. Previous studies by Targher et al.7 ($p<0.01$) and Adams et al.14 confirm that higher HbA1c independently predicts both NAFLD progression and CKD worsening.

Limitations

This study is limited by its single-centre design, cross-sectional nature, and a sample size of 100. Liver biopsy was not performed, and non-invasive scores may overestimate fibrosis in CKD due to uraemia-related confounders. Long-term outcomes data were not available.

CONCLUSION

NAFLD is highly prevalent (68%) among CKD patients, and hepatic fibrosis severity increases significantly with advancing renal impairment — a relationship confirmed across multiple assessment modalities, all reaching statistical significance ($p<0.001$). Advanced fibrosis was identified in 48% by FibroScan, 59% by FIB-4, 52% by APRI, and 55% by NFS.

Non-invasive fibrosis assessment tools, particularly FibroScan combined with FIB-4 or NFS, provide effective and safe risk stratification in this population. All renal biochemical markers, AST, and the AST:ALT ratio demonstrated significant stage-wise associations ($p<0.01$ to $p<0.001$), while lipid parameters did not. Routine NAFLD screening in CKD patients is clinically warranted, and a multidisciplinary hepato-nephrology approach is recommended to reduce hepatic, renal, and cardiovascular morbidity.

DECLARATIONS

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Conflict of Interest: None declared.

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