Original Article

The association of liver fibrosis and chronic kidney disease in patients with metabolic associated fatty liver disease

A cross-sectional study

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ABSTRACT

الأهداف: تقييم العلاقة بين تليف الكبد والقصور الكلوي المزمن لدى مرضى الكبد الدهني المرتبط بالتمثيل الغذائي، وإلقاء الضوء على عوامل الخطر لتليف الكبد لدى هؤلاء المرضى.

المنهجية: اشتملت الدراسة على 84 مريضًا يعانون من القصور الكلوي المزمن و مرض الكبد الدهني المرتبط بالتمثيل الغذائي و 80 مريضًا مصابين بمرض الكبد الدهني المرتبط بالتمثيل الغذائي بدون قصور كلوي مزمن في هذه الدراسة المقطعية التي أجريت في مستشفى جامعي مرجعي من مايو 2021م إلى يناير 2023م. وقد تم فحصهم بواسطة الموجات فوق الصوتية على البطن وتصوير المرونة العابر لتقييم التليف الكبدي.

النتائج: كان هناك انتشار أعلى للتليف في المرضى الذين يعانون من القصور الكلوي المزمن مقارنة مع أولئك الذين لا يعانون منه (75.6% مقابل 24.4%). وكشف التحليل اللوجستي أن الأمراض المصاحبة لمرض الكبد الدهني المرتبط بالتمثيل الغذائي، بما في ذلك أمراض القلب و الأوعية الدموية والسكري وارتفاع ضغط الدم، كانت مرتبطة بشكل مستقل مع القصور الكلوي المزمن. ولم يتم العثور على الجنس ومؤشر كتلة الجسم كعوامل مستقلة تتعلق بالقصور الكلوي المزمن. بالإضافة إلى ذلك، وبصرف النظر عن الأمراض المصاحبة، تم ربط عوامل مثل العمر وارتفاع مستوى حمض يوريك الدم ومستوى دهون الدم الثلاثية و مستوى كوليستيرول الدم و نسبة البليروبين بالدم، و نقص مستوى ألبومين الدم، و التهاب الكبد الفيروسي بشكل مستقل بمرض الكلى المزمن.

الخلاصة: يمكن أن يمثل مرض القصور الكلوي المزمن عامل خطر محتمل لتطور تليف الكبد لدى مرضى الكبد الدهني المرتبط بالتمثيل الغذائي.

Objectives: To examine the relation between liver fibrosis and chronic kidney disease (CKD) in metabolic-associated fatty liver disease (MAFLD) patients and its risk factors.

Methods: The current study was carried out at Tanta University Hospital, Tanta, Egypt, from May 2021 to January 2023 and included 84 MAFLD patients with CKD and 80 MAFLD patients without CKD. All participants had been examined by abdominal ultrasonography and transient elastography with controlled attenuation parameter.

Results: Chronic kidney disease patients exhibited a greater incidence of fibrosis compared to patients without CKD (75.6% vs. 24.4%). Logistic analysis demonstrated that the presence of multiple health conditions, such as MAFLD, diabetes mellitus, hypertension, and cardiovascular disease, were individually linked to CKD. Gender and body mass index were not independent factors related to CKD. Additionally, factors such as age, hyperuricemia, hypertriglyceridemia, hypercholesterolemia, hypoalbuminemia, hyperbilirubinemia, and viral hepatitis, apart from MAFLD comorbidities, were independently linked to CKD.

Conclusion: Chronic kidney disease may represent a potential risk influence for liver fibrosis development in MAFLD patients.

Keywords: CKD, MAFLD, fibroscan, liver fibrosis

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In 1980, the non-alcoholic fatty liver disease (NAFLD) was first identified as a distinct medical condition. It is characterized by accumulated fat in the liver like alcoholic fatty liver disease, although it occurs in non-drinkers and in absences of other liver disease causes.[1] Over the past 4 decades, advancements and increased disease prevalence have contributed to

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a deeper understanding of the disease's pathogenesis, with metabolic dysregulation identified as a significant factor. There has been considerable argument regarding the nomenclature of NAFLD.[2]

An international consensus has proposed that metabolic dysregulation is caused by fatty liver disease. Non-alcoholic fatty liver disease has been replaced by "metabolic (dysfunction) associated fatty liver disease (MAFLD)" term.[3]

Hepatic steatosis and one of these 3 criteria are necessary for MAFLD diagnosis: I) body mass index (BMI) of 25 kg/m^2 or higher; II) metabolic dysfunction; and III) type 2 diabetes mellitus (DM). In order for metabolic dysfunction to be present, 2 out of the following 7 metabolic risk factors must be met. They include a waist circumference of 94 cm or 80 cm for men and women, high-density lipoprotein (HDL)-cholesterol of 40 mg/dL or lower for men and women, plasma triglycerides of 150 mg/dL or higher, prediabetes, an insulin resistance score of more than 2.5 on the homeostatic model assessment (HOMA)-insulin test, blood pressure of more than or equal 130/85 mmHg, and plasma C-reactive protein (CRP) of 2 mg/dL or higher.[3,4]

Chronic kidney disease (CKD) is considered a chronic debilitating illness impacting approximately 15% of American adults, with 9 out of 10 CKD patients being unaware of their condition.[5] A 60 ml/min/1.73m^2 or lower estimated glomerular filtration rate (eGFR) defines CKD, according to KDIGO guideline.[6]

Recent trends in CKD research focus on assessing new modifiable risk factors for this chronic, devastating disease.[7] A proposed hypothesis links NAFLD and CKD as both conditions share chronic comorbidities, metabolic dysfunctions, and cardiovascular diseases (CVD).[8]

Although several studies have hypothesized that NAFLD is linked to 20-25% CKD prevalence, little is known regarding how CKD affects MAFLD development.[9-11] Impact of CKD on MAFLD and its effects on liver fibrosis were examined in this study. Chronic kidney disease patients with MAFLD were also examined for liver fibrosis risk factors.

Methods. This cross-sectional study recruited patients from Tanta University Hospital's Tropical Medicine, Internal Medicine, and Radiology Departments, Tanta, Egypt, from May 2021 to January 2023. A total of 480 diagnosed CKD patients as per KDIGO guidelines were included and examined for the presence of MAFLD.[6] We used simple linear research method by searching the systemic reviews and meta-analysis.

The presence of MAFLD and CKD divided patients into 2 groups. The control group, consisting of 80 MAFLD patients without CKD, was referred to as Group I. Group II included 84 patients with CKD and concomitant MAFLD, identified by a liver steatosis score of "more than 237 dB/min" by Fibroscan.

This study included patients, over the age of 18, meeting the following inclusion criteria for CKD and MAFLD: I) CKD is characterized by kidney structural damage (eGFR of <60 ml/min/1.73m^2) that lasts for a minimum of 3 months;[6] and II) for MAFLD diagnosis, hepatic steatosis - diagnosed by abdominal ultrasound (US) then confirmed by Fibroscan - and one of the following criteria are necessary: BMI of 25 kg/m^2 or higher, metabolic dysfunction and type 2 DM. In order for metabolic dysfunction to be present, 2 out of the following 7 metabolic risk factors must be met. They include a waist circumference of 94 cm or 80 cm for men and women, HDL-cholesterol of 40 mg/dL or lower for men and women, plasma triglycerides of 150 mg/dL or higher, prediabetes, an insulin resistance score of more than 2.5 on the HOMA-insulin test, blood pressure of more than or equal 130/85 mmHg and plasma CRP of 2 mg/dL or higher.[3,4]

The exclusion criteria were as follows: I) individuals who are below the age of 18; II) hepatotoxicity caused by drug exposure (DILI); III) historical record of alcohol consumption; IV) unwillingness to participate in the current study; and V) autoimmune hepatitis.

This study followed Helsinki Declaration principles. Permission was obtained from the local research ethics committee of Tanta University, Tanta, Egypt (approval code: 34667/5/21), and informed consent was procured. Confidentiality of the data and privacy of participants were ensured. Risks and benefits were disclosed, and any unexpected risks during the research were promptly communicated to participants and the ethical committee on time.

During the physical examination, the following demographic and clinical information was obtained from the subjects: age, gender, medical history, and blood pressure (systolic and diastolic).

Participants gave overnight fasting blood samples under aseptic conditions. Serum separating tubes for serum bilirubin, creatinine, aspartate aminotransferase (AST), alanine transaminase (ALT), fasting blood

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glucose, and albumin levels were collected. The Vantaa, Finland-based Konelab Prime-60i automated chemistry analyzer measured the samples. Total cholesterol and triglycerides were determined utilizing commercial kits (Spinreact, Spain). The complete blood count was automatically carried out using the Erma PCE-210 hematology analyzer, using tubes treated with ethylenediaminetetraacetic acid. Centrifuged citrated blood samples were quickly separated into plasma and analyzed for international normalized ratio using a coagulation analyzer (Sysmex CA1500, Siemens, Erlangen, Germany). The equation of CKD-EPI was employed to determine eGFR: (GFR=141 * min (Scr/κ,1) α * max (Scr/κ, 1)-1.209 * 0.993Age * 1.018 [if female] * 1.159 [if black]). The urine albumin-creatinine ratio (uACR) was determined by dividing the concentration of albumin in urine (mg) by the concentration of creatinine in urine (g).[12]

Abdominal and pelvic US was carried out to assess splenic size, liver condition, and the presence of ascites using Mindray DC30 Ecografo apparatus.

The 502 M and XL fibro scan probe (echosens-France) were used to measure hepatic stiffness and experts used FIBROSCAN® 502 TOUCH apparatus and the controlled attenuation parameter (CAP) to measure liver steatosis, following the manufacturer's guidelines. The patient was positioned lying on their back on a table, with the right arm fully extended away from the body; in order to accomplish the procedure, the intercostal transthoracic window must be accessed over the right hepatic lobe.

These CAP cutoff values, derived from a prior study, were used to identify hepatic steatosis (S) cases. Steatosis severity was graded from 0 (no steatosis, 237 dB/m) to 3 (severe steatosis, 291.0-400.0 dB/m). Fibrosis was classified as F0 (no fibrosis, 5.5 kPa), F1 mild (5.5-8.0 kPa), F2 moderate (8.0-10.0 kPa), F3 severe (11-16.0 kPa), and F4 cirrhosis.[13]

Statistical analysis. The data was analyzed using the Statistical Package for the Social Sciences, version 26.0 (IBM Corp., Armonk, NY, USA). Frequencies and percentages were used to display the categories. We used the Chi-square test for trend (linear-by-linear association) to examine the relationship between categorical and ordinal variables. The Shapiro-Wilk test normalized continuous data. The numerical variables standard deviation (SD) and mean followed normal distributions. Fisher's exact and Pearson's Chi-square were used to assess categorical variables. Independent t-tests were implemented to evaluate 2 distinct groups. For numerical variables that are not normal, employ the median + interquartile range (IQR). Two independent groups were compared using Mann-Whitney U. Additionally, a systematic multivariable regression analysis was carried out to predict CKD fibrosis. With 95% confidence intervals (CIs), the adjusted odds ratios were provided. Statistical significance was determined by *p*-values <0.05.[14]

Results. In this cross-sectional study, 164 patients were divided into 2 groups based on MAFLD and CKD presence. Tables 1 & 2 show baseline demographic, laboratory, and comorbidity data.

In the CKD group, the steatosis score was 341.3±49.1 dB\m; with more than half of the participants having S2 or S3 steatosis. Conversely, in the non-CKD group, the steatosis score was 257.3±46.8 dB\m. There was a significant increase in steatosis score in CKD group (p<0.001), with most cases exhibiting S3 steatosis. The median CKD fibrosis score was 7.4 kpa, with most cases having F2, F3, or F4 fibrosis. Non-CKD patients had a median fibrosis score of 9.2 kpa. Table 3 shows no significant difference in fibrosis between groups (p=0.414).

The CKD group was further subdivided into 5 stages according to eGFR. All cases in stage 5 (4 cases) had S2 steatosis. A significant increase in steatosis and fibrosis was detected in CKD patients (p<0.001, Table 4).

Patients were divided by fibrosis presence or absence. Patients with fibrosis showed significant increases in BMI (p<0.001) and age (p<0.002). All the following

Table 1 - Comparison between non-chronic kidney disease and chronic kidney disease groups regarding demographic data and comorbidities.

Variables	MAFLD patients		*P*-values
	Non-CKD group (n=80)	CKD group (n=84)	
Age (years), mean±SD	46.4±11.5	53.9±9.7	<0.001*
Gender			
Female	48 (60.0)	56 (66.7)	0.376
Male	32 (40.0)	28 (33.3)	
BMI (kg/m²), mean±SD	37.5±6.9	36.0±7.3	0.179
Comorbidities			
No	28 (35.0)	0 (0.0)	<0.001*
DM	32 (40.0)	12 (14.3)	
Hypertension	12 (15.0)	28 (33.3)	
DM and hypertension	8 (10.0)	40 (47.6)	
CVD	0 (0.0)	4 (4.8)	

Values are presented as numbers and percentages (%). *Significant at a *p*-value of <0.05. MAFLD: metabolic associated fatty liver disease, CKD: chronic kidney disease, SD: standard deviation, BMI: body mass index, DM: diabetes mellitus, CVD: cardiovascular disease

Table 2 - Laboratory characteristics of the studied non-chronic kidney disease and chronic kidney disease groups.

Parameters	MAFLD patients		*P*-values
	Non-CKD group (n=80)	CKD group (n=84)	
Serum uric acid(mg\dl)	5.1±0.8	7.0±1.3	<0.001*
Triglycerides(mg\dl)	131.3±52.0	172.7±26.7	<0.001*
Cholesterol(mg\dl)	212.8±38.8	230.5±46.5	0.009*
Serum albumin(g/dl)	4.0±0.3	3.5±0.5	<0.001*
Bilirubin(mg\dl)	1.0±0.1	1.0±0.2	0.142
ALT (U\L)	49.5±12.0	27.1±11.8	<0.001*
AST (U\L)	45.0±8.7	30.2±15.2	<0.001*
Viral hepatitis markers			
Negative	76 (95.0)	52 (61.9)	<0.001*
HCV positive	4 (5.0)	12 (14.3)	
HCV positive not received treatment	0 (0.0)	8 (9.5)	
HCV positive received treatment	0 (0.0)	12 (14.3)	

Values are presented as mean ± standard deviation (SD) or numbers and percentages (%). *Significant at a *p*-value of <0.05. MAFLD: metabolic associated fatty liver disease, CKD: chronic kidney disease, ALT: alanine transaminase, AST: aspartate aminotransferase, HCV: hepatitis C

Table 3 - Comparison of the steatosis and fibrosis status of the liver between the studied non-chronic kidney disease and chronic kidney disease groups.

Variables	MAFLD patients		*P*-values
	Non-CKD group (n=80)	CKD group (n=84)	
Steatosis score (dB\m), mean±SD	257.3±46.8	341.3 ±49.1	<0.001*
Steatosis degrees			
S0	0 (0.0)	8 (9.5)	<0.001*
S1	8 (10.0)	28 (33.3)	
S2	4 (5.0)	32 (38.1)	
S3	68 (85.0)	16 (19.0)	
Fibrosis score (kpa), median (IQR)	9.2 (7.3-11.0), mean rank: 85.60	7.4 (6.0-14.4), mean rank: 79.55	0.414
Fibrosis grades			
F0	16 (20.0)	24 (28.6)	<0.001*
F1	4 (5.0)	12 (14.3)	
F2	28 (35.0)	12 (14.3)	
F3	28 (35.0)	8 (9.5)	
F4	4 (5.0)	28 (33.3)	

Values are presented as numbers and percentages (%). *Significant at a *p*-value of <0.05. MAFLD: metabolic associated fatty liver disease, CKD: chronic kidney disease, SD: standard deviation, IQR: interquartile range

laboratory tests showed no statistically significant differences: blood urea (p=0.581), fasting blood glucose (p=0.527), serum cholesterol (p=0.363), steatosis score (p=0.135), serum creatinine (p=0.063), serum bilirubin (p=0.082), triglycerides (p=0.624), serum albumin (p=0.366), and uric acid (p=0.537). Significant increases were noted in AST (p<0.001), eGFR (p=0.036), and ALT (p<0.001), as shown in Table 5.

The stepwise multivariable regression analysis model for predicting liver fibrosis among CKD cases demonstrated an accuracy rate of 85.71%, a sensitivity of 93.33%, and a specificity of 66.67%. The findings suggested that eGFR, CKD stages 2 and 3, and steatosis stage 2 were independent risk factors for liver fibrosis progression in CKD patients (Table 6).

Discussion. Metabolic associated fatty liver disease has developed as the predominant factor behind chronic liver disease, often associated with chronic comorbidities like diabetes, obesity, and hypertension. Chronic kidney disease is yet another chronic condition that frequently coexists with MAFLD, though its contribution to liver fibrosis in MAFLD patients remains poorly understood. Nonetheless, there is a hypothesis suggesting that CKD may promote heightened inflammation and oxidative stress within the liver, potentially causing harm to liver cells and expediting the progression of fibrosis. Musso et al[15] demonstrated an evidence suggesting that NAFLD and CKD may have overlapping pathogenic mechanisms and potential targets for therapy.

Chronic renal injury and NAFLD have been extensively studied, but CKD effect on MAFLD progression is unknown. The current study examined the popularity and effects of CKD on liver fibrosis severity in MAFLD patients. There were 164 eligible MAFLD patients involved in this research. They were categorized into 2 groups: 80 without CKD and 84 with CKD. Subsequently, a comparative analysis of liver fibrosis and steatosis levels between these 2 groups was carried out.

This study revealed significant findings, demonstrating that patients diagnosed with CKD exhibit significantly elevated levels of liver fibrosis and steatosis compared to those without CKD. These

Table 4 - Associations between chronic kidney disease stages and the steatosis and fibrosis grades.

Variables	CKD stages					*P*-values
	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	
Steatosis degrees						
S0	0 (0.0)	0 (0.0)	8 (40.0)	0 (0.0)	0 (0.0)	<0.001*
S1	4 (50.0)	12 (50.0)	0 (0.0)	12 (42.9)	0 (0.0)	
S2	4 (50.0)	12 (50.0)	4 (20.0)	8 (28.6)	4 (100)	
S3	0 (0.0)	0 (0.0)	8 (40.0)	8 (28.6)	0 (0.0)	
Fibrosis grades						
F0	8 (100)	4 (16.7)	4 (20.0)	4 (14.3)	4 (100)	<0.001*
F1	0 (0.0)	4 (16.7)	4 (20.0)	4 (14.3)	0 (0.0)	
F2	0 (0.0)	8 (33.3)	0 (0.0)	4 (14.3)	0 (0.0)	
F3	0 (0.0)	0 (0.0)	8 (40.0)	0 (0.0)	0 (0.0)	
F4	0 (0.0)	8 (33.3)	4 (20.0)	16 (57.1)	0 (0.0)	

Values are presented as numbers and percentages (%). CKD: chronic kidney disease

Table 5 - Age, body mass index, and laboratory parameters of fibrosis and non-fibrosis groups (N=164).

Parameters	Fibrosis		*P*-values
	No (n=40)	Yes (n=124)	
Age, years	45.6±12.5	51.7±10.4	0.002*
body mass index	34.3±4.3	37.6±7.7	0.001*
Fasting blood glucose (mg\dl), median (IQR)	118.5 (0.0-130.0)	113.0 (0.0-130.0)	0.527
Steatosis score (dB\m)	287.8±43.0	301.6±68.9	0.135
Cholesterol (mg\dl)	216.4±52.5	223.6±40.5	0.363
Triglycerides (mg\dl)	155.6±34.3	151.5±49.2	0.624
Uric acid (mg\dl)	6.0±1.3	6.1±1.5	0.537
Creatinine (mg\dl), median (IQR)	2.00 (1.0-7.03)	1.10 (1.0-3.0)	0.063
Urea (mg\dl), median (IQR)	61.8 (29.0-112.0)	34.0 (23.0-111.0)	0.581
eGFR (ml/min/1.73m^2)	57.5±20.1	48.3±16.8	0.036*
Serum albumin (g/dl)	3.8±0.5	3.7±0.5	0.366
Bilirubin (mg\dl)	0.9±0.2	1.0±0.2	0.082
ALT (U\L)	29.9±14.8	40.7±16.0	<0.001*
AST(U\L)	26.3±9.9	41.0±13.9	<0.001*

Values are presented as mean ± standard deviation (SD) or median and interquartile range (IQR). *Significant at a *p*-value of <0.05. eGFR: estimated glomerular rate, ALT: alanine transaminase, AST: aspartate aminotransferase

findings recommend that CKD might represent a potential risk influence for liver fibrosis development in MAFLD patients.

Researchers concluded that CKD increases the incidence of liver fibrosis in MAFLD patients. This holds clinical importance as liver fibrosis can evolve into the life-threatening complications of liver cirrhosis. Therefore, prompt identifying MAFLD patients who are at risk of CKD, and vice versa, becomes paramount to ensuring thorough monitoring and timely intervention.

This study found that CKD patients had a superior incidence of fibrosis (75.6% vs. 24.4%). Hu et al[16] found a significant correlation between CKD and MAFLD prevalence, but MAFLD was not an independent risk factor. Conversely, Deng et al[17] found no independent link between MAFLD and CKD using national health and nutrition examination survey (NHANES) 2017-2018 data from the US. Thus, metabolic abnormalities like diabetes and hyperuricemia may affect MAFLD-CKD connection.

Furthermore, Sun et al[18] found no independent link between MAFLD and prevalent CKD, after controlling for factors like gender, ethnicity, age, pre-existing diabetes, and alcohol intake using NHANES 1988-1994 data. Instead, MAFLD is independently and substantially correlated with CKD and abnormal albuminuria, especially with a high liver fibrosis score.

A cohort study in southern China from July 2020 to June 2021 found that MAFLD raises the danger of CKD, with type 2 DM being the main driver. The MAFLD with type 2 DM group has a higher risk of CKD and higher uACR than the pre-diabetes and

Table 6 - Stepwise multivariable regression analysis model for prediction of fibrosis among cases with chronic kidney disease (N=84).

Predictors	B coefficient	*P*-values	AOR	95% CI	Sensitivity	Specificity	Accuracy	*P*-values
CKD stage 2	7.82	0.007*	2494.530	27.43-226846.45	93.33%	66.67%	85.71%	<0.001*
CKD stage 3	3.15	0.036*	23.48	1.22-451.93				
Steatosis S2	-5.88	0.002*	0.002	0.001-0.063				
eGFR	-0.135	0.001*	0.873	0.802-0.951				

*Significant at a *p*-value of <0.05. AOR: adjusted odds ratio, CI: confidence interval, CKD: chronic kidney disease, eGFR: estimated glomerular rate

regular glucose groups. Reaching metabolic goals in MAFLD significantly lowers CKD risk.[19]

Our study results revealed that the dominance of hepatic steatosis in the CKD group is 90%, while in the non-CKD group, it is 100%. Data from South Korea indicated that NAFLD fibrosis score (NFS) of ≥ -1.455 is associated with a more pronounced decline in eGFR in CKD patients. The estimated prevalence rates of advanced fibrosis were 40% in individuals with non-CKD and NAFLD, compared to 19% in the CKD group.[20]

Regarding the correlation between the steatosis and CKD stages, the current study observed that the steatosis was higher in stages 2 and 3 of CKD than in other stages, possibly due to smaller numbers in the different groups. This suggests an association between increased steatosis severity and more advanced CKD stages. According to a clinical trial of biopsy-proven non-alcoholic steatohepatitis (NASH) patients, lifestyle changes that improve liver fibrosis stages independently improve renal function.[21]

When considering the etiology of CKD, it is noteworthy that steatosis is absent in half of patients with diabetes as the sole cause of nephropathy and in all cases involving glomerulonephritis. Conversely, the degree of steatosis significantly increases in patients with hypertensive nephropathy, particularly when both diabetes and hypertension are implicated as etiological factors.

The study of comorbidities' impact on fibrosis progression in CKD patients is particularly significant, especially when dealing with combined pathologies such as diabetic and hypertensive nephropathy. Among patients with NAFLD or without NAFLD, diabetes did not significantly increase the risk of CKD in cross-sectional research including 4,637 people carried out in Japan. It is possible that this is because the sample size was too small.[24] In contrast, research by Hu et al[16] confirmed Deng et al's[17] NHANES 2017-2018 findings that DM is one of the main causes of CKD in the overall population.

Several investigations have pointed to NAFLD as a potential key player in CKD onset and progression. Mechanisms include metabolic syndrome, type 2 DM, visceral adipose dysfunction, intestinal dysbiosis, and platelet activation.[24]

Logistic analysis showed that MAFLD comorbidities like hypertension, diabetes, and cardiovascular disease independently predicted CKD. Gender and BMI did not independently predict CKD.

Additionally, apart from MAFLD comorbidities, factors such as age, hyperuricemia, hypertriglyceridemia, hypercholesterolemia, hypoalbuminemia, hyperbilirubinemia, and viral hepatitis were independently linked to CKD.

Subgroup analyses showed that age, BMI, eGFR, and elevated ALT and AST levels increase MAFLD fibrosis risk. However, after adjustment, only eGFR remained an independent risk factor for liver fibrosis, with an adjusted odds ratio of 0.873 (p=0.001).

To explore the aspects independently coupled with CKD, a multivariable regression analysis was carried out to predict liver fibrosis in CKD cases. Chronic kidney disease stage 2 exhibited the highest adjusted odds ratio (2494.530) with a confidence interval ranging from 27.43-226846.45 (p=0.007).

Study limitations. It is crucial to emphasize certain constraints of this investigation. It is cross-sectional, which precludes the determination of cause-and-effect relationships between CKD and liver fibrosis. The study also had a small sample size, which may limit generalizability.

In conclusion, in individuals with MAFLD, this research looked at how CKD correlated with liver fibrosis. Chronic kidney disease may represent a possible risk factor for liver fibrosis development, and eGFR is a distinct predictor of liver fibrosis. These findings need to be confirmed and the mechanisms of liver fibrosis with CKD need to be further studied in future researches.

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