Koşuyolu Heart Journal

Koşuyolu Heart J 2024;27(3):97–102
DOI: 10.51645/khj.2024.457

Original Article

Does Non-alcoholic Fatty Liver Disease Have an Impact on Contrast Induced Nephropathy and Adverse Events in Non-ST-elevation Myocardial Infarction?

Fatih Koca,[1] Ömer Furkan Demir,[1] Nur Özer Şensoy,[2] Barış Şensoy,[1] Nurcan Kat,[3] Fatih Levent,[1] Gökhan Özmen,[1] Mehmet Demir,[1] Erhan Tenekecioğlu[1]

[1]Department of Cardiology, Bursa Yüksek İhtisas Training and Research Hospital, Bursa, Türkiye
[2]Depertment of Nephrology, University of Health Science, Bursa City Hospital, Bursa, Türkiye
[3]Department of Radiology, Bursa Yüksek İhtisas Training and Research Hospital, Bursa, Türkiye

Abstract

Objectives: The primary aim of this study was to investigate the potential relationship between non-alcoholic fatty liver disease (NAFLD) and contrast-induced nephropathy (CIN) in patients with non-ST-elevation myocardial infarction (NSTEMI). As a secondary goal, we aimed to explore the impact of NAFLD on short-term adverse events and coronary artery disease (CAD) severity in patients with NSTEMI.

Methods: Three hundred and seven NSTEMI patients were included in this study. Laboratory analyses of these patients were performed before the procedure and 48–72 h after the procedure, and all patients underwent pre-procedure 2-dimensional transthoracic echocardiography and pre-discharge abdominal ultrasonography. The NAFLD (-) and (+) groups were compared statistically in terms of CIN, major cardiovascular-cerebrovascular adverse events (MACCE), and coronary artery severity (assessed by syntax score).

Results: The mean age of the 307 consecutive patients included in the study was 61.58±12.39 (min-max: 26–94). The rates of CIN (primary objective) and MACCE and syntax scores (secondary objective) were comparable in both groups.

Conclusion: In patients with NSTEMI, there was no relationship between NAFLD and CIN and short-term MACCE. Furthermore, NAFLD may not have an impact on the CAD severity in such patients. Based on these results, NAFLD is not a risk factor for CIN, short-term mortality, or CAD severity in NSTEMI patients.

Keywords: Contrast-induced nephropathy; hepatosteatosis; MACCE; NSTEMI; syntax score.

Cite This Article: Koca F, Demir ÖF, Şensoy NÖ, Şensoy B, Kat N, Levent F, et al. Does Non-alcoholic Fatty Liver Disease Have an Impact on Contrast Induced Nephropathy and Adverse Events in Non-ST-elevation? Koşuyolu Heart J 2024;27(3):97–102

Address for Correspondence:
Fatih Koca

Department of Cardiology, Bursa Yüksek İhtisas Training and Research Hospital, Bursa, Türkiye

E-mail: drfatihkoca@gmail.com

Submitted: June 14, 2024
Accepted: October 26, 2024
Available Online: December 06, 2024

Alkolsüz Yağlı Karaciğer Hastalığının ST Yükselmesiz Miyokard Enfarktüsünde Kontrast Nefropatisi ve Olumsuz Olaylara Bir Etkisi Var Mı?

Özet

Amaç: Bu çalışmanın temel amacı, ST yükselmesiz miyokard enfarktüsü (NSTEMI) hastalarında alkolik olmayan yağlı karaciğer hastalığı (AOYKH) ile kontrast nefropati (KN) arasındaki potansiyel ilişkiyi araştırmaktı. İkincil bir amaç olarak, NSTEMI hastalarında AOYKH'nin kısa vadeli olumsuz olaylar ve koroner arter hastalığı (KAH) şiddeti üzerindeki etkisini araştırmayı amaçladık.

Gereç ve Yöntem: Bu çalışmaya 307 NSTEMI hastası dahil edildi. Bu hastaların laboratuvar analizleri işlem öncesi ve işlem sonrası 48–72 saat içinde yapıldı ve tüm hastalara işlem öncesi 2 boyutlu transtorasik ekokardiyografi ve taburculuk öncesi batın ultrasonografisi yapıldı. AOYKH (-) ve (+) grupları KN, majör kardiyovasküler-serebrovasküler advers olaylar (MACCE) ve koroner arter şiddeti (Syntax skoru ile değerlendirilen) açısından istatistiksel olarak karşılaştırıldı.

Bulgular: Çalışmaya dahil edilen ardışık 307 hastanın yaş ortalaması 61,58±12,39 (min-maks: 26–94) idi. KN (birincil amaç) ve MACCE ve sözdizimi puanları (ikincil amaç) oranları her iki grupta da benzerdi.

Sonuç: NSTEMI hastalarında AOYKH ile KN ve kısa süreli MACCE arasında ilişki yoktu. Ayrıca AOYKH'nın bu tür hastalarda KAH şiddeti üzerinde bir etkisi olmayabilir. Bu sonuçlara göre AOYKH, NSTEMI hastalarında KN, kısa süreli mortalite veya KAH şiddeti için bir risk faktörü değildir.

Anahtar sözcükler: Kontrast nefropatisi; hepatosteatoz; MACCE; NSTEMI; syntax skoru.

Introduction

Liver steatosis that does not arise from excessive alcohol consumption is termed non-alcoholic fatty liver disease (NAFLD). NAFLD, the most common cause of chronic liver disease, is associated with metabolic syndrome and obesity. The global increase in these diseases has inflated the prevalence of NAFLD, currently estimated at 24%.[1,2]

Chronic kidney disease (CKD) is a prominent risk factor for cardiovascular disease (CVD).[3] There is plentiful evidence revealing an independent relationship between NAFLD and CKD.[4–8] The pathophysiological mechanisms that trigger NAFLD and CKD seem similar.[9] NAFLD is also associated with other metabolism-related diseases such as CVDs and diabetes mellitus. The newly proposed "cross-talk" concept may explain the relationship between all these diseases. Disrupted crosstalk can lead to loss of homeostatic balance and, hence, engendering organ damage. Contrast-induced nephropathy (CIN) is one of the most widespread causes of acute kidney damage in the hospital setting.[10]

In addition to studies displaying the presence and severity of coronary artery disease (CAD) in patients with NAFLD,[11,12] a limited number of studies have investigated the relationship between NAFLD and mortality in acute coronary syndrome.[13] However, the potential relationship between NAFLD and CIN remains unknown. The primary aim of this study was to investigate the possible relationship between NAFLD and CIN in patients with non-ST-elevation myocardial infarction (NSTEMI). Second, we aimed to explore the impact of NAFLD on short-term adverse events and CAD severity in patients with NSTEMI.

Materials and Methods

Study Population

This prospective and cross-sectional study was approved by the University of Health Sciences Bursa Yuksek Ihtisas Training and Research Hospital Ethics Committee (date: 16.11.2022; no: 2011-KAEK-25 2022/11-10). The exclusion criteria were as follows: non-NAFLD liver fat or chronic liver disease such as hepatitis cirrhosis and patients with chronic renal failure whose estimated GFR was below 30 mL/min. The study population comprised patients with NSTEMI admitted to our coronary care unit between December 2022 and July 2023. NSTEMI is defined as acute myocardial injury evidenced by abnormal cardiac biomarkers of acute myocardial ischemia without persistent ST elevation. Out of 355 consecutive patients, eight did not agree to participate in the study. Written consent was obtained from the remaining 347 patients who were included in the study. Of these patients, 23 were excluded from the study because they could not undergo angiography or refused angiography within the last minute. In addition, 17 patients were excluded from the study because their 1-month control examinations could not be performed; finally, 307 patients were included in the statistical analysis (Fig. 1). Patients were monitored for CIN and major adverse cardiovascular and cerebrovascular events (MACCE: cardiovascular death, unstable angina, myocardial infarction, stroke, unplanned revascularization, cardiovascular hospitalizations, and hemodialysis) during the in-hospital and post-discharge 1-month periods.

Laboratory Analysis

Fasting blood samples were collected from all patients before the procedure and were sent to the central laboratory of our hospital. The patients' fasting blood tests were repeated between 48 and 72 h after angiography and interventional procedures. CIN was defined as an increase in serum creatinine level by ≥0.5 mg/dL or more than 25% from the baseline within 48–72 h following the invasive procedure.

Echocardiographic and Ultrasonographic Evaluation

All patients underwent 2-dimensional (2D) transthoracic echocardiography before coronary angiography. Echocardiography was performed by a single echocardiographer blinded to the patient's data, and a Vivid E95 platform with a 3.5 Mhz transducer was used for the procedure software (GE Vingmed Ultrasound AS, Horten, Norway). Echocardiographic parameters were evaluated according to the recommendations of the American Society of Echocardiography guidelines.[14] Standard 2D, color, pulse, and continuous Doppler data were recorded while the patient was lying supine in the left decubitus position and during patient expiration. The modified Simpson method was used for the volumetric chamber and ejection fraction measurements.

All patients underwent abdominal ultrasonography by a radiologist blinded to the patients' data before discharge and after 8 h of fasting (3.5 MHz convex transducer, Toshiba USDI-790A, Otawara, Japan). Liver echogenicity was determined by comparison with the echogenicity of the right kidney. If the echogenicity of the kidney and liver was the same, this condition was considered normal and considered no NAFLD (grade 0). Liver steatosis was ultrasonographically divided into three groups as follows: mild (grade 1) with a slight diffuse increase in liver echogenicity, hepatic vessels, and diaphragm contours. Moderate (grade 2): moderate increase in liver echogenicity with slight distortions in the hepatic vessels and diaphragm boundaries. Advanced (grade 3): A significant increase in liver echogenicity, and hepatic vessels and diaphragm are not visible.[15,16]

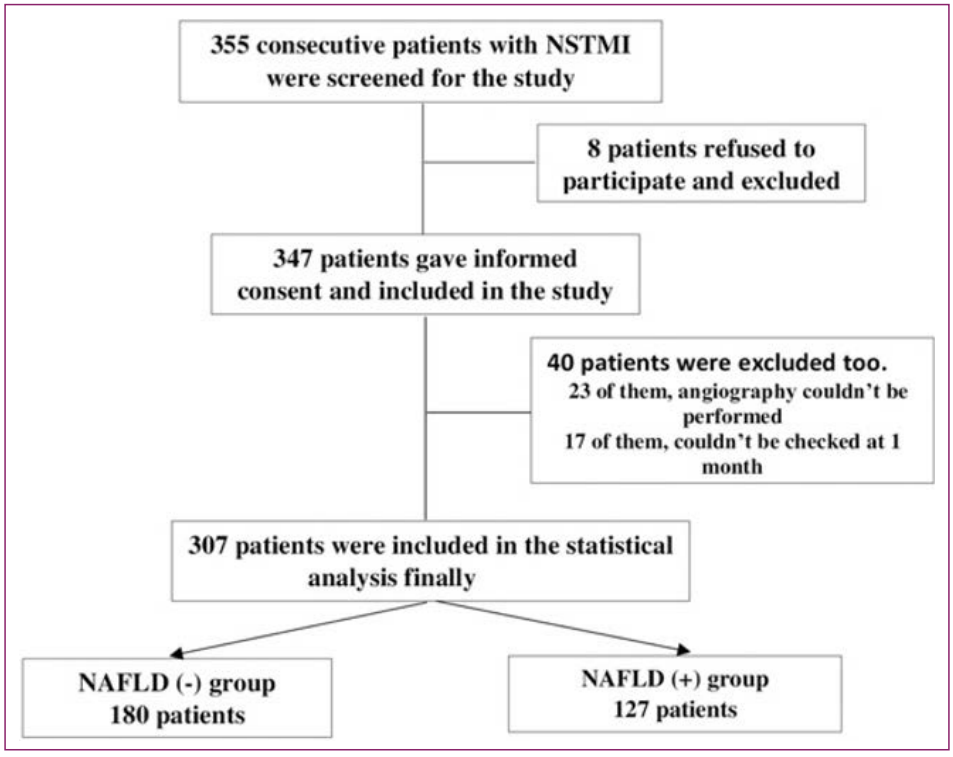

Figure 1. Study flowchart.

NSTMI: Non-ST segment myocardial infarction; NAFLD: Non-alcoholic fatty liver disease.

Angiography and Interventional Procedure

All patients included in the study underwent coronary angiography within 72 h of hospital admission, and if deemed necessary, percutaneous coronary intervention was performed in the same session. The decision for interventional procedure was left to the discretion of the invasive cardiologist performing the procedure. In these procedures, iopromide (Ultravist® 370 mg/mL, Berlin, Germany) was used as contrast agent. The syntax scores of the patients were obtained by two experienced invasive cardiologists who were unaware of the patients' clinical features, using a web-based online calculator (www.syntaxscore.com, version 2.1).

Statistical Analysis

All statistical analyses were performed using IBM SPSS Statistics (version 26.0; IBM Corp., Armonk, NY, USA). Patients were divided into two groups according to the presence or absence of NAFLD: NAFLD (-) and NAFLD (+). Continuous variables showing normal distribution according to the Kolmogorov–Smirnov test were expressed as mean±standard deviation, and those not showing normal distribution were expressed as median (interquartile range 25–75). Categorical variables were presented as numbers (n) and percentages (%). The independent Samples t-test was used to compare demographic, clinical, and procedure-related variables between the NAFLD (-) and NAFLD (+) groups for continuous variables showing normal distribution, the Mann–Whitney U-test was used for continuous variables not showing normal distribution, and the chi-square test was used to compare categorical variables. A $p<0.05$ was considered at significant.

Results

The mean age of the 307 consecutive patients included in the study was 61.58±12.39 years old (min-max: 26–94). NAFLD was observed in 127 (41.4%) patients. Of the 127 patients in the NAFLD (+) group, 88 (69%) had grade 1, 33 (26%) had grade 2, and 5 (3%) had Grade 3 hepatosteatosis. Demographic, drug, and echocardiographic variables of the two groups with and without NAFLD are shown in Table 1. MI, diabetes percentage, and metformin and insulin usage rates were statistically higher in the NAFLD (+) group ($p<0.05$).

On examination of the pre-procedure blood tests, HDL cholesterol was lower in the NAFLD (+) group, whereas ALT, triglycerides, HbA1C, albumin, and lymphocyte levels were significantly higher in this group ($p<0.05$). A comparison of the laboratory analysis results of the groups is presented in Table 2.

The rates of CIN (primary goal) and MACCE and syntax scores (secondary goal) were comparable in both study groups. Comparisons of patient angiographic and follow-up data between the groups are presented in Table 3.

Discussion

Regarding the relationship between NAFLD and CIN and short-term MACCE in patients with NSTEMI, no statistically significant difference was observed between subjects with and without NAFLD in terms of CIN and MACCE. To the best of our knowledge, this study is the first to explore the probable relationship between NAFLD and CIN in NSTEMI patients.

Table 1. Demographic, medication, and echocardiographic variables of NAFLD (-) and (+) groups

Variables	NAFLD (-) group n=180		NAFLD (+) group n=127		p
	n	%	n	%	
Male	128	71.1	80	63	0.183
Age, years, mean±SD	62.75±12.72		59.93±11.76		0.05
BMI, kg/m^2, mean±SD	27.80±4.88		30.57±5.27		**<0.005**
Diabetes	46	25.6	66	52	**<0.005**
Hypertension	111	61.7	84	66.1	0.422
Active smoking	70	38.9	53	41.7	0.683
ACE inh	128	71.1	92	72.4	0.799
Beta blocker	161	89.4	111	87.4	0.579
CCB	35	19.4	16	12.6	0.112
Statin	162	90	115	90.6	0.873
ASA	167	92.8	122	96.1	0.228
Klopidogrel	101	56.1	56	51.2	0.393
Ticagrelor	40	22.2	37	29.1	0.169
Metformin	26	14.4	36	28.3	**0.003**
Insulin	12	6.7	23	18.1	**0.002**
LAVI, median (25th–75th)	28.91 (22.37–40.52)		29.18 (22.22–36.86)		0.544
LVEDVI, median (25th–75th)	61.57 (50.64–77.97)		56.04 (47.05–72.68)		0.106
IVS, mm, median (25th–75th)	12 (10.25–13)		12 (11–149)		0.084
LVEF, %, mean±SD	48.14±12.65		49.83±12.68		0.250
Mitral E/A ratio, median (25th–75th)	0.94 (0.74–1.34)		0.89 (0.71–1.29)		0.370
Mitral E/e' ratio, median (25th–75th)	10.26 (8–16.37)		11.16 (8.37–14.5)		0.817
LV MPI, median (25th–75th)	0.51 (0.39–0.63)		0.50 (0.40–0.61)		0.773
RV FAC, mean±SD	42.88±7.99		44.27±6.99		0.116
TAPSE, mm, mean±SD	18.81±3.69		18.94±3.49		0.748
RV MPI, mean±SD	0.52±0.21		0.49±0.18		0.295
Tricuspid E/e' ratio, median (25th–75th)	4 (3.25–5.37)		4.27 (3.36–5.44)		0.374
PABs, mmHg, mean±SD	33.48±13.76		35.96±15.67		0.438
VCI CI, %, median (25th–75th)	53.33 (50–58.33)		53.33 (50–57.14)		0.704

NAFLD: Non-alcoholic fatty liver disease; SD: Standard deviation; BMI: Body mass index; ACE inh: Angiotensin-converting enzyme inhibitor; CCB: Calcium channel blocker; ASA: Acetylsalicylic acid; LAVI: Left atrial volume index; LVEDVI: Left ventricular end-diastolic volüme index; IVS: Interventricular septum; LVEF: Left ventricle ejection fraction; LV: Left ventricle; MPI: Myocardial performance index; RV: Right ventricle; FAC: Fractional area change; TAPSE: Tricuspid annular plane systolic excursion; PABs: Pulmoner artery systolic pressure; VCI: Vena cava inferior; CI: Collapsibility index.

Despite higher BMI and diabetes rates, the CIN rates in the NAFLD (+) group were comparable to those in the NAFLD (-) group. The rate of NAFLD found in 41.4% of all patients appears to be higher than that in epidemiological studies examining the entire population worldwide.[2,17] However, it is lower than the 48.3% rate detected in a study using ultrasonography on 113,239 patients who were healthy but applied to the hospital for check-ups in Turkey between 2007 and 2016, conducted by Değertekin et al.[18]

In 120 acute coronary syndrome patients, the 6-month mortality was higher in subjects with NAFLD, had a lower number of patients compared to our study, and also included patients with ST-segment elevation myocardial infarction (STEMI). During the literature review, we could not encounter any other study investigating the prognostic relationship between acute coronary syndrome and NAFLD.

Contrary to previous studies, in our study, no significant difference was detected between the groups with and without NAFLD, in terms of CAD severity, assessed using the syntax scoring in patients with NSTEMI. In a study conducted by Yılmaz et al.,[11] the rate of Grade 2 NAFLD in the CAD group, which is approximately twice (48.2%) that in our study (26%), may explain the difference in such outcomes. In that study, unlike ours, the severity of CAD was evaluated using the Gensini score. In another study with a limited number of patients with acute coronary syndrome (n=80), the syntax score was statistically higher in the NAFLD (+) group. [NAFLD (+) group: 18±8 vs. NAFLD (−) group: 11±5 (p<0.001)]. In that study, nearly half of the patients had STEMI, which was different from our study population, and the NAFLD (+) group included only 15 patients. Inci et al.[19] reported that in patients with chronic coronary syndrome, there was a meaningful correlation between moderate and advanced NAFLD and the severity of CAD using the Gensini score. Again, that was conducted on a limited number of patients (n=136) in a different study population, which may explain the differences from the results of our current study.

In a study by Boddi et al.,[20] in patients with non-diabetic STEMI, the prevalence of NAFLD was found to be considerably higher. NAFLD was found in 87% of all patients, which was about twice the prevalence rates in all epidemiological studies conducted so

Table 2. Laboratory analysis results of NAFLD (-) and (+) groups before and after the procedure

Variable	NAFLD (-) group n=180	NAFLD (+) group n=127	p
Basal kreatinin, mg/dL, median (25^{th}–75^{th})	0.81 (0.69–0.99)	0.82 (0.68–0.98)	0.565
Second kreatinin, mg/dL, median (25^{th}–75^{th})	0.96 (0.79–1.19)	0.99 (0.78–1.19)	0.954
Basal CrCl* mL/min, median (25^{th}–75^{th})	92.15 (73.37–103.45)	93.67 (77.31–104.57)	0.527
Second CrCl* mL/min, median (25^{th}–75^{th})	81.48 (59.24–97.21)	83.60 (59.15–96.27)	0.884
AST, IU/L, median (25^{th}–75^{th})	21 (17–30)	25 (18–36)	0.053
ALT, IU/L, median (25^{th}–75^{th})	17 (12–22)	22 (15–30)	**<0.005**
Total cholesterol, mg/dL, mean±SD	190.24±48.60	200.13±50.66	0.086
LDL, mg/dl, mean±SD	117.53±41.90	121.06±43.12	0.474
HDL, mg/dL, median(25^{th}–75^{th})	43.70 (37.97–51.85)	42 (34.2–49.8)	**0.027**
Triglyceride, mg/dL, mean±SD	113 (82–155.75)	152 (110–242)	**<0.005**
HbA1C, %, median (25^{th}–75^{th})	5.99 (5.49–6.49)	6.39 (5.69–8.28)	**<0.005**
Uric acid, median (25^{th}–75^{th})	5.5 (4.52–6.50)	5.6 (4.7–7)	0.255
Albumin, g/L, mean±SD	40.99±3.56	42.37±3.59	**0.001**
Hemoglobin, g/dL, mean±SD	13.70 (12.40–14.70)	13.90 (12.60–15.10)	0.167
Second hemoglobin, g/dL, mean±SD	12.90±1.95	13.12±1.95	0.066
White blood cell $\times10^3$/mL, mean±SD	9.19 (7.29–11.40)	9.6 (8.03–11.49)	0.191
Neutrophils $\times10^3$/mL, mean±SD	5.96 (4.43–7.85)	6.12 (4.93–7.80)	0.291
Lymphocytes $\times10^3$/mL, mean±SD	1.96 (1.64–2.80)	2.49 (1.63–3.11)	**0.015**
Platelet, $\times10^3$/L, mean±SD	240.50 (198–294.75)	242 (202–295)	0.905
TSH mIU/L, median (25^{th}–75^{th})	1.11 (0.69–1.84)	1.25 (0.72–1.77)	0.788
Troponin, ng/L, median (25^{th}–75^{th})	2005.45 (453.40–8499.82)	2069.90 (311.70–9489.60)	0.951
BNP, pg/mL, median (25^{th}–75^{th})	104.80 (35.17–367.70)	88.80 (22.7–248)	0.116
CRP, mg/L, median (25^{th}–75^{th})	3.83 (3.19–13.60)	4.44 (3.19–11.30)	0.863

*: Calculated with Modification of Diet in Renal Disease study. NAFLD: Non-alcoholic fatty liver disease; CrCl: Creatinine Clearance; AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; SD: Standard deviation; LDL: Low-density lipoprotein; HDL: High-density lipoprotein; HbA1C: Hemoglobin A1c; TSH: Thyroid-stimulating hormone; BNP: Brain natriuretic peptide; CRP: C-reactive protein.

Table 3. Angiographic variables and follow-up results of NAFLD (-) and (+) groups

Variables	NAFLD (-) group		NAFLD (+) group		p
	n	%	n	%	
Amount of contrast agent (mL), median (25^{th}–75^{th})	100 (60–150)		100 (60–200)		0.102
Syntax scores, median (25^{th}–75^{th})	6 (2–14)		6 (0–13)		0.257
Decision to medical treatment	65	36.1	40	31.5	NS
PCI counts	85	47.2	66	52	NS
Decision to CABG	30	16.7	21	16.5	NS
CIN	71	39.4	48	37.8	0.770
MACCE	13	7.2	12	9.4	0.482
In-hospital mortality	7	3.9	5	3.9	0.983
One month mortality	7	3.9	7	5.5	0.502

NAFLD: Non-alcoholic fatty liver disease; PCI: Percutaneous coronary intervention; CABG: Coronary artery by-pass grafting; CIN: Contrast induced nephropathy; MACCE: Major cardiovascular-cerebrovascular adverse events; NS: Non-significant.

far, and it was revealed that those with more severe NAFLD in STEMI patients were younger and had more severe CAD.

In a study conducted by Gholoobi et al.[21] on 296 patients with chronic coronary syndrome, an independent relationship was found between the prevalence and severity of NAFLD and CAD. In that study, the rate of more severe NAFLD in the CAD group was higher than that in the non-CAD group. The results may differ from those of our current study since the study was conducted on a different population.

In almost all of the studies mentioned above, the severity of CAD has been found to increase with advanced NAFLD degrees. Our study may differ from the above studies due to the high proportion of subjects with grade 1 NAFLD (69%). These results suggest that Grades 2 and 3 hepatosteatosis may be more influential for CRD, CVD, CIN, and MACCE. Therefore, further research with larger patient numbers, specifically those with higher Grade 2 and 3 NAFLD, may reveal any potential relationship between these diseases.

Study Limitations

The present study has some limitations. The biggest limitation is that patients were diagnosed with NAFLD, not by gold standard liver biopsy, but solely by ultrasound. In addition, the low

prevalence of subjects with Grade 2 and 3 NAFLD compared to those with Grade 1 NAFLD is another noteworthy limitation. It would be better for our study if the follow-up periods of the patients were longer, like 1-year instead of 1-month. Therefore, we plan to publish long-term (≥1-year) results in the near future.

Conclusion

In patients with NSTEMI, there seems to be no relationship between NAFLD, CIN, and short-term MACCE. Furthermore, NAFLD may not have an impact on the CAD severity in such patients. Based on these results, NAFLD is not a risk factor for CIN, short-term mortality, or CAD severity in NSTEMI patients. Further studies are required to confirm these results.

Disclosures

Ethics Committee Approval: The study was approved by the University of Health Sciences Bursa Yuksek Ihtisas Training and Research Hospital Ethics Committee (no: 2011-KAEK-25 2022/11-10, date: 16/11/2022).

Authorship Contributions: Concept – F.K., B.Ş., N.Ö.Ş., N.K.; Design – F.K., B.Ş., Ö.F.D.; Supervision – E.T., M.D.; Funding – F.K., F.L.; Data collection and/or processing – F.K., Ö.F.D., E.T., G.Ö., N.K.; Data analysis and/or interpretation – F.K., Ö.F.D., E.T.; Literature search – F.K., Ö.F.D., B.Ş., N.Ö.Ş., N.K.; Writing – F.K., B.Ş., Ö.F.D.; Critical review – M.D., E.T., B.Ş.

Conflict of Interest: All authors declared no conflict of interest.

Use of AI for Writing Assistance: No AI technologies utilized.

Financial Disclosure: The authors declared that this study received no financial support.

Peer-review: Externally peer-reviewed.

References

1. Mikolasevic I, Milic S, Turk Wensveen T, Grgic I, Jakopcic I, Stimac D, et al. Nonalcoholic fatty liver disease - A multisystem disease? World J Gastroenterol 2016;22(43):9488–505. doi: 10.3748/wjg.v22.i43.9488.
2. El Hadi H, Di Vincenzo A, Vettor R, Rossato M. Cardio-metabolic disorders in non-alcoholic fatty liver disease. Int J Mol Sci 2019;20(9):2215. doi: 10.3390/ijms20092215.
3. McCullough K, Sharma P, Ali T, Khan I, Smith WC, MacLeod A, et al. Measuring the population burden of chronic kidney disease: A systematic literature review of the estimated prevalence of impaired kidney function. Nephrol Dial Transplant 2012;27(5):1812–21. doi: 10.1093/ndt/gfr547.
4. Targher G, Chonchol MB, Byrne CD. CKD and nonalcoholic fatty liver disease. Am J Kidney Dis 2014;64(4):638–52. doi: 10.1053/j.ajkd.2014.05.019.
5. Baqai K, Mahmud M, Anwar M, Ali S, Hussain N, Wasim A. Association of low gfr and non-alcoholic hepatosteatosis on fibroscan of liver. J Ayub Med Coll Abbottabad 2023;35(1):60–3. doi: 10.55519/JAMC-01-11051.
6. Chang Y, Ryu S, Sung E, Woo HY, Oh E, Cha K, et al. Nonalcoholic fatty liver disease predicts chronic kidney disease in nonhypertensive and nondiabetic Korean men. Metabolism 2008;57(4):569–76. doi: 10.1016/j.metabol.2007.11.022.
7. Jia G, Di F, Wang Q, Shao J, Gao L, Wang L, et al. Non-alcoholic fatty liver disease is a risk factor for the development of diabetic nephropathy in patients with type 2 diabetes mellitus. PLoS One 2015;10(11):e0142808. doi: 10.1371/journal.pone.0142808
8. Sinn DH, Kang D, Jang HR, Gu S, Cho SJ, Paik SW, et al. Development of chronic kidney disease in patients with non-alcoholic fatty liver disease: A cohort study. J Hepatol 2017;67(6):1274–80. doi: 10.1016/j.jhep.2017.08.024.
9. Musso G, Cassader M, Cohney S, De Michieli F, Pinach S, Saba F, et al. Fatty liver and chronic kidney disease: Novel mechanistic insights and therapeutic opportunities. Diabetes Care 2016;39(10):1830–45. doi: 10.2337/dc15-1182.
10. McCullough PA, Adam A, Becker CR, Davidson C, Lameire N, Stacul F, et al; CIN Consensus Working Panel. Epidemiology and prognostic implications of contrast-induced nephropathy. Am J Cardiol 2006;98(6A):5K–13K. doi: 10.1016/j.amjcard.2006.01.019.
11. Yılmaz A, Yılmaz F, Beydilli İ, Sönmez BM, Duyan M, Özdemir M, et al. Ultrasonographically detected hepatosteatosis independently predicts the presence and severity of coronary artery disease. Afr Health Sci 2022;22(2):273–85. doi: 10.4314/ahs.v22i2.31.
12. Agaç MT, Korkmaz L, Cavusoglu G, Karadeniz AG, Agaç S, Bektas H, et al. Association between nonalcoholic fatty liver disease and coronary artery disease complexity in patients with acute coronary syndrome: A pilot study. Angiology 2013;64(8):604–8. doi: 10.1177/0003319713479155.
13. Perera N, Indrakumar J, Abeysinghe WV, Fernando V, Samaraweera WM, Lawrence JS. Non alcoholic fatty liver disease increases the mortality from acute coronary syndrome: An observational study from Sri Lanka. BMC Cardiovasc Disord 2016;16:37. doi: 10.1186/s12872-016-0212-8.
14. Rudski LG, Lai WW, Afilalo J, Hua L, Handschumacher MD, Chandrasekaran K, et al. Guidelines for the echocardiographic assessment of the right heart in adults: A report from the American Society of Echocardiography endorsed by the European Association of Echocardiography, a registered branch of the European Society of Cardiology, and the Canadian Society of Echocardiography. J Am Soc Echocardiogr 2010;23(7):685–713; quiz 786–8. doi: 10.1016/j.echo.2010.05.010.
15. Osawa H, Mori Y. Sonographic diagnosis of fatty liver using a histogram technique that compares liver and renal cortical echo amplitudes. J Clin Ultrasound 1996;24(1):25–9. doi: 10.1002/(SICI)1097-0096(199601)24:1<25::AID-JCU4>3.0.CO;2-N.
16. el-Hassan AY, Ibrahim EM, al-Mulhim FA, Nabhan AA, Chammas MY. Fatty infiltration of the liver: Analysis of prevalence, radiological and clinical features and influence on patient management. Br J Radiol 1992;65(777):774–8. doi: 10.1259/0007-1285-65-777-774.
17. Vernon G, Baranova A, Younossi ZM. Systematic review: The epidemiology and natural history of non-alcoholic fatty liver disease and non-alcoholic steatohepatitis in adults. Aliment Pharmacol Ther 2011;34(3):274–85. doi: 10.1111/j.1365-2036.2011.04724.x.
18. Değertekin B, Tozun N, Demir F, Söylemez G, Parkan Ş, Gürtay E, et al. The changing prevalence of non-alcoholic fatty liver disease (NAFLD) in Turkey in the last decade. Turk J Gastroenterol 2021;32(3):302–12. doi: 10.5152/tjg.2021.20062.
19. Inci MF, Özkan F, Ark B, Vurdem ÜE, Ege MR, Sincer I, et al. Sonographic evaluation for predicting the presence and severity of coronary artery disease. Ultrasound Q 2013;29(2):125–30. doi: 10.1097/RUQ.0b013e318291580e.
20. Boddi M, Tarquini R, Chiostri M, Marra F, Valente S, Giglioli C, et al. Non-alcoholic fatty liver in nondiabetic patients with acute coronary syndromes. Eur J Clin Invest 2013;43(5):429–38. doi: 10.1111/eci.12065.
21. Gholoobi A, Gifani M, Gholoobi A, Akhlaghi S, Pezeshki Rad M, Baradaran Rahimi V. Relationship between the prevalence and severity of non-alcoholic fatty liver disease and coronary artery disease: Findings from a cross-sectional study of a referral center in northeast Iran. JGH Open 2022;6(5):330–7. doi: 10.1002/jgh3.12746.