Frequency of Metabolic Associated Fatty Liver Disease among Chronic Kidney Disease Patients in Upper Egypt

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Abstract:

Background: Chronic kidney disease (CKD) and metabolic-associated fatty liver disease (MAFLD) are increasing chronic illnesses that contribute to a variety of major cardio-metabolic risk factors and pathogenic processes.

Objectives: To estimate the frequency of MAFLD and its risk factors among CKD Patients in Upper Egypt.

Patients and methods: a case-control study entangled 200 individuals with CKD and 60 person without as a control. All participants underwent a thorough history, physical examination, and laboratory testing which incorporate complete blood count, assessment of neutrophil-lymphocyte ratio (NLR), liver enzymes (ALT, AST), kidney function, estimated glomerular filtration rate (eGFR), and lipid profile. Pelvic-abdominal ultrasound and fibroscan were executed.

Results: We found a significantly increased proportion of MAFLD, HbA1c and NLR values in the cases group when compared with the control group, where a high prevalence of steatosis and liver stiffness among CKD patients. Multivariate logistic regression analysis demonstrated that high BMI, elevated LDL, elevated liver enzymes (AST & ALT) were predictive risk factors for the existence of MAFLD in the CKD patients.

Conclusion: There is a statistically significant correlation between CKD and MAFLD in the form of increased prevalence of MAFLD in CKD patients.

Keywords: Metabolic associated fatty liver disease ; chronic kidney disease; estimated glomerular filtration

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Introduction

Chronic kidney disease (CKD) and non-alcoholic fatty liver disease (NAFLD) are both escalating chronic conditions that exemplify a group of diseases ranging from relatively mild disease to severe exhausting disorders with end-stage organ damage, necessitating either chronic dialysis or organ transplantation to preserve life **(Byrne and Targher, 2020)**.

Obesity-related mechanisms of disease, such as oxidative stress, lipotoxicity, increased pro-inflammatory cytokine, and renin-angiotensin-aldosterone system (RAAS) axis activation, may cause both liver and kidney injury, which demonstrate the obvious relationship between NAFLD and CKD **(Kiapidou et al., 2020).**

The presence of metabolic syndrome (MetS) and its components, such as hypertension (HTN), obesity, type 2 diabetes mellitus (DM), and dyslipidemia, expanding the peril of progressing NAFLD. NAFLD has been linked to a number of extrahepatic symptoms, including cardiovascular disease and sleep apnea. NAFLD has recently been renamed MAFLD to emphasise the substantial link between overweight/obesity, type 2 diabetes, and the MetS **(Umbro et al., 2021)**.

The goal of our research was to determine the frequency of MAFLD, and its risk variables in Patients with Chronic Kidney Disease in Upper Egypt at the Outpatient Clinic of Tropical Medicine and Gastroenterology department This study took place in the period from June 2021 to December 2021.

Patients and methods

The study enrolled two hundred Egyptian patients with chronic kidney disease (CKD) and sixty healthy age and sex-matched volunteers (subjects without CKD) as a control group.

So, two groups of participants were formed:

- Group A: included 200 CKD patients.
- Group B: included 60 completely healthy subjects.

Inclusion criteria included: Patients aged 18 and 60 years old proved to have CKD.

Exclusion criteria included

Patients with ESRD on HD, patients with chronic viral hepatitis (B and C), patients consuming alcohol more than 20g/day

Subjects with renal transplantation, patients with major comorbidities or concomitant malignancies, Pregnant female and non-compliant patients.

All subjects involved in the current study were informed about the nature and details of the current work and written agreement was obtained for each them. The study was approved by the local Ethics Committee, Qena Faculty of Medicine, and South Valley University.

All patients underwent the following:

The following were performed on all participants

I. History and Clinical Assessment: -

1- History of accompanying comorbid conditions such as DM, HTN, and cardiac disease. History of drug intake.

2- Full clinical examination: for the CKD and chronic liver diseases indicators existence.

- Anthropometric measurements indices: weight, height, BMI calculation, arm and waist circumference (AC) & (WC). Adiposity indices was estimated:

BMI=weight (kg)/height^2 (m^2)
WHtR = WC (cm)/ height (cm)
conicity index (CI)=
0.109^{-1}WC (m) [Weight (kg)/ Height (m)] $^{-1/2}$

a body shape index (ABSI) = WC(m)/BMI$^{2/3}$(kg/m) × height $^{1/2}$

triponderal mass index (TMI) = Weight (kg)/ Height^3 (m).

II. Laboratory Investigations:

Blood sampling was performed in the morning, with at least 12 hours of post-absorptive fasting.

All participants had 5mm of blood drawn and divided into 2 samples; the initial (3 ml) was collected in a plain vacationer tube, the second (2ml) was collected into an EDTA tube used for CBC and HbA1c assay

1. Fasting blood glucose (FBG), 2 hours postprandial blood glucose (2HPPBG).
2. Lipid profile [total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C); high-density lipoprotein cholesterol (HDL-C)] and triglycerides (TG).
3. Calculation of Low-density lipoprotein (LDL)-cholesterol according to the formula of Friedewald et al. (1972), LDL-cholesterol = TC - HDL – VLDL (triglycerides ÷ 5).
4. Estimation of serum creatinine was done using COBAS 501 chemistry auto analyzer (Roche Diagnostics, USA).
5. Complete blood picture by Erma Automated Blood Count Machine (Tokyo, Japan). And the calculation of the neutrophil-lymphocyte ratio was executed.
6. HbA1c assay by Sysmex XT-1800i automated hematology analyzer (Sysmex, Japan).
7. Albumin to creatinine ratio (ACR): urine samples were collected for measuring creatinine and albumin by an Auto analyzer (Beckman Instruments, Fullerton, California, USA) then ACR was calculated.
8. Calculation of the estimated glomerular filtration rate using the CKD-EPI equation.

III. Imaging: All subjects were submitted to screening with the following procedures:

1- Abdominal Ultrasonography: Assessment of the liver size in both midline and mid-clavicular lines, the surface of the liver, and echogenicity.

2- Transient elastography (FibroScan 502 Echosens, Paris, France):

Hepatic stiffness evaluated by skilled operators using the convential (M probe). The procedure was carried out on the liver through the spaces in between the ribs, on its right lobe. Examined person directed to lie supine with his right arm abducted and it is better to came fasting before the examination. The success rate was 100 percent, and there were ten successful shots. Better-quality criteria of fibroScan' (10 valid measurements, IQR/M 30%) were used to compute the ratio of the inter-quartile range (IQR) of liver stiffness to the median (IQR/M). The usual liver stiffness value for healthy people is around 5.5 kPa, and the range of probable hepatic stiffness values determined with transient elastography is between 2.5 to 75.0 kPa.

Statistical analysis

The data were analyzed using the Statistical Software for Social Sciences (SPSS) version 26.0. The normality of the quantitative variables was tested with the Shapiro-Wilk-test. Quantitative data were shown as mean ± standard deviation (M ±SD) was compared by Student's t-test. The qualitative data were shown as frequency and percentages (%) and. The Chi-square test was used for the comparison of non-parametric data. Multiple linear regression was performed to identify predictors of MAFLD in the CKD group. The variables that were statistically significant in the univariate analysis were included in the multiple regression models. A (two-tailed) P-value < 0.05 was considered significant.

Results

Compared to the control group, CKD cases have:

Significantly increased percentage of DM and HTN (P < 0.001), **Table (1).**

Significantly increased BMI (P= 0.003) and TMI (P= 0.017), **Table (2).**

Significantly increased RDW (P < 0.001) and NLR (P = 0.022), but significantly decreased Hb and RBCs levels (P < 0.001), **Table (3).**

Significantly increased (P < 0.001) HbA1c, serum TC, LDL-c, TG, n-HDL, n-HDL /HDL, TG/HDL, ALT, and significantly decreased HDL-c and albumin. **Table (4).**

We observed a high prevalence of steatosis and liver stiffness among CKD patients when compared with healthy subjects (P = 0.001). **Table (5,6).**

Multivariate logistic regression analysis, demonstrated that the following factors were predictive for MAFLD in the CKD group: BMI, LDL, AST, and ALT. **Table (7).**

Table 1. Demographic data for both studied groups.

Variables		Cases (N = 200)	Control (N = 60)	P-value
Age (years) Mean ±SD		54.1 ±12.9	51.6± 11.8	0.170
Sex	Male	105 (52.5 %)	26(43.3%)	0.213
	Female	95 (47.5%)	34(56.7%)	
DM	NO	128 (64%)	58(96.7%)	**< 0.001**
	YES	72 (36%)	2(3.3%)	
HTN	NO	140 (70%)	56(93.3%)	**< 0.001**
	YES	60 (30%)	4(6.7%)	

Bold: significant; HS: p-value < 0.001 is considered highly significant. NS: p-value > 0.05 is considered non-significant. Independent sample T test.

DM: diabetes mellitus; HTN: hypertension.

.Table 2. Body mass building for both studied groups.

Mean ±SD	**Cases (N = 200)**	**Control (N = 60)**	**P-value**
Height	164.4±7.8	163.7±7.2	0.516
Weight	75.9±10.8	69.5±11.0	**< 0.001**
BMI	28.0±3.7	26.6±4.5	**0.003**
WC	129.3±22.5	124.2±26.6	0.063
AC	30.2±4.5	30.8±5.2	0.701
WHTR	0.79±0.14	0.77±0.17	0.197
ABSI	0.18±0.03	0.17±0.03	0.328
CL	1.73±0.25	1.70±0.26	0.391
TMI	17.6±4.9	16.3±2.9	**0.017**

Bold: HS: p-value < 0.001 is considered highly significant significant; S: p-value < 0.05 is considered significant. NS: p-value > 0.05 is considered non-significant Mann Whitney U tes. BMI: body mass index; WC: Waist circumference; AC: arm circumference; BRI: body roundness index, ABSI: body shape index; CI: conicity index, TMI: triponderal mass index.

Table 3. Mean value of hematological indices for both studied groups.

Mean ±SD	Cases (N = 200)	Control (N = 60)	P-value
HB	9.6±7.3	14.2±1.6	**< 0.001**
RBCs	3.4±0.7	5.2±0.6	**< 0.001**
MCV	82.9±10.5	84.5±2.6	0.729
RDW	44.6±14.3	40.2±3.3	**< 0.001**
PLTs	311.4±89.4	288.4±109.0	0.145
WBCs	9.2±3.9	7.9±1.8	0.118
Neut. Abs	7.0±3.8	5.7±2.0	0.100
Lymph abs	1.5±0.9	1.7±1.0	0.083
NLR	7.1±6.5	4.8±3.3	**0.022 S**

Bold: significant; HS: p-value < 0.001 is considered highly significant. S: p-value < 0.05 is considered significant. NS: p-value > 0.05 is considered non-significant Mann Whitney U test. HB:hemoglobin;RBCS:red blood cells;RDW;NLR:neutrophils lymphocytes ratio.

Table 4. Metabolic assessment for both studied groups.

Mean ±SD	Cases (N = 200)	Control (N = 60)	P-value
FBG	119.3±48.9	118.5±36.3	0.516
HbA1C	5.7±1.6	4.8±0.7	**< 0.001**
T-CHOL	197.8±47.5	154.4±36.8	**< 0.001**
LDL-C	104.2±24.9	82.3±34.0	**< 0.001**
TG	188.7±84.9	117.7±99.2	**< 0.001**
HDL-C	43.0±12.6	79.0±32.6	**< 0.001**
VLDL-C	32.2±10.3	53.1±45.8	0.636
n-HDL	145.8±88.6	38.7±110.6	**< 0.001**
LDL/VLDL	3.7±1.7	3.6±2.9	0.263
n-HDL/HDL	1.5±0.4	0.9±0.5	**< 0.001**
T-CHOL/HDL	139.5±45.4	144.2±98.3	0.097
TG/HDL	4.8±2.8	2.1±3.6	**< 0.001**
Albumin	3.10±0.69	4.10± 0.62	**< 0.001**
ALT	25.61±8.08	19.97±10.33	**< 0.001**

Bold: significant; HS: p-value < 0.001 is considered highly significant. NS: p-value > 0.05 is considered non-significant. Mann Whitney U test.

Table 5. Hepatic fibrosis measurement for both studied groups.

Mean ±SD	Cases (N = 200)	Control (N = 60)	P-value
APRI	0.22±0.12	0.24±0.19	0.992
LSM	6.42±3.62	4.17 ±0.89	**< 0.001**
CAP	255.5±70	201.7±37.9	**< 0.001**

Bold: significant; HS: p-value < 0.001 is considered highly significant. S: p-value < 0.05 is considered. NS: p-value > 0.05 is considered non-significant; Mann Whitney U test. LSM: liver stiffness measurement; CAP: controlled attenuation parameter

Table 6. Liver U/S findings for both studied groups.

No (%)		Cases (N = 200)	Control (N = 60)	P-value
Liver size	Normal	147 (73.5%)	56 (93.3%)	**0.001**
	Enlarged	53	4 (6.7%)	
Fatty liver	Absent	144 (72%)	56 (93.3%)	**0.001**
	Present	56 (28%)	4 (6.7%)	

Bold: significant; HS: p-value < 0.001 is considered highly significant. NS: p-value > 0.05 is considered non-significant. independent sample t-test. Chi-square test

Table 7. Multivariate logistic regression analysis for factors of MAFLD in CKD group

Variables	B	SE	p-value	Odds	95% CL	
BMI	0.04	0.017	**0.029**	1.01	0.98	1.09
LDL	- 0.014	0.007	**0.041**	0.98	0.97	0.99
ALT	3.3	1.27	**0.009**	27.6	2.2	334.1
AST	0.039	0.018	**0.032**	1.04	1.0	1.07
LSM	0.81	0.12	**< 0.001**	2.26	1.77	2.8
CAP	0.013	0.003	**< 0.001**	1.01	1.0	1.01

Bold: significant; B: Regression coefficient, SE: Standard error, CL: Confidence interval. BMI: body mass index; LSM: liver stiffness measurement; CAP: controlled attenuation parameter

Discussion

With a worldwide prevalence of 25% and a huge clinical and economic impact, NAFLD is the most known cause of chronic liver disease (**Younossi et al., 2016**).

Results of the present study revealed that obesity affects renal function and is a risk factor for CKD. When compared to the control group, the case group had a higher BMI. This was in line with the findings of **Chen et al. (2013)**, who managed a research in China with 264 individuals diagnosed with T2DM and renal biopsy-confirmed DN and found that obesity was associated with considerably increased proteinuria. **Abougalambou et al. (2016)** conducted an observational prospective longitudinal follow-up study with 1077 T2DM patients and discovered that an elevated BMI was substantially connected to DN progression.

Results of the current study demonstrate a statistically significant relation (P < 0.05) between CKD and MAFLD. We found a statistically significant increased percentage of increased liver echogenicity in the cases group when compared to control group (P=0.001). Also, we demonstrate an increased prevalence of steatosis and liver stiffness between CKD patients in comparison to healthy individuals. This was in the agreement with **Levin et al. (2013)** conducted a large meta-analysis comprising about 64,000 volunteers in 20 cross-sectional studies and 13 longitudinal investigations and found that NAFLD was incorporated with a 2-fold increase in CKD prevalence (OR, 2.12; 95 percent CI, 1.69 to 2.66) and incidence (HR, 1.79; 95 percent CI, 1.65 to 1.95). NASH was linked to a higher prevalence and incidence of CKD than simple hepatic steatosis in this meta-analysis, and advanced fibrosis was linked to a higher prevalence (OR, 5.20; 95 percent CI, 3.14 to 8.61) and incidence (HR, 3.29; 95 percent CI, 2.30 to 4.71) of CKD than non-advanced fibrosis.Also, **Li et al. (2020)** found a link between the severity of NASH histology,

the advanced form of NAFLD, and hepatic fibrosis and the grade of kidney injury.
Furthermore, an Italian study of 570 fair skin people with NAFLD diagnosed by ultasonography .subject with high and intermediate possibility of developing liver fibrosis had unfavourable risk factors in the form of: higher values of waist circumference, insulin resistance, high sensitivity c-reactive protin, fibrinogen, uric acid, and lower insulin-like growth factor-1 level. So; patients with high risk for developing liver fibrosis had a 5.1-fold increased risk of developing CKD compared with low-risk patients, while intermediate-risk subjects had a 3-fold increased risk of developing liver fibrosis and had 3 times increased risk of developing CKD compared to low-risk patients **(Sesti et al., 2014).**

NAFLD, according to **Jang et al. (2018)**, is an independent risk factor for the development of CKD. Patients with NAFLD (perhaps linked to a significant extensive liver fibrosis score) and those with a decreased eGFR, whether or not coupled with proteinuria, had a higher risk of CKD development.They revealed a 40% elevation in the long-term risk of incident CKD linked to the severity of hepatic fibrosis; they also found that the risk of CKD in NAFLD individuals remained significant even after controlling for age, gender, high BMI, elevated blood pressure, type 2 diabetes mellitus, within normal eGFR, or drugs.
In a comprehensive study and meta-analysis, **Mantovani et al. (2018)** demonstrated the same results (including 9 observational cohort studies incorporating nearly 96,500 Asian participates , over a median follow-up duration of 5.2 years). They discovered a 40% elevation in the long-term risk of incident CKD linked to the severity of hepatic fibrosis; they also discovered that even after controlling for age, gender, high BMI, elevated blood pressure, type 2 diabetes mellitus, within normal eGFR, or drugs, the risk of CKD in NAFLD patients was still significant.

In addition, after adjusting for similar risk variables, a broad prospective cohort study of 8329 Korean males not having type 2 diabetes mellitus, elevated blood pressure, or renal function at baseline revealed that persons with NAFLD had a considerably increased chance of CKD development (Zhang et al., 2020). Furthermore, **Armstrong et al. (2014)** showed that yearly eGFR and microalbuminuria screening of NAFLD patients for CKD could detect recent kidney affection among persons having NAFLD.

However, **Targher et al. (2010)** observed that individuals with NASH proved via biopsy had a greater prevalence of CKD and albuminuria than age-, gender-, and obesity-matched controls, and that the histology of hepatic fibrosis stage was correlated to lowering eGFR, regardless of age, gender, measures of adiposity, elevated blood pressure, triglyceride levels in plasma, or HOMA-estimated insulin resistance. Furthermore, in the Valpolicella Heart Diabetes Study, which included 1,760 persons with type 2 diabetes mellitus who had normal renal function , NAFLD found on ultrasound in association with a 50 percent higher risk of incident CKD (adjusted hazard ratio 1.49; 95 percent CI 1.1– 2.2) over a 6.5-year follow-up duration, regardless of age, gender, measures of adiposity, blood pressure, smoking, diabetes duration, hemoglobin A1c, lipid profile, baseline eGFR, microalbuminuria, and the use of medications either blood glucose lowering agents, antihyperlipidemic, antihypertensive, or antiplatelet medications **(Targher et al., 2008).**

In a meta-analysis of 33 observational studies, the relationship between NAFLD and the risk of prevalent and incident CKD was investigated (20 cross-sectional and 13 longitudinal).

According to a 47 meta-analysis of data from 20 cross-sectional studies, NAFLD was connected to a 2-fold increased prevalence of CKD (random-

effects odds ratio 2.12, 95 percent confidence interval 1.69– 2.66). (including about 30,000 people). NAFLD was connected to an approximately 80% increased risk of developing CKD (random-effects hazard ratio [HR] 1.79; 95 percent confidence interval [CI] 1.65– 1.95), according to a meta-analysis of data from 13 longitudinal studies (covering about roughly 28,500 person). In a subgroup analysis of individual patient data from 5 small studies (incorporating a total of 430 NAFLD people proved through biopsy), the authors found that progressive fibrosis of the liver was associated with a higher prevalence (random-effects odds ratio 5.20; 95 percent CI 3.14– 8.61) and incidence (random-effects HR 3.29; 95 percent CI 2.30– 4.71) of CKD than either non-progressive fibrosis or simple steatosis. Even when pre-existing T2DM and other CKD risk factors such as age, ethnicity, obesity, and smoking history were taken into consideration, the development and progression of NAFLD was connected to a higher prevalence and incidence of CKD in all prior investigations **(Musso et al., 2014)**.

A systematic review and meta-analysis that involved about 9 observational cohort studies with aggregate data on 96,500 middle-aged participants (34.1 percent with NAFLD) of predominantly Asian descent and 5,000 new cases of incident CKD over a median follow-up duration of 5.2 years produced similar results. NAFLD was found to be liked to a 40% elevation in the long-term risk of incident CKD (random-effects HR 1.37; 95 percent CI 1.20– 1.53; I2 = 33.5 percent), a risk that was found to elevate with NAFLD severity, as evaluated by the fibrosis score of NAFLD or other non-invasive markers of progressive fibrosis, and was still significant in studies where common risk factors and potential confounders were controlled for (i.e. age, gender, obesity , hypertension, history of smoking, diabetes, baseline eGFR and certain drugs administration) **(Mantovani et al., 2018)**.

Moreover, **Vilar-Gomez et al. (2017)** discovered that histologic resolution of NASH and regression in the stage of liver fibrosis were both linked to elevated eGFR.In a recent study, **Önnerhag et al. (2019)** looked at the total mortality risk in people with biopsy-proven NAFLD and found a stronger link between CKD and NAFLD .

When comparing Hb of the cases group(9.6± 7.3) to Hb of the control group(14.2±1.6). , we found statistically significant (P <0.001)lower Hb, RBCs, and higher RDW in the cases group .

This finding was in line with that of **Nalado et al. (2019),** who discovered that 43.18 percent of CKD patients had anaemia. And they discovered a strong link between CKD stages and anaemia (P< 0.001) , as well as a positive relationship between Hb levels and eGFR (r =0.334, P 0.0001).

When comparing the cases group (25.6 8.08) to the control group (19.97 10.33), we detected a substantial (P < 0.001) rise in ALT in the cases group (25.6 8.08) compared to the control group (19.97 10.33). This was in line with Adiga and **Malawadi's (2018)** findings, which looked at the renal and liver function of 68 T2DM individuals and 58 healthy controls and observed a strong link between eGFR and liver enzymes. **Jia et al. (2015)** demonstrated a link between DN and liver enzymes in 238 patients with T2DM, and showed that an elevation in liver enzymes was linked to rapid albuminuria progression and low GFR.

The NLR is a chronic inflammatory marker that reflects a delicate balance between two immune system elements: neutrophils, which are the active non-specific inflammatory mediators that contribute the first line of defense, and lymphocytes, which are the regulatory or protective aspect of inflammation. NLR has been found to have a favorable correlation not only with the presence of

metabolic syndrome, but also with the severity of it **(Buyukkaya et al., 2014)**.

When comparing the cases group (7.1 ± 6.5) to the control group (4.8 ± 3.3), we discovered a statistically significant (P = 0.022) increase in NLR in the cases group. This was in agreement with the findings of Akbas and colleagues, who demonstrated that NLR was considerably higher in diabetics with nephropathy who had greater albuminuria, implying a link between inflammation and endothelial dysfunction **(Akbas et al., 2014).**

Likewise, Khandare and colleagues showed that the mean NLR was considerably greater in diabetic patients with albuminuria (2.83 ±0.85) than in those without albuminuria (1.94 ±0.65).. Furthermore, levels of absolute neutrophil count (ANC) and absolute lymphocyte count (ALC) were suggested to be substantially linked with albuminuria patients **(Khandare et al., 2017).**

Also, Huang and colleagues discovered that NLR levels in T2DM patients with signs of DN (2.48±0.59) were substantially greater than in diabetic patients without nephropathy (2.20±0.62) and healthy controls (1.80±0.64) **(Huang et al., 2015)**.

Furthermore, Moursy and colleagues discovered that NLR values in diabetic patients with retinopathy (P<0.001), neuropathy (P=0.025), and nephropathy (P<0.001), were considerably greater than those in diabetic patients with no microvascular problems and healthy controls **(Moursy et al., 2015)**.

In another study involving a total of 112 T2DM patients, **Kahraman et al. (2016)** established that elevated NLR levels related to albuminuria and glomerular filtration rate in diabetic patients .

Based on the findings obtained in the present study, having elevated BMI, having raised liver enzymes, and having a high LDL were all risk factors for having NAFLD in CKD patients. Furthermore, findings show a link between higher steatosis and increased fibroscan-measured liver stiffness and an increased risk of NAFLD in our CKD group; this was in accordance with **Targher et al. (2010), Yilmaz et al. (2010), Mikolasevic et al. (2013), and Choe et al. (2013) all agreed on this (2020).**

Our findings follow the results of **(Behairy et al., 2021)** who conducted a cross-sectional study that included 80 non-diabetic CKD patients, found a significant association between the severity of hepatic steatosis degree with decreased GFR and increased CKD stage. The degree of liver stiffness was significantly related to an increased hepatic steatosis grade. A significant positive correlation was found between the degree of NAFLD and serum levels of ALT, AST, and LDL ($P < 0.05$). Notably, NAFLD was significantly associated with an obvious history of cardiovascular disease among the studied patients. Furthermore, (**Qin et al., 2015**) identified liver stiffness as a risk factor for CKD and it is a potential indicator for CKD in NAFLD patients.

Our findings support those of (Behairy et al., 2021), who showed a significant link between the severity of hepatic steatosis and lower GFR and higher CKD stage in a cross-sectional research including 80 non-diabetic CKD patients. The degree of liver stiffness was found to be linked to the severity of hepatic steatosis. The degree of NAFLD and blood levels of alanine transaminase, aspartate aminotransferase ,and low density lipoprotein were revealed to have a significant positive connection (P< 0.05). Among the patients evaluated, NAFLD was found to be substantially linked to a history of cardiovascular disease. In addition, **(Qin et al., 2015)** found hepatic stiffness as a risk factor for CKD and a possible indicator of CKD in NAFLD patients.

Conclusion

The prevalence of MAFLD was greater in CKD patients, and NLR levels were considerably higher in CKD patients compared to healthy people. Advanced hepatic fibrosis and greater steatosis as

measured by TE were both linked to an increased risk of CKD.

A list of abbreviations

Abbreviation	Full Term
ACR	albumin-to-creatinine ratio
BMI	Body Mass Index
CBC	Complete Blood Count
CKD	chronic kidney disease
eGFR	estimated glomerular filtration rate
HCC	Hepatocellular Carcinoma
IR	Insulin resistance
MAFLD	Metabolic disorder associated Fatty Liver Disease
NAFLD	non-alcoholic fatty liver disease
NASH	non-alcoholic steatohepatitis
SD	Standard Deviation
T2DM	Type II diabetes mellitus
NLR	Neutrophil lymphocytes ratio
CAP	controlled attenuation parameter
LSM	liver stiffness measurement
ESRD	End stage renal disease
HD	hemodialysis

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