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Frequency of Non-Alcoholic Fatty Liver Disease in Patient with Chronic Kidney Disease

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ABSTRACT

Background: Non-alcoholic fatty liver disease (NAFLD) is increasingly recognized as a major comorbidity in patients with chronic kidney disease (CKD). Both conditions share common risk factors, including obesity, diabetes, hypertension, and dyslipidemia. Early detection of NAFLD in CKD patients is crucial for preventing progression of both hepatic and renal dysfunction. **Objectives:** To determine the frequency of non-alcoholic fatty liver disease among patients with chronic kidney disease and to evaluate its association with clinical parameters and biochemical abnormalities. **Study Design:** A cross-sectional study. **Place and Duration of study:** Department of General Medicine, Bolan Medical Complex Hospital, Quetta, from December 2023 to June 2024. **Methods:** A cross-sectional study was conducted among 142 patients with confirmed CKD attending the nephrology outpatient department. NAFLD was diagnosed using abdominal ultrasonography after excluding alcohol intake and other hepatic causes. Relevant biochemical and clinical data were collected, including body mass index, fasting glucose, and lipid profile. Statistical analysis was performed using SPSS version 24.0, with $p < 0.05$ considered significant. **Results:** Among 142 CKD patients (mean age = 52.4 ± 11.2 years), 82 (57.7%) were males and 60 (42.3%) females. NAFLD was detected in 81 patients (57.0%). Patients with NAFLD had significantly higher mean body mass index (28.6 ± 3.9 kg/m^2 vs 25.2 ± 3.6 kg/m^2, $p = 0.001$) and serum triglycerides (186.4 ± 42.5 mg/dL vs 148.3 ± 38.1 mg/dL, $p = 0.002$). Elevated fasting glucose and alanine transaminase levels were also more prevalent among NAFLD patients ($p < 0.05$). The frequency increased with higher CKD stages. **Conclusion:** Non-alcoholic fatty liver disease was highly prevalent among patients with chronic kidney disease, particularly those with obesity and metabolic abnormalities. The significant association between NAFLD and advanced CKD underscores the need for routine hepatic evaluation and metabolic control in renal patients. Early diagnosis and management may help reduce disease progression and improve overall outcomes in this high-risk population.

INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) represents a spectrum of hepatic conditions ranging from simple steatosis to non-alcoholic steatohepatitis (NASH), fibrosis, and cirrhosis, occurring in individuals with little or no alcohol consumption. It is now considered the most prevalent chronic liver disorder worldwide, affecting approximately 25–30% of the global population, with even higher rates among those with metabolic syndrome, obesity, and diabetes mellitus [1]. The coexistence of NAFLD with chronic kidney disease (CKD) has recently garnered significant clinical attention due to shared pathophysiological mechanisms involving insulin resistance, systemic inflammation, and oxidative stress [2]. CKD is characterized by progressive loss of renal function over time, classified into stages based on the estimated glomerular filtration rate (eGFR). Its prevalence is rising globally due to the increasing burden of diabetes and hypertension [3]. Recent studies have revealed that patients with CKD are more prone to hepatic steatosis and other metabolic disturbances, making NAFLD a frequent comorbidity [4]. The two diseases not only share common risk factors but may also mutually exacerbate disease progression. Insulin resistance promotes lipid accumulation in hepatocytes, while reduced renal clearance of toxins and cytokines further aggravates hepatic inflammation and fibrosis [5]. Emerging evidence indicates that NAFLD is an independent predictor of CKD onset and progression, even after adjusting for traditional metabolic factors [6]. Conversely, impaired renal function may worsen hepatic outcomes by altering lipid metabolism and promoting oxidative stress [7]. Several

studies using imaging and non-invasive indices, such as the fatty liver index and hepatic steatosis index, have demonstrated a significantly higher prevalence of NAFLD among CKD patients compared to the general population [8]. In Pakistan and other South Asian countries, the dual epidemic of metabolic syndrome and renal dysfunction presents a growing public-health challenge. The limited availability of local data regarding the prevalence of NAFLD in CKD patients further necessitates region-specific study to understand its frequency and risk factors [9]. Early detection and management of NAFLD in CKD patients can potentially slow disease progression, reduce cardiovascular risk, and improve overall survival. Therefore, this study aimed to determine the frequency of NAFLD among patients with CKD using ultrasonographic assessment and to analyze its association with metabolic and biochemical parameters. Identifying the extent of this comorbidity in our population can guide clinicians toward integrated care approaches for managing both hepatic and renal dysfunctions effectively.

METHODOLOGY

This descriptive cross-sectional study was conducted in general medicine outpatients' department of a Bolan Medical Complex Hospital, Quetta, over six months, from December 2023 to June 2024. A total of 142 patients with clinically and biochemically confirmed chronic kidney disease were included. NAFLD was diagnosed based on ultrasonographic evidence of hepatic steatosis after excluding patients with alcohol consumption or other known hepatic disorders. Demographic information, body mass index (BMI), fasting blood glucose, lipid profile, and liver enzyme levels were recorded. CKD staging was determined using estimated glomerular filtration rate (eGFR) calculated by the MDRD equation. Data were entered and analyzed using SPSS version 24.0. Continuous variables were expressed as mean ± standard deviation, and categorical variables as frequencies and percentages.

Inclusion Criteria

Patients aged 18 years or older diagnosed with chronic kidney disease (stages 1–5) confirmed by eGFR values and attending the nephrology outpatient department during the study period.

Exclusion Criteria

Patients with a history of alcohol consumption, viral hepatitis (HBV, HCV), autoimmune liver disease, drug-induced hepatotoxicity, or known secondary causes of hepatic steatosis were excluded from the study.

Ethical Approval Statement

The study was approved by the Institutional Review Board of Bolan Medical Complex Hospital Quetta (IRB #1124/DOS/MTI/HMC). Written informed consent was obtained from all participants prior to data collection. Confidentiality and anonymity were maintained in accordance with the Declaration of Helsinki ethical principles for biomedical study.

Data Collection

Data were collected using a pre-designed proforma, including demographic details, clinical history, laboratory investigations, and ultrasonographic findings. All ultrasonographic examinations were performed by a qualified radiologist blinded to patients' laboratory results. The diagnosis of NAFLD was based on characteristic hepatic echogenicity relative to renal cortex brightness.

Statistical Analysis

Statistical analysis was carried out using SPSS version 24.0 (IBM Corp., Armonk, NY). Quantitative variables were expressed as mean ± SD, while qualitative variables were presented as frequencies and percentages. Associations between NAFLD and clinical variables were assessed using chi-square and independent t-tests, with $p < 0.05$ considered statistically significant.

RESULTS

A total of 142 CKD patients were included, comprising 82 (57.7%) males and 60 (42.3%) females, with a mean age of 52.4 ± 11.2 years. The prevalence of NAFLD among CKD patients was 57.0% (n = 81). Among those with NAFLD, 68% were overweight or obese, and 61% had diabetes mellitus. Mean BMI was significantly higher in the NAFLD group (28.6 ± 3.9 kg/m^2) than in non-NAFLD patients (25.2 ± 3.6 kg/m^2, p = 0.001). Similarly, mean serum triglycerides (186.4 ± 42.5 mg/dL) and fasting glucose (119.8 ± 24.1 mg/dL) were significantly elevated (p = 0.002 and p = 0.003, respectively). Alanine transaminase was raised in 49% of NAFLD patients compared with 18% of non-NAFLD patients (p = 0.004). The frequency of NAFLD was higher in advanced CKD stages, particularly stages 4–5 (p = 0.01). These findings suggest a strong correlation between NAFLD and metabolic abnormalities in CKD patients.

Table 1

Baseline Demographic and Clinical Characteristics of Patients (n = 142)

Variable	Total (n = 142)	NAFLD Present (n = 81)	NAFLD Absent (n = 61)	*p*-Value
Mean age (years)	52.4 ± 11.2	53.6 ± 10.8	51.0 ± 11.6	0.241
Male gender, n (%)	82 (57.7)	50 (61.7)	32 (52.5)	0.291
BMI (kg/m^2)	27.0 ± 4.2	28.6 ± 3.9	25.2 ± 3.6	0.001
Diabetes mellitus, n (%)	84 (59.1)	49 (60.5)	35 (57.4)	0.715
Hypertension, n (%)	103 (72.5)	60 (74.0)	43 (70.5)	0.654
Dyslipidemia, n (%)	76 (53.5)	46 (56.8)	30 (49.1)	0.370

Table 2

Biochemical Parameters in CKD Patients with and without NAFLD

Parameter	NAFLD Present (n = 81)	NAFLD Absent (n = 61)	*p*-Value
Fasting glucose (mg/dL)	119.8 ± 24.1	103.6 ± 21.7	0.003
Triglycerides (mg/dL)	186.4 ± 42.5	148.3 ± 38.1	0.002
Total cholesterol (mg/dL)	202.7 ± 35.4	181.6 ± 32.8	0.012
HDL-C (mg/dL)	38.2 ± 8.6	43.9 ± 9.2	0.010
ALT (U/L)	45.6 ± 16.3	33.8 ± 14.1	0.004
Serum creatinine (mg/dL)	3.5 ± 1.1	3.1 ± 1.3	0.082

Table 3
Distribution of NAFLD According to CKD Stage

CKD Stage	Total n (%)	NAFLD Present n (%)	NAFLD Absent n (%)	*p*-Value
Stage 1 (eGFR ≥ 90)	14 (9.9)	5 (35.7)	9 (64.3)	0.045
Stage 2 (eGFR 60–89)	27 (19.0)	13 (48.1)	14 (51.9)	0.038
Stage 3 (eGFR 30–59)	49 (34.5)	30 (61.2)	19 (38.8)	0.014
Stage 4 (eGFR 15–29)	33 (23.2)	21 (63.6)	12 (36.4)	0.010
Stage 5 (eGFR < 15)	19 (13.4)	12 (63.2)	7 (36.8)	0.008

Table 4
Logistic Regression Analysis for Predictors of NAFLD in CKD Patients

Variable	Odds Ratio (OR)	95% Confidence Interval (CI)	*p*-Value
BMI (> 27 kg/m^2)	2.84	1.42 – 5.67	0.003
Diabetes Mellitus	1.76	0.89 – 3.49	0.092
Triglycerides (> 170 mg/dL)	2.45	1.18 – 5.06	0.016
ALT (> 40 U/L)	2.32	1.09 – 4.95	0.027
eGFR (< 45 mL/min/1.73 m^2)	1.98	1.03 – 3.81	0.041

DISCUSSION

The present study demonstrated a high frequency (57%) of non-alcoholic fatty liver disease (NAFLD) among patients with chronic kidney disease (CKD), indicating a strong association between hepatic steatosis and renal dysfunction. This finding supports emerging evidence that NAFLD is not merely a hepatic manifestation of metabolic syndrome but also a potential contributor to systemic and renal disease progression. The observed prevalence in our cohort is consistent with earlier international and regional studies, which reported NAFLD in 45–60% of CKD patients depending on diagnostic modality and population characteristics [10,11]. The high burden of NAFLD in CKD can be attributed to overlapping pathophysiologic mechanisms such as insulin resistance, dyslipidemia, oxidative stress, and chronic inflammation [12]. Insulin resistance promotes hepatic triglyceride accumulation through enhanced lipogenesis and decreased fatty acid oxidation. Furthermore, CKD leads to the retention of uremic toxins and inflammatory cytokines, which aggravate hepatic inflammation and fibrosis [13]. Studies have shown that pro-inflammatory markers like TNF-α and interleukin-6 are elevated in both conditions, suggesting a shared inflammatory pathway [14]. Our study also observed significant associations between NAFLD and higher body mass index (BMI), triglyceride, fasting glucose, and alanine transaminase (ALT) levels. These findings are consistent with the report by Targhee et al., who noted that metabolic abnormalities including obesity, hypertriglyceridemia, and insulin resistance are strong predictors of NAFLD in renal patients [15]. Similarly, Musso et al. reported that individuals with NAFLD have a two-fold higher risk of developing CKD compared to those without fatty liver, independent of other risk factors [16]. A recent meta-analysis further confirmed that NAFLD severity correlates positively with the risk and progression of CKD.In our study, the prevalence of NAFLD increased significantly in advanced CKD stages (4–5). This pattern agrees with the findings of Mantovani et al., who demonstrated a graded relationship between lower eGFR and higher hepatic steatosis scores [17]. The association may be due to the accumulation of lipotoxic metabolites and oxidative injury secondary to renal impairment. Additionally, CKD-related metabolic acidosis and anemia may accelerate hepatic injury and fibrosis [18]. Local data from South Asian populations also highlight similar trends. Ahmed et al. reported a NAFLD prevalence of 54% among Pakistani CKD patients, emphasizing obesity and diabetes as key determinants [24]. Another study from India showed that 63% of CKD patients had hepatic steatosis, reinforcing the regional significance of metabolic risk factors [19]. Our findings further strengthen the evidence that NAFLD and CKD are interconnected conditions sharing a bidirectional relationship, particularly in populations with high metabolic disease burden. Beyond epidemiological association, recent mechanistic study has provided molecular insights into the NAFLD-CKD link. Hepatic steatosis promotes systemic inflammation and the release of pro-fibrotic mediators such as fibroblast growth factor-21 (FGF-21) and fetuin-A, which may induce glomerular injury [20]. Conversely, CKD leads to altered gut microbiota and increased intestinal permeability, facilitating endotoxemia that contributes to hepatic inflammation. These complex interactions suggest that management strategies should target both hepatic and renal metabolic dysfunction rather than treating them as isolated entities.

Given the substantial overlap between NAFLD and CKD, routine hepatic screening in CKD patients using ultrasonography or non-invasive biomarkers should be considered. Early identification may allow for timely lifestyle interventions such as weight reduction, dietary modification, and improved glycemic control, which have been shown to improve both hepatic steatosis and renal function.

CONCLUSION

Non-alcoholic fatty liver disease was highly prevalent among patients with chronic kidney disease and showed strong associations with obesity, dyslipidemia, and hyperglycemia. Early screening and integrated management of metabolic risk factors in CKD patients may prevent disease progression, improve hepatic and renal outcomes, and enhance overall patient quality of life.

Limitations

The study was limited by its single-center design, relatively small sample size, and cross-sectional nature, which precludes causal inference. Ultrasonography, though practical, may underestimate mild steatosis compared to histology. Future studies incorporating longitudinal follow-up and liver biopsies are required to establish temporal and mechanistic relationships between NAFLD and CKD progression.

Future Findings

Future study should explore genetic, molecular, and inflammatory mechanisms linking NAFLD and CKD, emphasizing multi-omics approaches for early biomarker discovery. Interventional trials evaluating lifestyle

modification, insulin sensitizers, and Reno protective agents could clarify whether targeted therapies improve both hepatic and renal outcomes, reducing morbidity and mortality in high-risk metabolic populations.

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