Research Article

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MAFLD in kidney transplant recipients

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Prevalence of metabolic dysfunction-associated fatty liver disease in kidney transplant recipients: A cross-sectional study using FibroScan

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Abstract

Background and Aim: Metabolic dysfunction-associated fatty liver disease (MAFLD) is expected to be prevalent among kidney transplant recipients (KTRs). In this study, we evaluated the prevalence of MAFLD among KTRs, data that have not been investigated by any clinical study to date.

Materials and Methods: We included a total of 52 KTRs and 53 age-, sex-, and BMI-matched individuals as the control group through prospective consecutive recruitment. We detected the presence of hepatic steatosis and liver fibrosis using the controlled attenuation parameter (CAP) and liver stiffness measurement (LSM) defined by FibroScan.

Results: Among the KTRs, 18 (34.6%) had metabolic syndrome. The prevalence of MAFLD among the KTRs and controls was 42.3% and 51.9%, respectively (p=0.375). The CAP and LSM values did not differ significantly between the KTRs and controls (p=0.222 and p=0.119). Among the KTRs, patients with MAFLD had significantly higher age, BMI, waist circumference, LDL, and total cholesterol levels (p<0.001, p=0.011, p=0.033, p=0.022, and p=0.029, respectively). In multivariable analysis, age was the only independent factor for MAFLD among the KTRs (OR: 1.120, 95% confidence interval (CI): 1.039–1.208).

Conclusion: MAFLD among KTRs did not show a significantly higher prevalence compared to the normal population. Further clinical studies with larger populations are needed.

Keywords: FibroScan; kidney transplant recipients; mafld.

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Introduction

Nonalcoholic fatty liver disease (NAFLD) and chronic kidney disease (CKD) constitute a global public health problem, affecting approximately 25% and 10% of the world population, respectively.[1,2] In recent years, both NAFLD and CKD have shown increasing incidences.[2,3] Considering the progressive nature of NAFLD and CKD, both diseases have attracted the public's and healthcare authorities' attention in terms of increasing awareness toward the burden of the diseases.[2,3] The concomitance of NAFLD and CKD was demonstrated to be associated with the shared risk factors.[4] However, the accumulating data have shown the independent association of NAFLD and CKD regardless of traditional risk factors such as type 2 diabetes mellitus (T2DM), obesity, and hypertension.[5,6]

Kidney transplant recipients (KTRs) constitute a special group in renal replacement therapy. In KTRs, as a side effect of immunosuppressive therapy including steroids and calcineurin inhibitors, the risk of developing metabolic complications is increased.[7] In line with this knowledge, in their cross-sectional study, Mikolasevic et al.[8] showed that NAFLD is prevalent in more than 50% of KTRs. To the best of our knowledge, there has been a paucity of data since their study.

In 2020, a consensus statement recommended replacing the acronym NAFLD with metabolic dysfunction-associated fatty liver disease (MAFLD), which includes a positive set of criteria rather than an exclusion diagnosis. The positive diagnostic criteria were composed of mainly metabolic factors, which are the causative drivers of the disease.[9] Corticosteroids are widely used in the continuation therapy of KTRs, which have a steatogenic effect on the liver.[10] However, the use of steatogenic medications is an exclusion criterion in NAFLD diagnosis.[11] Therefore, due to the high rate of corticosteroid use, NAFLD could not be a diagnosis among KTRs. On the other hand, considering the strong relationship between CKD and NAFLD, NAFLD would probably be a common disorder among KTRs if these patients did not receive steatogenic medications. In that case, considering the newly defined diagnostic criteria, MAFLD can be diagnosed among KTRs due to the availability of only positive diagnostic criteria rather than the exclusion of other factors in the development of hepatic steatosis, which reflects the hepatic burden among those patients.

In this study, we aimed to evaluate the prevalence of MAFLD defined by transient elastography (TE) in KTRs. Furthermore, we investigated the independent predictors of MAFLD among KTRs.

Materials and Methods

Design

The study is a single-center study that includes 52 KTRs followed by nephrology clinics of Dr. Lutfi Kirdar Kartal Trainig and Research Hospital in 2013. The study protocol was approved by the ethical committee of Kartal Lutfi Kirdar Training and Research Hospital (protocol number: 89513307/1009/215, approval date: December 19, 2013) and conducted in adherence to the Declaration of Helsinki. Informed consent was obtained from all participants.

Population

For this cross-sectional study, we recruited a total of 52 consecutive KTRs and 53 age-, sex-, and BMI-matched controls. The demographic characteristics of the patients were extracted from the medical records, which also included a detailed medical history. The exclusion criteria were pregnancy at the time of recruitment and measurement failure of unreliable measurements in TE examinations.

The diagnosis of MAFLD was confirmed in the patients who fulfilled at least one of the following criteria: (1) being overweight (BMI>25 kg/m^2), (2) having T2DM, and (3) having evidence of at least two of the metabolic dysfunction criteria, in addition to hepatic steatosis confirmed by controlled attenuation parameter (CAP). The metabolic dysfunction criteria were as follows: (1) waist circumference ≥102/88 cm in men and women, (2) blood pressure ≥130/85 mmHg or specific drug treatment, (3) plasma triglycerides level ≥150 mg/dL or specific drug treatment, (4) plasma HDL level <40 mg/dL for men and <50 mg/dL for women or specific drug treatment, (5) prediabetes (i.e., fasting glucose levels 100–125 mg/dL or 2-h post-load glucose levels 140–199 mg/dL or glycated hemoglobin 5.7%–6.4%), (6) homeostasis model assessment-insulin resistance score ≥2.5, and (7) plasma high-sensitivity C-reactive protein level >2 mg/L.[9] Metabolic syndrome and T2DM were diagnosed following the Adult Treatment Panel III and American Diabetes Association criteria.[12] The blood pressure measurements were obtained after 15 min of seated rest using an automated sphygmomanometer. Obesity was defined according to the classification of the World Health Organization.[13]

The immunosuppressive protocol included induction therapy with rabbit anti-thymocyte globulin (in cadaveric and immunologically intermediate-high risk living transplantation) or basiliximab (in immunologically low-risk living transplantation), followed by maintenance therapy with a calcineurin inhibitor (tacrolimus or cyclosporine), antimetabolite (mycophenolate mofetil and mycophenolate sodium) and low-dose prednisolone (5 mg/day). Mycophenolate mofetil or mycophenolate sodium was replaced by everolimus with the observed side effects of antimetabolites. In the case of an acute rejection episode, the KTRs were given bolus steroids. Further treatment was adjusted under rejection severity.

Laboratory Data Collection

Blood examinations, including serum creatinine, urea, glucose, hemoglobinA1c, lipid fractions, uric acid, liver function tests, ferritin, C-reactive protein (CRP), parathormone, and vitamin D, were conducted following overnight fasting. The normal aspartate transaminase (AST) and alanine transaminase (ALT) levels were >37 U/L and >40 U/L, respectively.[14] Twenty-four-hour creatinine clearance (24-h CCL) was measured following a 24-h urine collection and calculated as urine creatinine × 24-h urine volume/serum creatinine × 1440.[15] Microalbuminuria and macroalbuminuria were defined as a urinary albumin-to-creatinine level of 30–300 mg/g creatinine and >300 mg/g creatinine, respectively.[16] Urine albumin excretion was determined in the first-morning urine sample.

TE Examinations

The TE examinations were performed following at least 6 h of fasting using a FibroScan® 502 Touch device (Echosens SA, Paris, France) by a single operator (Y.Y.) under the manufacturer's instructions. Starting the examinations, the patients were placed in the dorsal decubitus position and the transducer M probe was placed in the intercostal space of the right lobe of the liver. When prompted by the device, the probe was switched to XL according to the automatic probe selection tool displayed in real-time. The presence of at least 10 valid measurements and an interquartile range-to-median ratio of ≤0.3 were considered reliable TE measurements.[17] The hepatic steatosis grades are determined as follows: mild steatosis (11%–33% fat, 238–259 dB/m), moderate steatosis (34%–66% fat, 260–292 dB/m), and severe steatosis (>67% fat; ≥293 dB/m).[18] The cutoff value of liver stiffness measurement (LSM) was used to determine significant fibrosis (F ≥2). An LSM value of ≥8.95 kPa defined the presence of significant fibrosis.[19]

Statistical Analysis

The normality of the data was analyzed using the Shapiro–Wilk test. The normally distributed data were presented as mean±standard deviation (SD), while the nonnormally distributed data were expressed as median (minimum–maximum). Student's t-test was used to compare the means of continuous variables that were normally distributed and had equal variances. The Mann–Whitney U test was used to compare the medians of the nonnormally distributed continuous variables. A logistic regression analysis was conducted to determine the independent impact on each parameter. Statistical analysis was performed using the IBM SPSS v.24 for Windows software and was reported with 95% confidence interval (CI). The level of significance was set at $p<0.05$.

The reports of the study conform to the STROBE statement along with references to the STROBE statement and the broader EQUATOR guidelines.[20]

Results

Demographic and Clinical Characteristics

The study population comprised 52 KTRs and 53 age-, sex-, and BMI-matched controls. Most of the KTRs had undergone living donor transplantations (n=47, 90.4%). Hypertension was a significantly frequent comorbidity in KTRs in comparison with controls (n=30, 58% vs n=6, 11%, respectively; $p<0.001$). Obesity was prevalent in 15.4% (n=8) of the KTRs and 18.9% (n=10) of the controls (p=0.479). Among the KTRs, 18 had metabolic syndrome (34.6%). The demographic and clinical characteristics of the patients are depicted in Table 1.

Laboratory Data

Due to ethical reasons, blood examinations were performed only in KTRs in concordance with their routine. Only 9.6% (n=5) of the KTRs showed elevated AST and/or ALT levels. The data derived from laboratory examinations are presented in Table 2.

Table 1. General characteristics of the study and control groups

	Kidney transplant recipients (n=52)	Controls (n=53)	p
Age, years, mean (range)	41 (20–58)	35 (19–78)	0.403
Gender (male/female), n (%)	31 (59.6%)/21 (40.4%)	26 (49.1%)/27 (50.9%)	0.278
BMI, kg/m^2, mean (range)	25.6 (17.9–42.4)	27.3 (17.1–41.7)	0.094
Waist circumference, cm, mean (range)	101 (73–192)	93 (57–120)	**<0.001**
Diabetes mellitus (yes/no), n (%)	7 (13.5%)/45 (86.5%)	5 (9.4%)/48 (80.6%)	0.517
Hypertension (yes/no), n (%)	30 (57.7%)/22 (42.3%)	6 (11.3%)/47 (88.7%)	**<0.001**
Donor type (cadaveric/living), n (%)	5 (9.6%)/47 (90.4%)	NA	NA

BMI: Body mass index; NA: Not applicable. Significant p values are written in bold. Normally distributed data are presented as mean±standard deviation and analyzed with an independent samples t-test. Nonnormally distributed data are expressed as median (minimum–maximum) and analyzed using the Mann–Whitney U test. Categorical data are given as counts and percentages and compared with the Chi-squared test.

Table 2. Laboratory findings of the kidney transplant recipients

Parameters	
AST, U/L, mean (range)	18 (10–212)
ALT, U/L, mean (range)	15 (7–57)
Elevated transaminase, n (%)	5 (9.6%)
Creatinine, mg/dL, mean±SD	1.1±0.4
Albumin, mg/dL, mean±SD	4.4±0.3
Creatinine clearance, mL/min, mean±SD	85±20
24-h-protein in urine, mg/day, mean (range)	136 (22–7025)
Glucose, mg/dL, mean (range)	90 (72–203)
HbA1c, %, mean (range)	5.0 (4.4–8.0)
Uric acid, mg/dL, mean±SD	5.8±1.5
Total cholesterol, mg/dL, mean±SD	192±41
HDL, mg/dL, mean (range)	49 (28–109)
LDL, mg/dL, mean±SD	113±31
Triglycerides, mg/dL, mean (range)	116 (30–363)

ALT: Alanine transaminase; AST: Aspartate transaminase; SD: Standard deviation; HbA1c: Hemoglobin A1c; HDL: High-density lipoprotein; LDL: Low-density lipoprotein. Normally distributed data are presented as mean±standard deviation and nonnormally distributed data as median (minimum–maximum). Categorical data are given as counts and percentages.

TE Measurements

Characteristics of the patients regarding TE examinations are summarized in Table 3. Accordingly, CAP and LSM results did not significantly differ between KTRs and controls (p=0.222 and p=0.058, respectively). There was no individual with significant fibrosis except one patient among the controls. MAFLD was observed in 42.3% (n=22) of the KTRs, which was not significantly different from the controls (p=0.436).

Determinants of Being MAFLD among KTRs

In univariate analysis, age, BMI, waist circumference, and total cholesterol and LDL levels revealed a significant difference between KTRs with MAFLD and without MAFLD (p<0.001, p=0.011, p=0.033, p=0.022, and p=0.029, respectively). A comparison of MAFLD and non-MAFLD KTRs is summarized in Table 4. In logistic regression analysis, age persisted as the only independent risk factor for having MAFLD (OR: 1.120, 95% CI: 1.039–1.208).

Discussion

In our cross-sectional study, we showed that the prevalence of MAFLD among KTRs was 42.3%, which was lower than our age-, sex-, and BMI-matched controlled group with a MAFLD prevalence of 51.9%. However, the difference between KTRs and controls was not significant (p=0.375). To the best of our knowledge, this is the first study that presented the prevalence of MAFLD among KTRs. Previously, Mikolasevic et al.[8] reported that NAFLD was prevalent in over half of the KTR patients. However, the authors did not consider the steatogenic effect of corticosteroids in their population. Therefore, the prevalence of NAFLD in their study corresponds to hepatic steatosis rather than NAFLD itself.

In our study, we detected a relatively high prevalence of MAFLD in both KTRs and controls compared to the estimated NAFLD prevalence of the general world population.[1] However, it is known that the prevalence of NAFLD is also region dependent. The prevalence of NAFLD varies between 10% and 40%, with the lowest prevalence in Africa and the highest in the Middle East.[1] The studies conducted with a Turkish population reported a prevalence of 48%–61%. A recent multicenter study performed with patients who were presented in ambulatory settings with complaints of dyspepsia exhibited the prevalence of MAFLD as 45.5%.[21] In our study, we found similar results that show coherence with the data from the literature. The insignificant difference can be explained by the BMI-matched nature of the study.

It is well known that the survival time of KTRs is significantly shortened by atherosclerotic cardiovascular disease.[22] Moreover, as in NAFLD patients, cardiovascular diseases are an important causality contributing to morbidity and mortality also in KTRs.[23] The recipients are potentially at higher risk for NAFLD due to cardiometabolic risk factors, including T2DM, obesity, hypertension, and transplant-related conditions, which share physiopathologic features with NAFLD, such as chronic relapsing immune activation and potentially hepatotoxic immunosuppressive drugs.[24] In our study population, the rates of hypertension, T2DM, and obesity were 58%, 13%, and 15%, respectively, among the KTRs. Moreover, the total cholesterol and LDL levels were significantly higher in KTRs with MAFLD. Although we did not investigate the cardiovascular outcome of the patients in our study, considering the high prevalence of cardiovascular risk factors and MAFLD, cardiovascular-related disorders would be an important morbidity in those patients.

Accumulating evidence indicates an increased risk of all-cause mortality and a strong link between NAFLD and CKD.[25] Sinn et al.[26] found an increased risk of incident CKD in patients with NAFLD, thus advocating the screening of NAFLD patients for CKD. Recently, Sun

Table 3. Comparison of MAFLD and liver fibrosis status in kidney transplant recipients and controls

	Kidney transplant recipients (n=52)	Controls (n=53)	p
CAP, dB/m, mean (range)	233 (109–313)	238 (100–340)	0.222
LSM, kPa, mean±SD	4.6±1.1	5.0±1.6	0.119
MAFLD, n (%)	22 (42.3%)	27 (50.9%)	0.375
Mild/moderate/severe hepatosteatosis, n (%)	18 (34.6%) / 4 (7.7%) / 2 (3.8%)	9 (17.0%) / 10 (18.9%) / 8 (15.1%)	**0.026**
Significant fibrosis, n (%)	0 (0%)	1 (1.9%)	0.320

CAP: Controlled attenuation parameter; LSM: Liver stiffness measurement; MAFLD: Metabolic dysfunction-associated fatty liver disease; SD: Standard deviation. Significant p values are written in bold. Normally distributed data are presented as mean±standard deviation and analyzed with an independent samples t-test. Nonnormally distributed data are expressed as median (minimum–maximum) and analyzed using the Mann–Whitney U test. Categorical data are given as counts and percentages and compared with the Chi-squared test.

Table 4. Comparison of MAFLD and non-MAFLD kidney transplant recipients

	MAFLD (n=22)	Non-MAFLD (n=30)	p
Age, years, mean±SD	44±8	35±9	**<0.001**
Gender (male/female), n (%)	15 (68.2%)/7 (31.8%)	16 (53.3%) / 14 (46.7%)	0.281
BMI, kg/m^2, mean (range)	26.8 (22.8–42.4)	23.6 (17.9–33.70)	**0.011**
Donor type (living/cadaveric), n (%)	19 (86.4%)/3 (13.6%)	28 (93.3%)/2 (6.7%)	0.400
Diabetes mellitus (yes/no), n (%)	4 (18.2%)/18 (81.8%)	3 (10.0%)/27 (90.0%)	0.393
Hypertension (yes/no), n (%)	12 (54.5%)/10 (45.5%)	18 (60.0%)/12 (40.0%)	0.694
Waist circumference, cm, mean (range)	104 (89–128)	98 (73–192)	**0.033**
Glucose, mg/dL, mean (range)	91 (79–127)	89 (72–203)	0.258
HbA1c, %, mean (range)	5.2 (4.4–8.0)	5.0 (4.6–8.0)	0.642
Creatinine, mg/dL, mean (range)	1.0 (0.3–1.7)	1.0 (0.6–2.1)	0.566
Albumin, mg/dL, mean (range)	4.4 (3.6–4.8)	4.4 (3.9–5.2)	0.844
Uric acid, mg/dL, mean±SD	6.2±1.6	5.5±1.3	0.110
Creatinine clearance, mL/min, mean (range)	90.1 (62.5–127.1)	84.8 (30.1–105.0)	0.229
24 h protein in urine, mg/day, mean (range)	165 (51–7025)	126 (22–2005)	0.364
AST, U/L, mean (range)	17 (10–212)	19 (11–45)	0.388
ALT, U/L, mean (range)	15 (8–48)	16 (7–57)	0.540
Total cholesterol, mg/dL, mean±SD	207±42	181±37	**0.022**
LDL, mg/dL, mean±SD	124±32	105±28	**0.029**
HDL, mg/dL, mean (range)	54 (30–109)	47 (28–91)	0.359
Triglycerides, mg/dL, mean (range)	140 (30–290)	104 (55–363)	0.291
CAP, dB/m, mean (range)	255 (238–313)	211 (109–245)	**<0.001**
LSM, kPa, mean (range)	4.5 (2.8–7.5)	4.1 (3.3–8.0)	0.475

ALT: Alanine transaminase; AST: Aspartate transaminase; BMI: Body mass index; CAP: Controlled attenuation parameter; HbA1c: Hemoglobin A1c; HDL: High-density lipoprotein; LDL: Low-density lipoprotein; LSM: Liver stiffness measurement; MAFLD: Metabolic dysfunction-associated fatty liver disease; SD: Standard deviation. Significant p values are written in bold. Normally distributed data are presented as mean±standard deviation and analyzed with an independent samples t-test. Nonnormally distributed data are expressed as median (minimum–maximum) and analyzed using the Mann–Whitney U test. Categorical data are given as counts and percentages and compared with the Chi-squared test.

et al.[27] conducted a population-based study to investigate the impact of the acronym change from NAFLD to MAFLD in CKD patients. Accordingly, MAFLD was able to identify patients with CKD better than NAFLD. Having MAFLD and MAFLD with increased noninvasive fibrosis scores were independently associated with the presence of CKD. On the other hand, Deng et al.[28] demonstrated that the association between MAFLD and CKD was not independent but mediated by metabolic dysfunction parameters such as diabetes. Considering the strong association between CKD and NAFLD, it has been proposed that NAFLD may be associated with loss of graft function for KTRs.

In KTRs, gaining weight and developing metabolic dysfunction constitute a major problem.[7] The underlying mechanism is probably due to modern immunosuppressive medications and the use of corticosteroids which facilitates insulin resistance. Moreover, the therapy is also responsible for promoting oxidative stress and lipid peroxidation, causing dyslipidemia, hypertension, and dyslipidemia as the final effect.[7] The prevalence of metabolic syndrome among KTRs was reported to vary between 30% and 60%.[29] In our study, we found a prevalence of 35%. In light of this evidence, timely detection of the metabolic syndrome in KTRs and its complications con-

stitute a major clinical importance as the condition is a prominent risk factor for graft loss, cardiovascular morbidity, and mortality.[7]

The findings of this study must be seen considering some limitations. First, our study was conducted with a relatively small population. Therefore, the study results may not represent the general KTR population. Second, due to ethical reasons, we could not perform blood examinations in the control group. On the other hand, we defined hepatic steatosis using FibroScan, which is an accurate diagnostic tool in the detection of hepatic steatosis.[30]

Conclusion

In our study, we detected a high prevalence of MAFLD among KTRs. However, the prevalence of MAFLD was not significantly different from that in the normal population. Among KTRs, only age was found to be an independent predictor of MAFLD. Further studies with larger populations are required for the estimation of the hepatic burden among KTRs.

Ethics Committee Approval: The Kartal Lutfi Kirdar Training and Research Hospital Ethics Committee granted approval for this study (date: 19.12.2013, number: 89513307/1009/215).

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