HEPATOLOGY FORUM

doi: 10.14744/hf.2023.2023.0042

From NAFLD to MASLD: Meta-analysis and systematic review of NAFLD patients in Turkiye in terms of metabolic profile and MASLD potential

Sevginur Akdas[1], Nuray Yazihan[1,2]

[1]Department of Metabolism and Clinical Nutrition, Institute of Health Sciences, Interdisciplinary Food, Ankara University, Ankara, Turkiye; [2]Department of Pathophysiology, Ankara University School of Medicine, Ankara, Turkiye

Abstract

Non-alcoholic Fatty Liver Disease (NAFLD) is both a cause and a consequence of metabolic disturbances. Consequently, the disease term has recently changed to Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD). Turkiye is one of the leading countries with high incidences of diseases such as diabetes, obesity, metabolic syndrome, and fatty liver. This study aims to identify the metabolic parameters and MASLD potential of NAFLD in Turkiye. All NAFLD studies conducted in Turkiye were systematically searched using the keywords "fatty liver disease" AND " Turkiye " on PubMed, Scopus, and Web of Science databases. A total of 2653 articles were scanned, and 120 studies were eligible for meta-analysis. The metabolic parameters were meta-analyzed from a broad perspective. According to the meta-analysis results, there were significant increases in waist circumferences (mean difference: 10.90, p<0.00001), HOMA-IR (mean difference: 2.13, p<0.00001), aspartate aminotransferase (AST) (mean difference: 17.82, p<0.00001), systolic blood pressure (SBP) (mean difference: 5.86, p<0.00001), and C-reactive protein (CRP) levels (mean difference: 0.95, p<0.00001). These parameters are representative biochemical findings of disturbed glucose metabolism, lipid profile, blood pressure, and acute phase response mechanisms. Furthermore, the analysis of all related parameters commonly found among the articles confirmed these metabolic dysfunctions. NAFLD is a metabolic disease that encompasses multiple pathways related to glucose and lipid metabolism, vascular function, inflammation, and acute phase responses. Additionally, our results suggest that Turkish NAFLD patients identified in previous studies mostly have MASLD. This is the first meta-analysis study indicating changes in metabolism-related parameters with a cumulative meta-analysis of all Turkish NAFLD studies.

Keywords: Diabetes; fatty liver disease; hypertension; inflammation; lipid profile; metabolism.

How to cite this article: Akdas S, Yazihan N. From NAFLD to MASLD: Meta-analysis and systematic review of NAFLD patients in Turkiye in terms of metabolic profile and MASLD potential. Hepatology Forum 2024; 5(3):126–138.

Received: August 17, 2023; **Revised:** January 23, 2024; **Accepted:** February 27, 2024; **Available online:** July 02, 2024

Corresponding author: Nuray Yazihan; Ankara Universitesi Tip Fakultesi, Patofizyoloji Klinigi, Ankara, Turkiye
Phone: +90 312 595 80 56; **e-mail:** nurayyazihan@yahoo.com

Hepatology Forum - Available online at www.hepatologyforum.org

Introduction

Non-alcoholic fatty liver disease (NAFLD) is defined as the presence of ≥5% hepatic steatosis without a competing liver disease such as viral hepatitis, autoimmune hepatitis, hemochromatosis, Wilson's disease, or alcoholic liver disease, and without the use of steatosis-inducing medications. Non-alcoholic steatohepatitis (NASH)[1] occurs with histopathological findings that cause hepatic damage, fibrosis, cirrhosis, and mortality in a smaller subset of patients with NAFLD.[2] Furthermore, with the participation and agreement of 236 panelists from 56 countries, new medical terms were introduced to the scientific field. Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD) is the new designation for NAFLD and is defined by the detection of liver steatosis (via liver histology, non-invasive biomarkers, or imaging) together with at least one of three criteria: overweight or obesity, type 2 diabetes mellitus, or clinical evidence of metabolic dysfunction such as high waist circumference or abnormal lipid or glycemic profiles. Similarly, the term for nonalcoholic steatohepatitis was changed to "metabolic-associated steatohepatitis" (MASH) to refer to steatohepatitis patients with metabolic dysfunctions.[3,4] Also, diagnostic criteria were updated. In the presence of steatotic liver disease (SLD), the identification of any cardiometabolic risk factor alone would lead to a diagnosis of MASLD, provided no other causes of hepatic steatosis are evident. If additional contributors to steatosis are discovered, it suggests a combination etiology. Specifically, in cases involving alcohol, it is referred to as MASLD with increased alcohol intake (MetALD). In situations where explicit cardiometabolic criteria are absent, other potential causes must be ruled out. If none are found, this is categorized as cryptogenic SLD. However, depending on clinical judgment, it could also be considered as a possible MASLD, warranting periodic reassessment on a case-by-case basis.[4]

NAFLD is a significant burden of health problems that cause chronic liver diseases worldwide. A very recent meta-analysis examined the up-to-date incidence of NAFLD with data from 1,201,807 individuals across 63 studies. According to this global analysis, the incidence of NAFLD was found to be 4,613 per 100,000 person-years, particularly high in men, with a dramatic increase of more than threefold between 2000 and 2015.[5] According to regional results in 2019, NAFLD occurs in 31.29% of the Middle East, 30.45% of South America, 27.37% of Asia, 24.13% of North America, 23.71% of Europe, and 13.48% of Africa.[6] Nearly 30% of the world's population is currently challenged with this health problem.[7]

In America, the number of NAFLD patients, which was 83 million in 2015, is expected to increase to 100.9 million by 2030, a 21% increase, while the prevalence of NASH cases will increase by 63% from 16.52

million to 27.00 million cases.[8] The prevalence of NAFLD is estimated to be 20%–30% in the European Union, and about 3% is NASH. The advanced fibrosis incidence in NASH patients was 67.95 in 1,000 person-years. Liver-specific mortality in the pooled NAFLD versus non-NAFLD incidence rate ratio was found to be 1.94. The adjusted liver-specific mortality hazard ratio for NAFLD patients was 2.60. Although the prevalence of advanced fibrosis among NAFLD patients in the USA and Europe was 10–15%, fibrosis development was found to be lower in the Asia region compared to Western countries.[9] In Turkiye, multi-center prevalence studies are limited in showing the current NAFLD status. However, recent published data pointed to an alarming prevalence of 48.3%, which seems reasonable when compared with the obesity prevalence in Turkiye.[10]

NAFLD is a part of the metabolic syndrome hepatic outcomes and is commonly seen in obese and diabetic patients. Whether NAFLD is a cause or consequence of insulin resistance has been debated for a long time. On the other hand, "lean-NAFLD" can be seen in non-obese subjects, especially in low-income countries or rural areas.[11] This meta-analysis aimed to evaluate all NAFLD cases and their control data in the literature to show the metabolic profile of the disease in Turkiye cumulatively for the first time. The MASLD potential of these patients was discussed according to meta-analysis results.

Materials and Methods

Study Design

To determine the metabolic profile of Turkish NAFLD patients, all NAFLD studies conducted in Turkiye were systematically searched using the keywords "fatty liver disease" and "Turkiye" on PubMed, Scopus, and Web of Science databases. All characteristics and biochemical data were screened and collected for related meta-analysis. Inclusion criteria were established as providing suitable data (using international units) of biochemical parameters for NAFLD-diagnosed patients and a healthy control group.

The parameters of NAFLD diagnosis were generally based on ultrasound screening. Many studies confirmed ultrasound screening results with histopathological examinations after liver biopsy and used elevated liver enzyme levels as inclusion criteria. Exclusion criteria were generally similar across studies, excluding individuals with viral hepatitis, hemochromatosis, Wilson's disease, autoimmune hepatitis, primary biliary cirrhosis, sclerosing cholangitis, biliary obstruction, α1-antitrypsin deficiency, ischemic cardiac or cerebrovascular disease, impaired renal function, malignancies, previous abdominal surgery, medication use, and daily alcohol intake exceeding 20 g/day. Several studies also excluded chronic conditions such as coronary artery disease, acute chronic renal failure, hypertension, and diabetes. Some studies set the alcohol intake exclusion limit at 30 or 40 g/day. Detailed information about the inclusion and exclusion criteria of each study was given in Appendix 1.

There were no additional restrictions for individual characteristics. The systematic search continued until July 2023. PRISMA statement guidelines were followed for this meta-analysis. Since this article is a meta-analysis, ethics committee approval is not required.

Statistical Analysis

Cumulative data analysis was conducted to show the metabolic comorbidities of Turkish NAFLD patients. All analysis procedures were performed according to the Cochrane Handbook (cochrane.org/handbook). Mean and standard deviation values of each marker that was cumulatively assessed were entered into the RevMan 5.3 program. Weighted

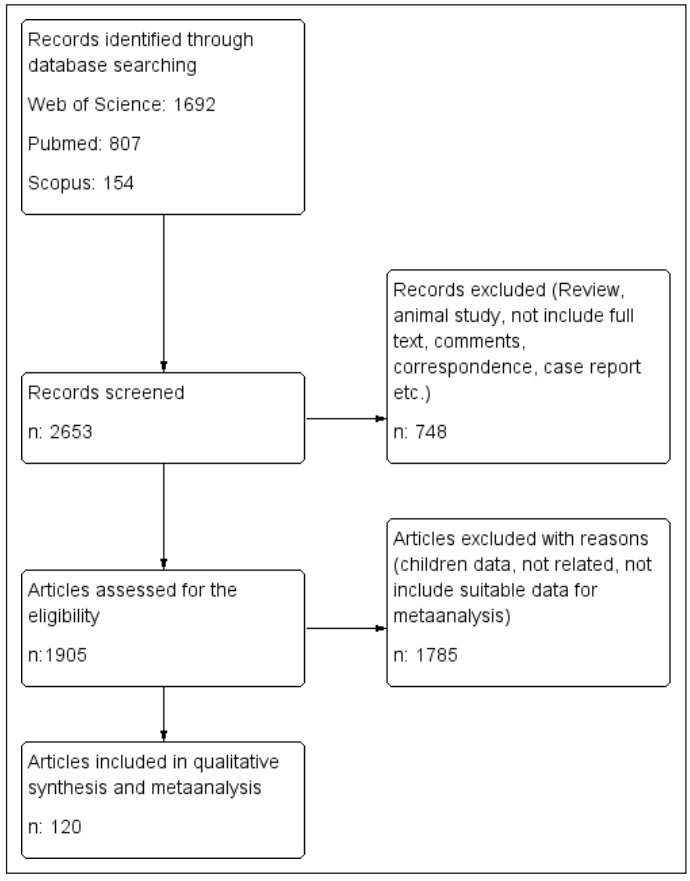


Figure 1. Flow diagram of study selection.

analysis was automatically performed by RevMan 5.3 according to the power of articles. Study power is calculated by RevMan, based on values for effect size magnitude, sample size, the number of studies, and the amount of between-study variability. The I2 was used to measure heterogeneity, which can be seen at the bottom of each figure. I2% values of 0–25, 25–50, 50–75, and 75–100 represent no, low, moderate, and high heterogeneity, respectively. The fixed and random effect models were used according to the heterogeneity tau2 value to combine the results. If the tau2 value is found as 0, the fixed effect model can be used. However, in all our results, the tau2 value was found to be different from 0, which led us to use the random effect model for a better assessment. RevMan 5.3 (Cochrane Collaboration, Copenhagen, 2014) software was used for the meta-analysis, and GraphPad Prism 6 software was used for correlation analysis and visualizing the results.

Results

In total, 2653 articles were scanned. As a result of the screening, 2533 studies were found ineligible for this meta-analysis. The remaining 120 studies were eligible for meta-analysis, and all data on patient and control groups from these studies[10,12–130] were evaluated (Fig. 1).

Obesity-Glucose Metabolism Related Parameters

Data from 14138 NAFLD and 15335 healthy individuals showed that the BMI level is significantly higher in the NAFLD group (Mean difference: 3.48, 95% CI: [3.02, 3.94], p<0.00001). Waist circumference in the NAFLD group (n=4650) was increased compared to the control group

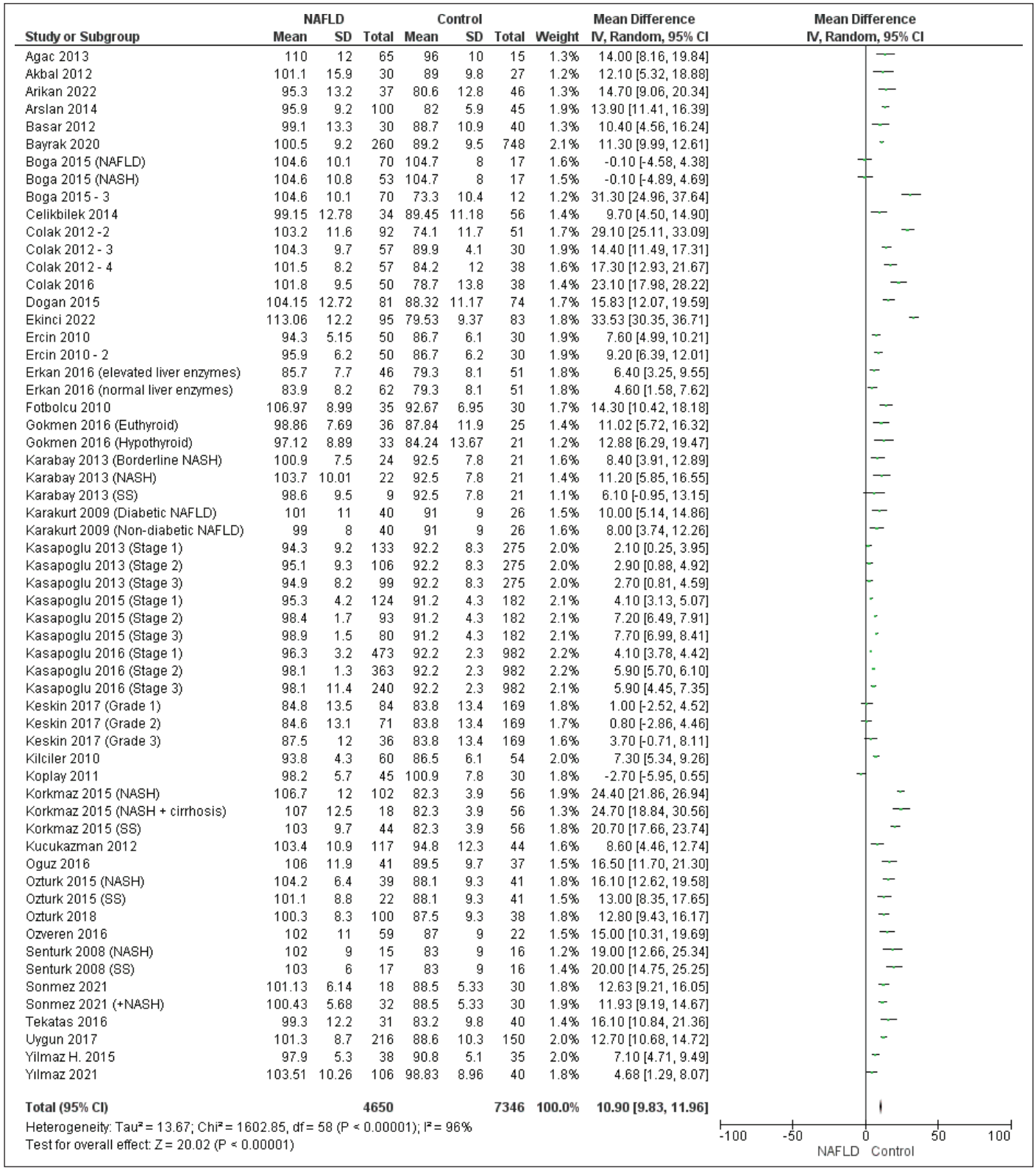


Figure 2. The random effect model of cumulative meta-analysis for waist circumference data obtained from NAFLD and control individuals.

(n=7346) (Mean difference: 10.90 cm, 95% CI: [9.83, 11.96], p<0.00001) (Fig. 2). Data from 6769 NAFLD and 7646 healthy individuals showed that fasting blood glucose levels were higher in the NAFLD group (Mean difference: 12.32 mg/dl, 95% CI: [9.96, 14.69], p<0.00001). HbA1c% values were higher in the NAFLD group (n=1254) than in the control group (n=1327) (Mean difference: 0.52, 95% CI: [0.28, 0.76], p<0.0001).

Study or Subgroup	NAFLD Mean	NAFLD SD	NAFLD Total	Control Mean	Control SD	Control Total	Weight	Mean Difference IV, Random, 95% CI
Akbal 2012	4.5	3.2	30	2.2	1.9	27	0.8%	2.30 [0.95, 3.65]
Akkiz 2021	4.62	6.58	200	1.29	0.32	61	1.1%	3.33 [2.41, 4.25]
Arslan 2014	3.6	3.1	100	0.95	0.36	45	1.3%	2.65 [2.03, 3.27]
Aygun 2008	5.8	4.6	40	1.94	0.88	40	0.8%	3.86 [2.41, 5.31]
Aygun 2014 (elevated liver enzymes)	7.1	1.2	31	1.8	0.2	20	1.4%	5.30 [4.87, 5.73]
Aygun 2014 (normal liver enzymes)	7.9	1.7	20	1.8	0.2	20	1.2%	6.10 [5.35, 6.85]
Boga 2015 (NAFLD)	5.3	3.9	70	3.4	1.4	17	1.0%	1.90 [0.77, 3.03]
Boga 2015 (NASH)	5.9	4.2	53	3.4	1.4	17	0.9%	2.50 [1.19, 3.81]
Boga 2015 - 3	5.2	3.8	70	1.9	1	12	1.0%	3.30 [2.25, 4.35]
Cengiz 2009	3.63	3.24	76	1.6	0.47	24	1.2%	2.03 [1.28, 2.78]
Cetindagli 2017	3.22	1.3	93	1.54	0.47	37	1.4%	1.68 [1.38, 1.98]
Colak 2011	3.2	1.2	60	1.2	0.8	52	1.4%	2.00 [1.63, 2.37]
Colak 2012	2.8	1.86	50	1.35	0.53	28	1.3%	1.45 [0.90, 2.00]
Colak 2012 - 4	4.21	4.1	92	1.35	0.53	51	1.1%	2.86 [2.01, 3.71]
Colak 2016	2.7	1.8	50	0.94	0.5	38	1.3%	1.76 [1.24, 2.28]
Ekinci 2022	7.56	7.95	95	2.48	1.76	83	0.7%	5.08 [3.44, 6.72]
Eminler 2014	5.81	0.88	40	2.04	0.21	40	1.4%	3.77 [3.49, 4.05]
Fotbolcu 2010	3.59	1.51	35	1.28	0.29	30	1.3%	2.31 [1.80, 2.82]
Gokmen 2016 (Euthyroid)	2.62	1.43	36	1.39	0.53	25	1.3%	1.23 [0.72, 1.74]
Gokmen 2016 (Hypothyroid)	2.61	1.3	33	1.34	0.67	21	1.3%	1.27 [0.74, 1.80]
Gulsen 2005	5.58	1.67	71	2.53	1.07	30	1.3%	3.05 [2.50, 3.60]
Kara 2013	3.4	2.3	103	1.5	0.8	57	1.4%	1.90 [1.41, 2.39]
Karabay 2013 (Borderline NASH)	2.7	1.41	24	0.96	0.26	21	1.3%	1.74 [1.17, 2.31]
Karabay 2013 (NASH)	3.98	2.6	22	0.96	0.26	21	1.0%	3.02 [1.93, 4.11]
Karabay 2013 (SS)	1.61	0.93	9	0.96	0.26	21	1.3%	0.65 [0.03, 1.27]
Karakurt 2009 (Diabetic NAFLD)	3.4	2.6	40	1.5	0.9	26	1.1%	1.90 [1.02, 2.78]
Karakurt 2009 (Non-diabetic NAFLD)	1.7	1.2	40	1.5	0.9	26	1.3%	0.20 [-0.31, 0.71]
Karaoğullarından 2023	4.43	5.88	290	1.29	0.38	108	1.2%	3.14 [2.46, 3.82]
Kasapoglu 2013 (Stage 1)	2.1	0.5	133	1.4	0.8	275	1.5%	0.70 [0.57, 0.83]
Kasapoglu 2013 (Stage 2)	2.4	0.4	106	1.4	0.8	275	1.5%	1.00 [0.88, 1.12]
Kasapoglu 2013 (Stage 3)	2.4	0.3	99	1.4	0.8	275	1.5%	1.00 [0.89, 1.11]
Kasapoglu 2015 (Stage 1)	2.6	0.9	124	1.4	0.7	182	1.5%	1.20 [1.01, 1.39]
Kasapoglu 2015 (Stage 2)	3.2	0.9	93	1.4	0.7	182	1.5%	1.80 [1.59, 2.01]
Kasapoglu 2015 (Stage 3)	3.5	1	80	1.4	0.7	182	1.5%	2.10 [1.86, 2.34]
Kasapoglu 2015 - 2 (Stage 1)	2.6	0.9	88	1.5	1.4	136	1.4%	1.10 [0.80, 1.40]
Kasapoglu 2015 - 2 (Stage 2)	3.6	1.1	38	1.5	1.4	136	1.4%	2.10 [1.68, 2.52]
Kasapoglu 2015 - 2 (Stage 3)	3.7	1	24	1.5	1.4	136	1.4%	2.20 [1.74, 2.66]
Kasapoglu 2016 (Stage 1)	2.6	0.9	473	1.5	1.4	982	1.5%	1.10 [0.98, 1.22]
Kasapoglu 2016 (Stage 2)	3.6	1.1	363	1.5	1.4	982	1.5%	2.10 [1.96, 2.24]
Kasapoglu 2016 (Stage 3)	3.7	1	240	1.5	1.4	982	1.5%	2.20 [2.05, 2.35]
Korkmaz 2015 (NASH)	4.9	3.1	102	2.5	1	56	1.3%	2.40 [1.74, 3.06]
Korkmaz 2015 (NASH + cirrhosis)	7.7	3.9	18	2.5	1	56	0.6%	5.20 [3.38, 7.02]
Korkmaz 2015 (SS)	3.8	3.2	44	2.5	1	56	1.1%	1.30 [0.32, 2.28]
Kucukazman 2014	3.45	3.29	154	2.85	2.73	57	1.1%	0.60 [-0.28, 1.48]
Kutlu 2019	4.1	2.8	51	1.6	0.78	30	1.2%	2.50 [1.68, 3.32]
Oral 2019	2.6	1.61	225	1.71	0.77	142	1.5%	0.89 [0.64, 1.14]
Oral 2019 - 2	2.6	1.61	225	1.71	0.77	142	1.5%	0.89 [0.64, 1.14]
Ozturk 2015 (NASH)	4.5	3.2	39	2.1	0.9	41	1.0%	2.40 [1.36, 3.44]
Ozturk 2015 (SS)	3.9	2.4	22	2.1	0.9	41	1.0%	1.80 [0.76, 2.84]
Ozturk 2018	4.5	2.6	100	2.1	0.9	38	1.3%	2.40 [1.82, 2.98]
Ozveren 2016	3.9	2.2	59	1.5	0.5	22	1.3%	2.40 [1.80, 3.00]
Purnak 2012	2.7	0.8	50	2.6	0.88	26	1.4%	0.10 [-0.30, 0.50]
Sapmaz 2016	3.39	3	176	1.95	1.6	90	1.3%	1.44 [0.89, 1.99]
Sargin 2005	3.3	1.5	35	2.1	1.1	34	1.3%	1.20 [0.58, 1.82]
Senates 2011	3.78	2.41	88	1.5	0.8	88	1.3%	2.28 [1.75, 2.81]
Senates 2012	3.7	2.6	97	1.5	0.8	66	1.3%	2.20 [1.65, 2.75]
Senturk 2008 (NASH)	6	2.2	15	2.5	0.4	16	1.0%	3.50 [2.37, 4.63]
Senturk 2008 (SS)	3.9	1.1	17	2.5	0.4	16	1.3%	1.40 [0.84, 1.96]
Tekatas 2016	2.7	3.4	31	0.8	0.7	40	0.9%	1.90 [0.68, 3.12]
Ulaşoğlu 2021	3.9	3.5	175	1.5	0.6	74	1.3%	2.40 [1.86, 2.94]
Uygun 2017	4.6	5.4	216	2.2	1	150	1.2%	2.40 [1.66, 3.14]
Yalniz 2006	7	9.2	37	1.75	0.67	25	0.3%	5.25 [2.27, 8.23]
Yesilova 2005	4.39	0.22	46	2.45	0.28	30	1.5%	1.94 [1.82, 2.06]
Yilmaz Y. 2009	4.7	3.4	40	1.1	0.7	14	1.0%	3.60 [2.48, 4.72]
Yilmaz Y. 2010	3.5	2.2	59	1.4	0.4	77	1.3%	2.10 [1.53, 2.67]
Yilmaz Y. 2010 - 2 (Borderline NASH)	3.8	1.9	17	1.2	0.6	58	1.1%	2.60 [1.68, 3.52]
Yilmaz Y. 2010 - 2 (NASH)	3.3	1.7	26	1.2	0.6	58	1.2%	2.10 [1.43, 2.77]

Mean Difference IV, Random, 95% CI

Continued on next page →

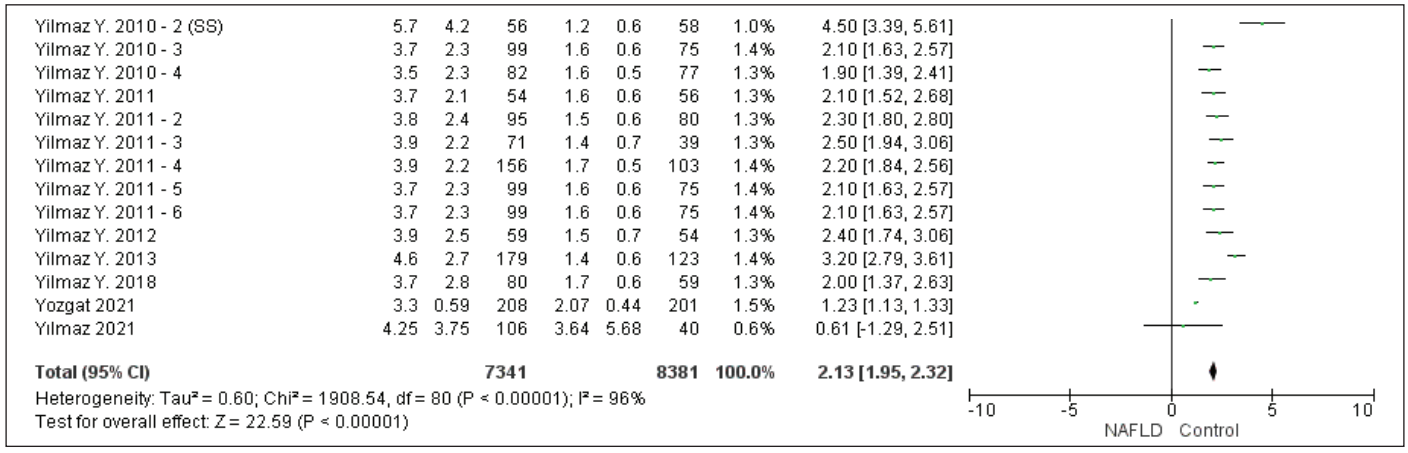


Figure 3. The random effect model of cumulative meta-analysis for HOMA-IR data obtained from NAFLD and control individuals.

Insulin levels were also higher in the NAFLD group (n=3194) compared to the control group (n=1881) (Mean difference: 6.73, 95% CI: [5.94, 7.53], p<0.00001). The HOMA-IR values showed a significant increase in the NAFLD group (n=7341) compared to the control group (n=8381) (Mean difference: 2.13, 95% CI: [1.95, 2.32], p<0.00001) (Fig. 3).

Liver Function Parameters

AST values of the NAFLD group (n=9357) were higher than those of the control group (n=11080) (mean difference: 17.82 IU/L, 95% CI: [15.47, 20.17], p<0.00001) (Fig. 4). Similarly, ALT levels in the NAFLD group (n=12535) were increased compared to the healthy group (n=14434) (mean difference: 35.11 IU/L, 95% CI: [31.27, 38.95], p<0.00001). Increased ALP levels were observed in NAFLD patients (n=2615) compared to healthy controls (n=4452) (mean difference: 12.10 IU/L, 95% CI: [8.38, 15.83], p<0.00001). GGT levels in the NAFLD group (n=5756) were higher than in the control group (n=7634) (mean difference: 21.73, 95% CI: [19.35, 24.10], p<0.00001). No significant difference was found in total bilirubin levels between the NAFLD group (n=830) and control group (n=735) (mean difference: 0.07, 95% CI: [-0.01, 0.16], p=0.10). Similarly, albumin levels showed no significant difference between groups (NAFLD group n=1994, control group n=1752) (mean difference: -0.02, 95% CI: [-0.09, 0.05], p=0.55).

Hyperlipidemia Related Parameters

Increased levels of triglycerides were found in NAFLD patients (n=9052) compared to healthy individuals (n=10489) (Mean difference: 49.34 mg/dl, 95% CI: [44.24, 54.44], p<0.00001). HDL levels of the NAFLD group (n=9097) were lower than those of the control group (n=10522) (Mean difference: -2.59 mg/dl, 95% CI: [-3.86, -1.32], p<0.0001). LDL levels of the NAFLD group (n=8695) were higher than those of the control group (n=10249) (Mean difference: 13.52, 95% CI: [10.94, 16.10], p<0.00001). Total cholesterol levels of NAFLD patients (n=8823) were also increased compared to controls (n=9699) (Mean difference: 22.59, 95% CI: [18.94, 26.24], p<0.00001).

Blood Pressure Parameters

Systolic blood pressure (SBP) was higher in NAFLD patients (n=3778) compared to controls (n=2987) (mean difference: 5.86 mmHg, 95% CI: [5.39, 8.14], p<0.00001) (Fig. 5). Diastolic blood pressure was also increased in NAFLD patients (n=3778) compared to controls (n=2987) (mean difference: 3.83 mmHg, 95% CI: [2.55, 5.11], p<0.00001).

Acute Phase Reactants

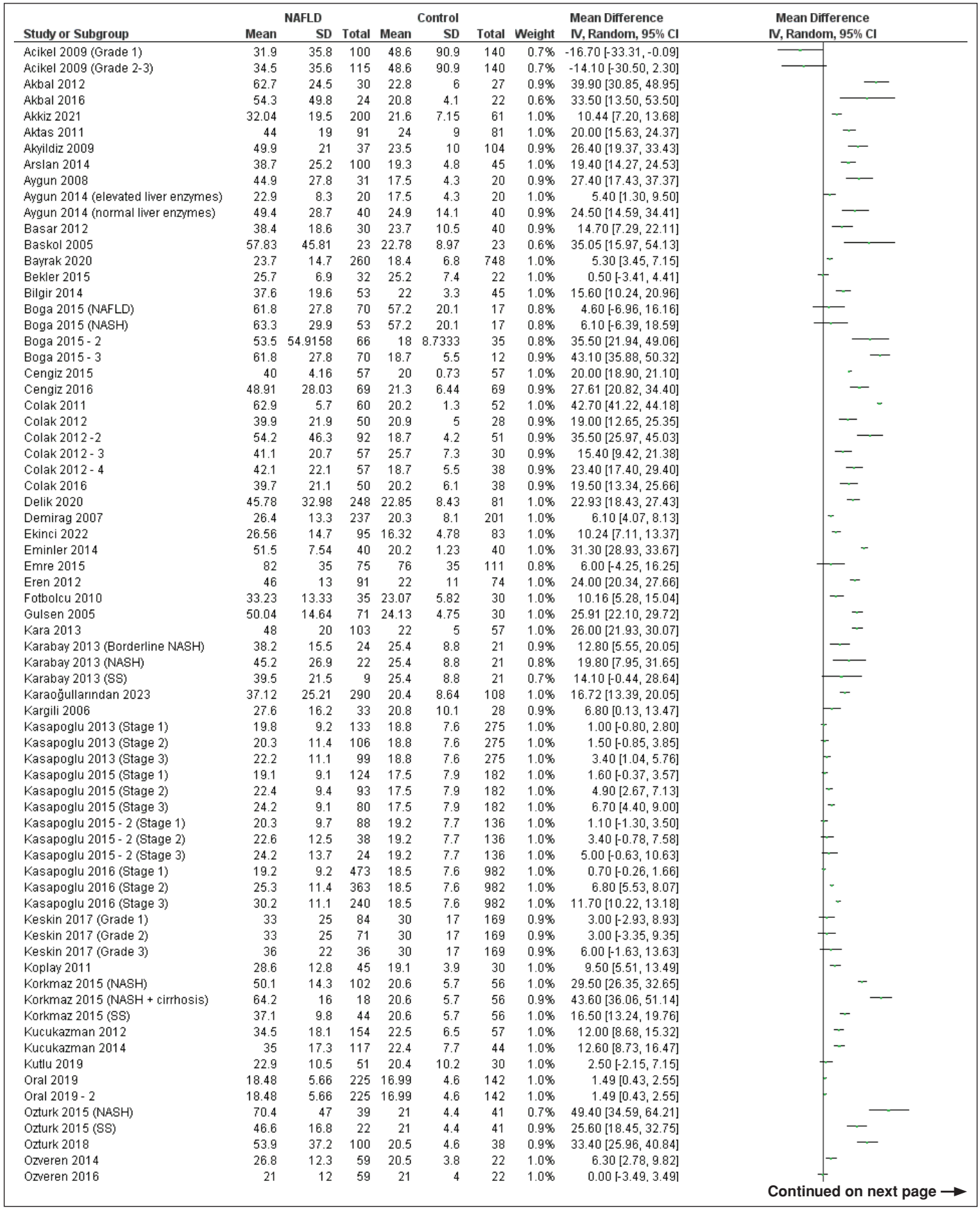
CRP values of the NAFLD group (n=3765) were higher than those of the control group (n=5859) (Mean difference: 0.95 mg/L, 95% CI: [0.72, 1.19], p<0.00001) (Fig. 6. ESR was prolonged in the NAFLD group (n=786) compared to the healthy group (n=482) (mean difference: 2.35 mm/hr, 95% CI: [0.47, 4.23], p<0.01). Ferritin levels in NAFLD patients (n=1921) were increased compared to the control group (n= 3812) (Mean difference: 45.63 ng/mL, 95% CI: [32.72, 58.54], p<0.00001). Hemoglobin levels were also higher in the NAFLD group (n=398) than in the control group (n=780) (Mean difference: 0.28, %95 CI: [0.12, 0.43], p=0.0004). Serum creatinine levels of NAFLD patients (n=2650) were higher than those of healthy controls (n=2479) (mean difference: 0.07 mg/dL, 95% CI: [0.05, 0.09], p<0.00001).

Correlation Results

Correlation analysis indicated that obesity and glucose metabolism parameters such as fasting blood glucose, waist circumference, insulin, and HOMA-IR levels were associated with liver function, as evidenced by increases in ALT, AST, and GGT enzyme levels. Fasting blood glucose correlated with AST (p<0.0001, r=0.401), ALT (p<0.0001, r=0.276), and GGT (p=0.018, r=0.245). Waist circumference levels were found to be correlated with AST (p<0.0001, r=0.371), ALT (p<0.0001, r=0.368), and ALP (p=0.04, r=0.50). Similarly, insulin/HOMA-IR levels correlated with AST (p=0.001/p<0.0001, r=0.342/0.760), ALT (p<0.0001/p<0.0001, r=0.369/0.710), and GGT levels (p=0.017/p<0.0001, r=0.289/0.495).

Discussion

Our meta-analysis showed that Turkish NAFLD patients have glucose metabolism disorders, hyperlipidemia, and impaired liver functions compared to the control group. Blood pressure values were elevated in NAFLD patients. Furthermore, CRP, ESR, Ferritin, Hemoglobin, and Creatinine levels, which were determined as acute phase reactants, were elevated in NAFLD patients in Turkiye. These results suggest that NAFLD patients in Turkiye carry a high risk of metabolic dysfunction and that Turkish NAFLD patients detected in previous studies might mostly have MASLD.



Study or Subgroup	NAFLD Mean	NAFLD SD	NAFLD Total	Control Mean	Control SD	Control Total	Weight	Mean Difference IV, Random, 95% CI
Acikel 2009 (Grade 1)	31.9	35.8	100	48.6	90.9	140	0.7%	-16.70 [-33.31, -0.09]
Acikel 2009 (Grade 2-3)	34.5	35.6	115	48.6	90.9	140	0.7%	-14.10 [-30.50, 2.30]
Akbal 2012	62.7	24.5	30	22.8	6	27	0.9%	39.90 [30.85, 48.95]
Akbal 2016	54.3	49.8	24	20.8	4.1	22	0.6%	33.50 [13.50, 53.50]
Akkiz 2021	32.04	19.5	200	21.6	7.15	61	1.0%	10.44 [7.20, 13.68]
Aktas 2011	44	19	91	24	9	81	1.0%	20.00 [15.63, 24.37]
Akyildiz 2009	49.9	21	37	23.5	10	104	0.9%	26.40 [19.37, 33.43]
Arslan 2014	38.7	25.2	100	19.3	4.8	45	1.0%	19.40 [14.27, 24.53]
Aygun 2008	44.9	27.8	31	17.5	4.3	20	0.9%	27.40 [17.43, 37.37]
Aygun 2014 (elevated liver enzymes)	22.9	8.3	20	17.5	4.3	20	1.0%	5.40 [1.30, 9.50]
Aygun 2014 (normal liver enzymes)	49.4	28.7	40	24.9	14.1	40	0.9%	24.50 [14.59, 34.41]
Basar 2012	38.4	18.6	30	23.7	10.5	40	0.9%	14.70 [7.29, 22.11]
Baskol 2005	57.83	45.81	23	22.78	8.97	23	0.6%	35.05 [15.97, 54.13]
Bayrak 2020	23.7	14.7	260	18.4	6.8	748	1.0%	5.30 [3.45, 7.15]
Bekler 2015	25.7	6.9	32	25.2	7.4	22	1.0%	0.50 [-3.41, 4.41]
Bilgir 2014	37.6	19.6	53	22	3.3	45	1.0%	15.60 [10.24, 20.96]
Boga 2015 (NAFLD)	61.8	27.8	70	57.2	20.1	17	0.8%	4.60 [-6.96, 16.16]
Boga 2015 (NASH)	63.3	29.9	53	57.2	20.1	17	0.8%	6.10 [-6.39, 18.59]
Boga 2015 - 2	53.5	54.9158	66	18	8.7333	35	0.8%	35.50 [21.94, 49.06]
Boga 2015 - 3	61.8	27.8	70	18.7	5.5	12	0.9%	43.10 [35.88, 50.32]
Cengiz 2015	40	4.16	57	20	0.73	57	1.0%	20.00 [18.90, 21.10]
Cengiz 2016	48.91	28.03	69	21.3	6.44	69	0.9%	27.61 [20.82, 34.40]
Colak 2011	62.9	5.7	60	20.2	1.3	52	1.0%	42.70 [41.22, 44.18]
Colak 2012	39.9	21.9	50	20.9	5	28	0.9%	19.00 [12.65, 25.35]
Colak 2012 -2	54.2	46.3	92	18.7	4.2	51	0.9%	35.50 [25.97, 45.03]
Colak 2012 - 3	41.1	20.7	57	25.7	7.3	30	0.9%	15.40 [9.42, 21.38]
Colak 2012 - 4	42.1	22.1	57	18.7	5.5	38	0.9%	23.40 [17.40, 29.40]
Colak 2016	39.7	21.1	50	20.2	6.1	38	0.9%	19.50 [13.34, 25.66]
Delik 2020	45.78	32.98	248	22.85	8.43	81	1.0%	22.93 [18.43, 27.43]
Demirag 2007	26.4	13.3	237	20.3	8.1	201	1.0%	6.10 [4.07, 8.13]
Ekinci 2022	26.56	14.7	95	16.32	4.78	83	1.0%	10.24 [7.11, 13.37]
Eminler 2014	51.5	7.54	40	20.2	1.23	40	1.0%	31.30 [28.93, 33.67]
Emre 2015	82	35	75	76	35	111	0.8%	6.00 [-4.25, 16.25]
Eren 2012	46	13	91	22	11	74	1.0%	24.00 [20.34, 27.66]
Fotbolcu 2010	33.23	13.33	35	23.07	5.82	30	1.0%	10.16 [5.28, 15.04]
Gulsen 2005	50.04	14.64	71	24.13	4.75	30	1.0%	25.91 [22.10, 29.72]
Kara 2013	48	20	103	22	5	57	1.0%	26.00 [21.93, 30.07]
Karabay 2013 (Borderline NASH)	38.2	15.5	24	25.4	8.8	21	0.9%	12.80 [5.55, 20.05]
Karabay 2013 (NASH)	45.2	26.9	22	25.4	8.8	21	0.8%	19.80 [7.95, 31.65]
Karabay 2013 (SS)	39.5	21.5	9	25.4	8.8	21	0.7%	14.10 [-0.44, 28.64]
Karaoğullarından 2023	37.12	25.21	290	20.4	8.64	108	1.0%	16.72 [13.39, 20.05]
Kargili 2006	27.6	16.2	33	20.8	10.1	28	0.9%	6.80 [0.13, 13.47]
Kasapoglu 2013 (Stage 1)	19.8	9.2	133	18.8	7.6	275	1.0%	1.00 [-0.80, 2.80]
Kasapoglu 2013 (Stage 2)	20.3	11.4	106	18.8	7.6	275	1.0%	1.50 [-0.85, 3.85]
Kasapoglu 2013 (Stage 3)	22.2	11.1	99	18.8	7.6	275	1.0%	3.40 [1.04, 5.76]
Kasapoglu 2015 (Stage 1)	19.1	9.1	124	17.5	7.9	182	1.0%	1.60 [-0.37, 3.57]
Kasapoglu 2015 (Stage 2)	22.4	9.4	93	17.5	7.9	182	1.0%	4.90 [2.67, 7.13]
Kasapoglu 2015 (Stage 3)	24.2	9.1	80	17.5	7.9	182	1.0%	6.70 [4.40, 9.00]
Kasapoglu 2015 - 2 (Stage 1)	20.3	9.7	88	19.2	7.7	136	1.0%	1.10 [-1.30, 3.50]
Kasapoglu 2015 - 2 (Stage 2)	22.6	12.5	38	19.2	7.7	136	1.0%	3.40 [-0.78, 7.58]
Kasapoglu 2015 - 2 (Stage 3)	24.2	13.7	24	19.2	7.7	136	1.0%	5.00 [-0.63, 10.63]
Kasapoglu 2016 (Stage 1)	19.2	9.2	473	18.5	7.6	982	1.0%	0.70 [-0.26, 1.66]
Kasapoglu 2016 (Stage 2)	25.3	11.4	363	18.5	7.6	982	1.0%	6.80 [5.53, 8.07]
Kasapoglu 2016 (Stage 3)	30.2	11.1	240	18.5	7.6	982	1.0%	11.70 [10.22, 13.18]
Keskin 2017 (Grade 1)	33	25	84	30	17	169	0.9%	3.00 [-2.93, 8.93]
Keskin 2017 (Grade 2)	33	25	71	30	17	169	0.9%	3.00 [-3.35, 9.35]
Keskin 2017 (Grade 3)	36	22	36	30	17	169	0.9%	6.00 [-1.63, 13.63]
Koplay 2011	28.6	12.8	45	19.1	3.9	30	1.0%	9.50 [5.51, 13.49]
Korkmaz 2015 (NASH)	50.1	14.3	102	20.6	5.7	56	1.0%	29.50 [26.35, 32.65]
Korkmaz 2015 (NASH + cirrhosis)	64.2	16	18	20.6	5.7	56	0.9%	43.60 [36.06, 51.14]
Korkmaz 2015 (SS)	37.1	9.8	44	20.6	5.7	56	1.0%	16.50 [13.24, 19.76]
Kucukazman 2012	34.5	18.1	154	22.5	6.5	57	1.0%	12.00 [8.68, 15.32]
Kucukazman 2014	35	17.3	117	22.4	7.7	44	1.0%	12.60 [8.73, 16.47]
Kutlu 2019	22.9	10.5	51	20.4	10.2	30	1.0%	2.50 [-2.15, 7.15]
Oral 2019	18.48	5.66	225	16.99	4.6	142	1.0%	1.49 [0.43, 2.55]
Oral 2019 - 2	18.48	5.66	225	16.99	4.6	142	1.0%	1.49 [0.43, 2.55]
Ozturk 2015 (NASH)	70.4	47	39	21	4.4	41	0.7%	49.40 [34.59, 64.21]
Ozturk 2015 (SS)	46.6	16.8	22	21	4.4	41	0.9%	25.60 [18.45, 32.75]
Ozturk 2018	53.9	37.2	100	20.5	4.6	38	0.9%	33.40 [25.96, 40.84]
Ozveren 2014	26.8	12.3	59	20.5	3.8	22	1.0%	6.30 [2.78, 9.82]
Ozveren 2016	21	12	59	21	4	22	1.0%	0.00 [-3.49, 3.49]

Continued on next page →

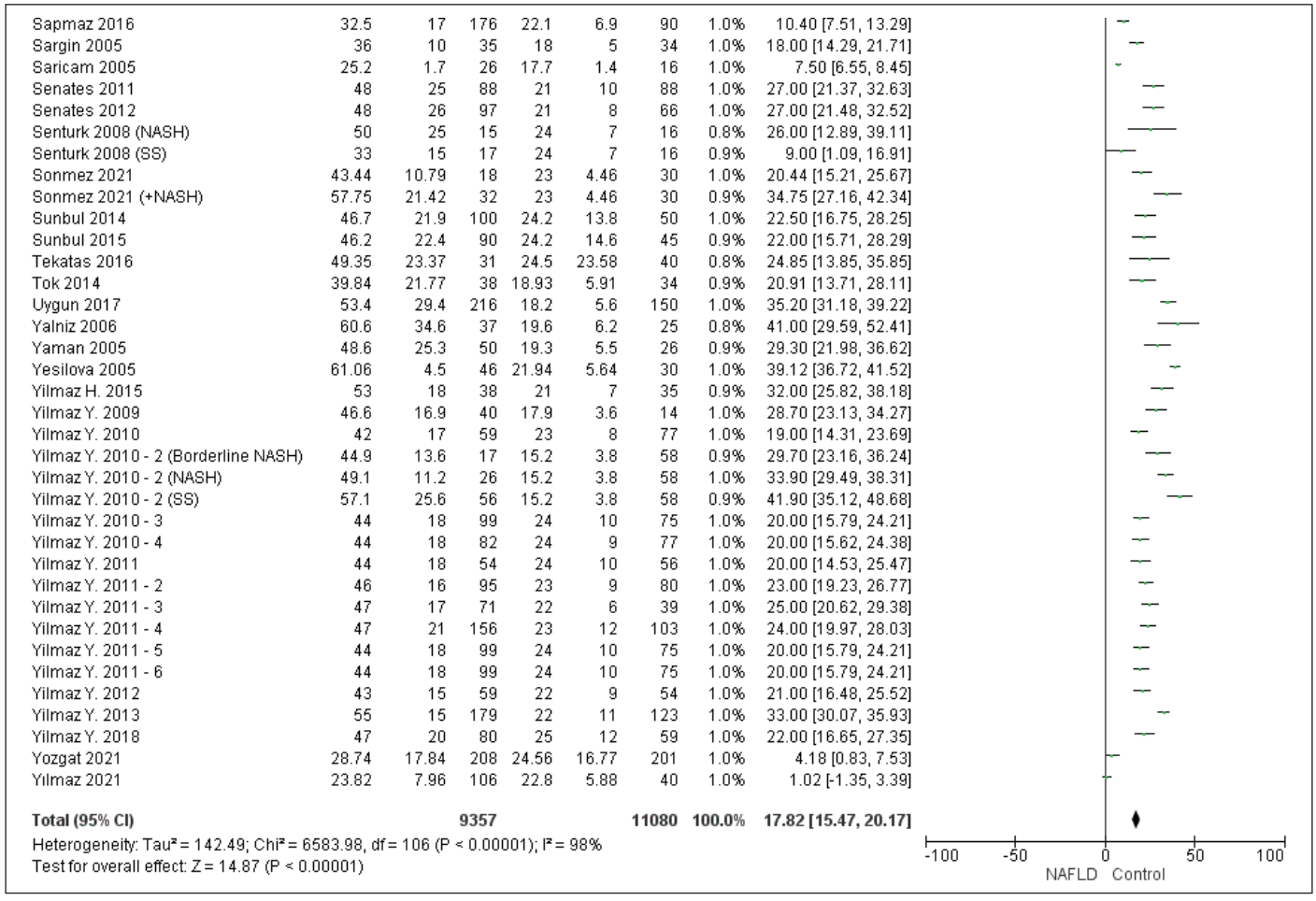


Figure 4. The random effect model of cumulative meta-analysis for AST data obtained from NAFLD and control individuals.

The pathogenesis of NAFLD, specifically whether NAFLD precedes insulin resistance or vice versa, has been debated for a long time. Diacylglycerol is recognized as a key factor of lipid-induced insulin resistance in the liver. Elevated diacylglycerol activates protein kinase C, which phosphorylates and inhibits the insulin receptor, thereby impairing glucose metabolism in NAFLD primarily through this mechanism.[131]

The global prevalence of NAFLD is 30%,[7] and a 2016 meta-analysis reported a pooled analysis for NASH prevalence at 59.10% among biopsied NAFLD patients. According to comorbidity analysis, the prevalence of obesity was 51.34%/81.83%, diabetes was 22.51%/43.63%, hyperlipidemia was 69.16%/72.13%, hypertriglyceridemia was 40.74%/83.33%, hypertension was 39.34%/67.97%, and metabolic syndrome was 42.54%/70.65% among NAFLD/NASH patients worldwide. These results indicate that the prevalence of comorbidities rises with the development of NASH compared to NAFLD without steatohepatitis.[8] However, a 2023 meta-analysis showed that the incidence of NAFLD was higher among those with obesity, diabetes, hyperlipidemia, and metabolic syndrome, though the differences were not significant. Only tobacco use status showed significant incidence differences among patient characteristics.[5]

A single-center study investigating Turkiye's NAFLD profile revealed that 90.4% of NAFLD patients had biopsy-proven NASH, and simple steatosis was rare (9.6%). The clinical outcomes indicated that significant fibrosis was present in 6.4%, advanced fibrosis in 32.6%, and cirrhosis in 61% of patients. Overweight (32.6%), obesity (61%), diabetes (33.5%), and metabolic syndrome (63%) were frequently seen comorbidities in these patients. This may be because this hospital is a tertiary referral center, and Fibroscan is commonly used to indicate biopsy.[9] These results provide evidence that NAFLD/NASH is an epidemic in Turkiye. A study conducted in five different centers in the East-Southeastern Anatolia Regions of Turkiye showed that 85% were overweight, 37% were obese, 18% had type 2 diabetes mellitus, and 80.6% had hyperlipidemia. According to multivariate regression analysis, age, diabetes, and aspartate aminotransferase were related to the severity of the disease.[24]

Is It "Non-Alcoholic Fatty Liver Disease" or "Metabolic Dysfunction Associated Steatotic Liver Disease"?

Our results show that NAFLD is not solely a liver-based disease; it is both a cause and consequence of metabolic disturbances. Insulin resistance and glucose metabolism-related parameters support this hypothesis. After many critical meetings, authorities agreed that MASLD is a more appropriate overarching term. This new designation integrates the current understanding of patient heterogeneity encompassed by the acronym NAFLD and offers terminology suggestions that more accurately reflect the pathogenesis. It is believed that this new term will accelerate the transition to novel treatments and will facilitate sub-phenotyping efforts of the disease with future studies.[4,132,133]

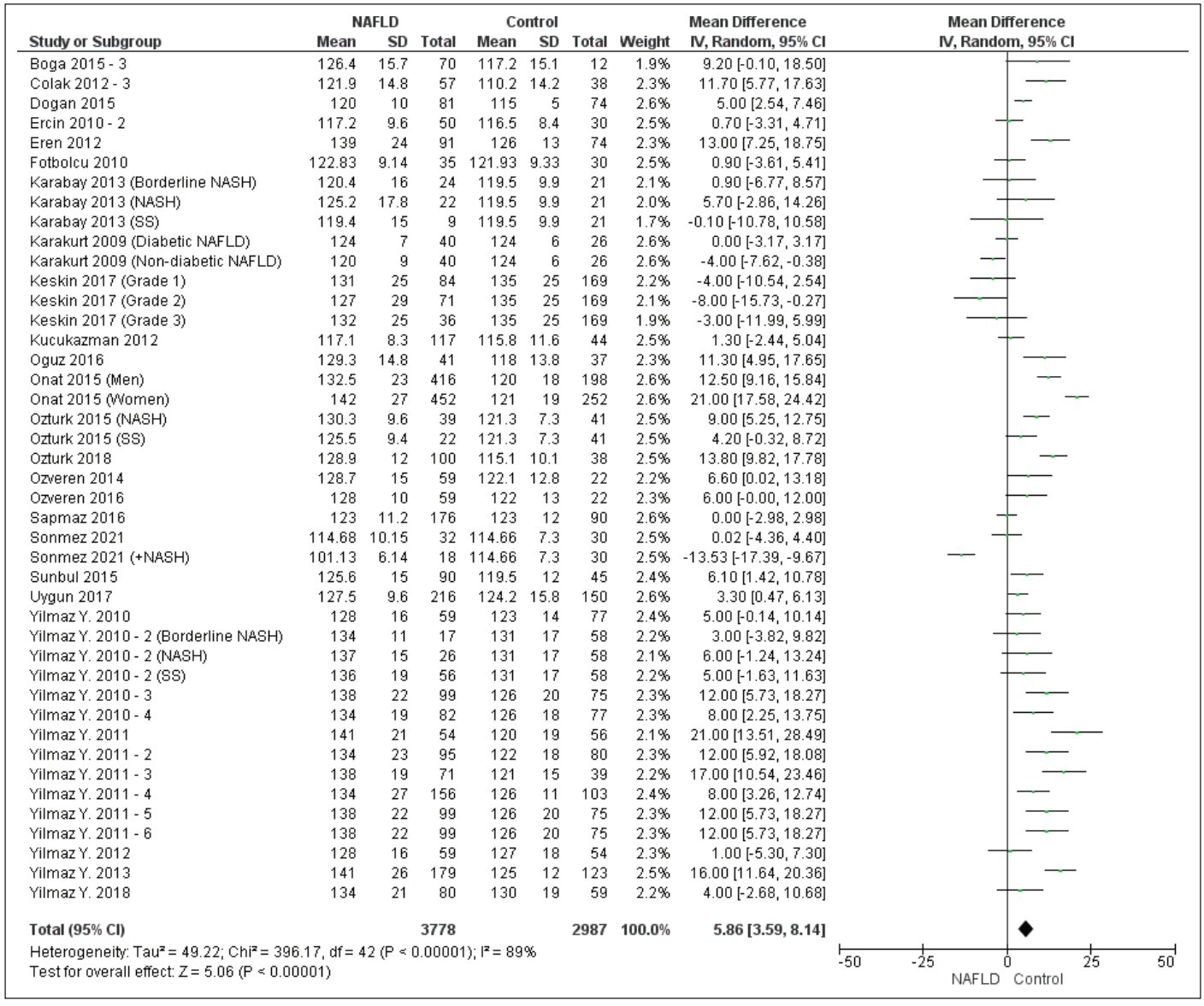


Figure 5. The random effect model of cumulative meta-analysis for Systolic Blood Pressure (SBP) data obtained from NAFLD and control individuals.

A recent meta-analysis involving cohorts from the USA, Japan, and Turkiye revealed negative implications of type 2 diabetes in relation to NAFLD. The study found that participants with type 2 diabetes had a significantly elevated risk of hepatic decompensation at 1, 3, and 5 years compared to those without type 2 diabetes. After considering various confounding factors, it was determined that type 2 diabetes and glycated hemoglobin were independent predictors of hepatic decompensation. Furthermore, even after adjusting for baseline liver stiffness assessed by magnetic resonance elastography, the association between type 2 diabetes and hepatic decompensation remained consistent. Notably, type 2 diabetes emerged as an independent predictor of hepatocellular carcinoma development.[134]

Another recent meta-analysis aimed to explore the relationship between the triglyceride and glucose (TyG) index, calculated as fasting triglyceride divided by fasting glucose, and the risk of NAFLD. The results revealed a positive and linear association between the TyG index and the risk of NAFLD. Each additional unit of the TyG index was associated with a higher risk of NAFLD, with a summary odds ratio (OR) of 2.84.[135]

The findings of our meta-analysis, combined with results from other studies, emphasize the importance of assessing metabolic parameters in understanding the development and prognosis of NAFLD. This highlights the need for countries with a high incidence of NAFLD, such as Turkiye, to focus on developing metabolic approaches for the treatment and monitoring of these conditions. By emphasizing metabolic factors, healthcare professionals can better manage and address the challenges posed by NAFLD.

Our meta-analysis has for the first time indicated the overall metabolic profile and MASLD potential of NAFLD patients in Turkiye. While acknowledging the limitations of our work due to the quality of the studies and data in the literature, we recognize several specific constraints. Some limitations stem from the characteristics of the fatty liver patients, the design of the studies, and the procedures of the centers where they were performed, affecting the determination of the disease or patients' states and introducing heterogeneity. High statistical heterogeneity of the data was observed. Additionally, we

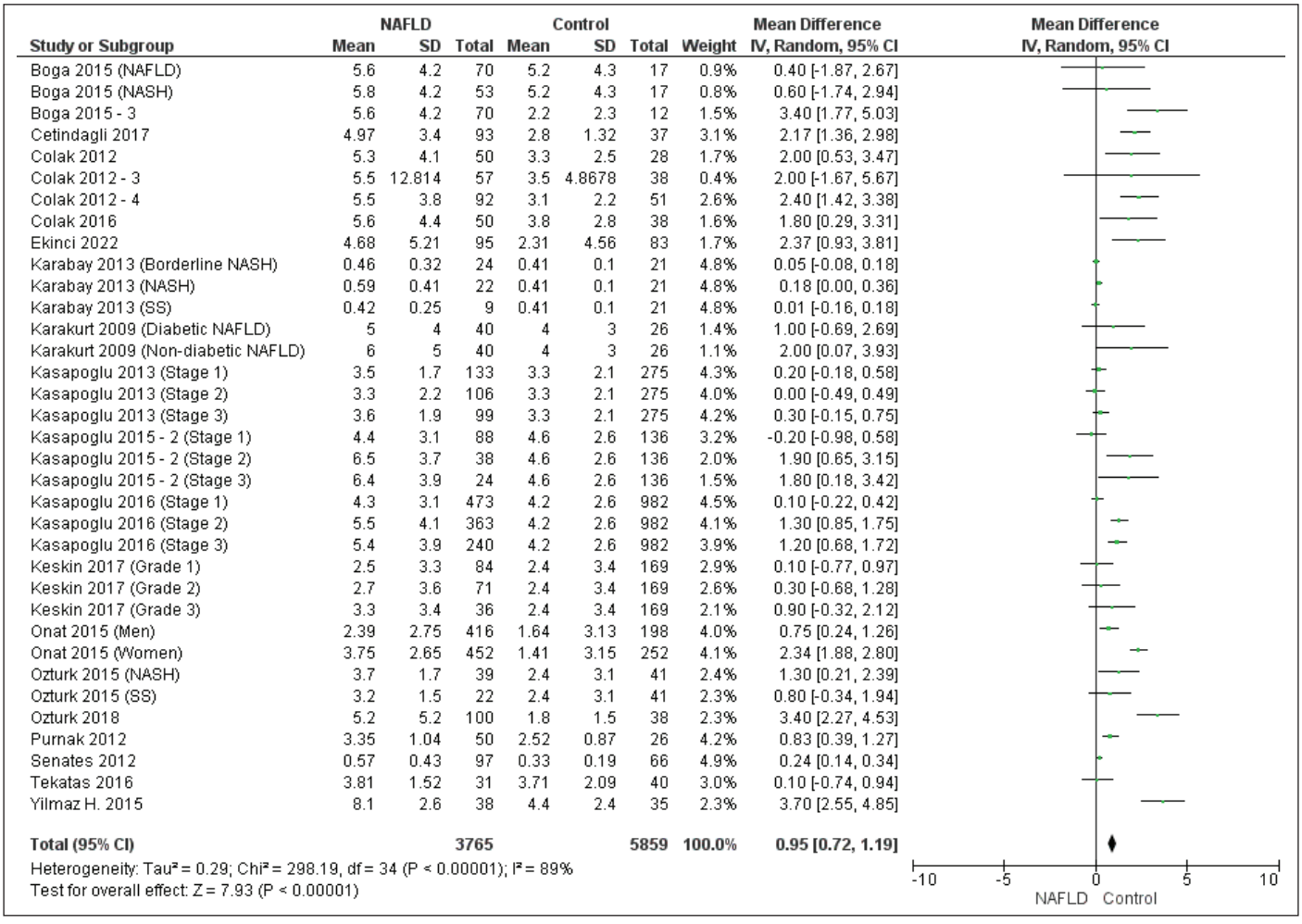


Figure 6. The random effect model of cumulative meta-analysis for CRP data obtained from NAFLD and control individuals.

did not include comorbidity or disease severity status (in terms of liver fibrosis) in our analysis due to the limited amount of studies and the heterogeneity among these studies. Our primary goal was to analyze the metabolic profile of the patients cumulatively, and we acknowledge that further studies and meta-analyses are needed to assess the effects of disease stages on the metabolic profile. It would also be beneficial to evaluate these in future studies due to changes in terminology and disease diagnosis.

We are aware of the risk of bias among studies, particularly those using the same cohort in studies conducted by the same group within a close time period. Although inclusion and exclusion criteria were mainly similar among studies, some excluded specific chronic diseases that could affect the biochemical profiles of selected patients. We accepted this heterogeneity as a limitation of our meta-analysis. However, we believe that our meta-analysis provides a comprehensive overview with a significant amount of data, specifically from Turkiye. These bias risks and limitations might have a minor impact given the extensive dataset.

Given the recency of the MASLD terminology, there are not many studies targeting exact MASLD patients according to specific diagnostic criteria for MASLD. Therefore, our study couldn't distinctly show the MSFLD and NAFLD difference or the MASLD profile of Turkiye. We acknowledge these limitations in the ongoing debate,[136] yet our results support the notion that many patients included in our meta-analysis might have MASLD, according to our cumulative results.

Conclusion

In conclusion, NAFLD is a metabolic disease that involves multiple pathways related to glucose and lipid metabolism, vascular function, inflammation, and acute phase responses. This was demonstrated through the cumulative meta-analysis of all Turkish NAFLD studies to date. These cumulative results are important for defining the metabolic profile of NAFLD patients in Turkiye and could serve as a valuable reference for many countries in Europe, Asia, and the Middle East. Additionally, the new term MASLD could be more appropriate, reflecting the related metabolic outcomes assessed cumulatively in our meta-analysis.

Author Contributions: Concept – SA, NY; Design – SA, NY; Supervision – NY; Data Collection and/or Processing – SA, NY; Analysis and/or Interpretation – SA, NY; Literature Search – SA, NY; Writing – SA; Critical Reviews – NY.

Conflict of Interest: The authors have no conflict of interest to declare.

Use of AI for Writing Assistance: Not declared.

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

Peer-review: Externally peer-reviewed.

References

1. Fujii Y, Nanashima A, Hiyoshi M, Imamura N, Yano K, Hamada T. Risk factors for development of nonalcoholic fatty liver disease after pancreatoduodenectomy. Ann Gastroenterol Surg 2017;1(3):226-231.
2. Chalasani N, Younossi Z, Lavine JE, Diehl AM, Brunt EM, Cusi K, et al. The diagnosis and management of non-alcoholic fatty liver disease: Practice Guideline by the American Association for the Study of Liver Diseases, American College of Gastroenterology, and the American Gastroenterological Association. Hepatology 2012;55(6):2005-2023.
3. Eslam M, Newsome PN, Sarin SK, Anstee QM, Targher G, Romero-Gomez M, et al. A new definition for metabolic dysfunction-associated fatty liver disease: An international expert consensus statement. J Hepatol 2020;73(1):202-209.
4. Rinella ME, Lazarus JV, Ratziu V, Francque SM, Sanyal AJ, Kanwal F, et al. A multi-society Delphi consensus statement on new fatty liver disease nomenclature. Ann Hepatol 2024;29:101133.
5. Le MH, Le DM, Baez TC, Wu Y, Ito T, Lee EY, et al. Global incidence of non-alcoholic fatty liver disease: A systematic review and meta-analysis of 63 studies and 1,201,807 persons. J Hepatol 2023;79(2):287-295.
6. Younossi Z, Tacke F, Arrese M, Chander Sharma B, Mostafa I, Bugianesi E, et al. Global perspectives on nonalcoholic fatty liver disease and nonalcoholic steatohepatitis. Hepatology 2019;69(6):2672-2682.
7. Younossi ZM, Golabi P, Paik JM, Henry A, Van Dongen C, Henry L. The global epidemiology of nonalcoholic fatty liver disease (NAFLD) and nonalcoholic steatohepatitis (NASH): a systematic review. Hepatology (Baltimore) 2023;77(4):1335.
8. Estes C, Razavi H, Loomba R, Younossi Z, Sanyal AJ. Modeling the epidemic of nonalcoholic fatty liver disease demonstrates an exponential increase in burden of disease. Hepatology 2018;67(1):123-133.
9. Younossi ZM, Koenig AB, Abdelatif D, Fazel Y, Henry L, Wymer M. Global epidemiology of nonalcoholic fatty liver disease—meta-analytic assessment of prevalence, incidence, and outcomes. Hepatology 2016;64(1):73-84.
10. Değertekin B, Tözün N, Demir F, Söylemez G, Parkan Ş, Gürtay E, Mutlu D, Toraman M, Seymenoğlu TH. The changing prevalence of non-alcoholic fatty liver disease (NAFLD) in Turkey in the last decade. Turk J Gastroenterol 2021;32(3):302.
11. Younossi ZM, Stepanova M, Negro F, Hallaji S, Younossi Y, Lam B, et al. Nonalcoholic fatty liver disease in lean individuals in the United States. Medicine (Baltimore) 2012;91(6):319-327.
12. Acikel M, Sunay S, Koplay M, Gündogdu F, Karakelleoglu S. Evaluation of ultrasonographic fatty liver and severity of coronary atherosclerosis, and obesity in patients undergoing coronary angiography. Anatol J Cardiol 2009;9(4):273.
13. Ağaç MT, Korkmaz L, Çavuşoğlu G, Karadeniz AG, Ağaç 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):604-608.
14. Akbal E, Koçak E, Taş A, Yüksel E, Köklü S. Visfatin levels in nonalcoholic fatty liver disease. J Clin Lab Anal 2012;26(2):115-119.
15. Akbal E, Koçak E, Akyürek Ö, Köklü S, Batgi H, Şenes M. Liver fatty acid-binding protein as a diagnostic marker for non-alcoholic fatty liver disease. Wien Klin Wochenschr 2016;128(1-2):48-52.
16. Alkayyali T, Qutranji L, Kaya E, Bakir A, Yilmaz Y. Clinical utility of noninvasive scores in assessing advanced hepatic fibrosis in patients with type 2 diabetes mellitus: a study in biopsy-proven non-alcoholic fatty liver disease. Acta Diabetol 2020;57(5):613-618.
17. Aktas B, Yilmaz Y, Eren F, Yonal O, Kurt R, Alahdab YO, et al. Serum levels of vaspin, obestatin, and apelin-36 in patients with nonalcoholic fatty liver disease. Metabolism 2011;60(4):544-559.
18. Akyildiz M, Gunsar F, Nart D, Sahin O, Yilmaz F, Akay S, et al. Macrophage migration inhibitory factor expression and MIF gene− 173 G/C polymorphism in nonalcoholic fatty liver disease. Eur J Gastroentero Hepatol 2010;22(2):192-198.
19. Akyuz U, Yesil A, Yilmaz Y. Characterization of lean patients with nonalcoholic fatty liver disease: potential role of high hemoglobin levels. Scand Gastroenterol 2015;50(3):341-346.
20. Arslan MS, Turhan S, Dincer I, Mizrak D, Corapcioglu D, Idilman R. A potential link between endothelial function, cardiovascular risk, and metabolic syndrome in patients with Non-alcoholic fatty liver disease. Diabetol Metab Syndr 2014;6(1):109.
21. Ayaz T, Kirbas A, Durakoglugil T, Durakoglugil ME, Sahin SB, Sahin OZ, et al. The relation between carotid intima media thickness and serum osteoprotegerin levels in nonalcoholic fatty liver disease. Metab Syndr Relat Disord 2014;12(5):283-289.
22. Aygun C, Senturk O, Hulagu S, Uraz S, Celebi A, Konduk T, et al. Serum levels of hepatoprotective peptide adiponectin in non-alcoholic fatty liver disease. Eur J Gastroenterol Hepatol 2006;18(2):175-180.
23. Aygun C, Kocaman O, Sahin T, Uraz S, Eminler AT, Celebi A, et al. Evaluation of metabolic syndrome frequency and carotid artery intima-media thickness as risk factors for atherosclerosis in patients with nonalcoholic fatty liver disease. Dig Dis Sci 2008;53(5):1352-1357.
24. Bahcecioglu IH, Koruk M, Yilmaz O, Bolukbas C, Bolukbas F, Tuncer I, et al. Demographic and clinicopathological characteristics of nonalcoholic fatty liver disease in the east-southeastern Anatolia regions in Turkey. Med Princ Pract 2006;15(1):62-68.
25. Başar Ö, Akbal E, Köklü S, Koçak E, Tuna Y, Ekiz F, et al. A novel appetite peptide, nesfatin-1 in patients with non-alcoholic fatty liver disease. Scand J Clin Lab Invest 2012;72(6):479-483.
26. Başkol M, Başkol G, Deniz K, Ozbakir O, Yücesoy M. A new marker for lipid peroxidation: serum paraoxonase activity in non-alcoholic steatohepatitis. Turk J Gastroenterol 2005;16(3):119-123.
27. Bekler A, Gazi E, Erbag G, Binnetoglu E, Barutcu A, Sen H, et al. Right ventricular function and its relationship with grade of hepatosteatosis in non-alcoholic fatty liver disease. Cardiovasc J Africa 2015;26(3):109.
28. Bilgin BO, Sunbul M, Kani HT, Demirtas CO, Keklikkiran C, Yilmaz Y. Arterial stiffness is associated independently with liver stiffness in biopsy-proven nonalcoholic fatty liver disease: a transient elastography study. Eur J Gastroenterol Hepatol 2020;32(1):54-57.
29. Bilgir O, Bilgir F, Bozkaya G, Calan M. Changes in the levels of endothelium-derived coagulation parameters in nonalcoholic fatty liver disease. Blood Coagul Fibrinolysis 2014;25(2):151-155.
30. Boga S, Alkim H, Alkim C, Koksal AR, Bayram M, Yilmaz Ozguven MB, et al. The relationship of serum hemojuvelin and hepcidin levels with iron overload in nonalcoholic fatty liver disease. J Gastrointestin Liver Dis 2015;24(3):293-300.
31. Boga S, Alkim H, Koksal AR, Bayram M, Ozguven MBY, Ergun M, et al. Increased plasma levels of asymmetric dimethylarginine in nonalcoholic fatty liver disease: relation with insulin resistance, inflammation, and liver histology. J Invest Med 2015;63(7):871-877.
32. Boga S, Koksal AR, Alkim H, Yilmaz Ozguven MB, Bayram M, Ergun M, et al. Plasma pentraxin 3 differentiates nonalcoholic steatohepatitis (NASH) from non-NASH. Metab Syndr Relat Disord 2015;13(9):393-399.
33. Celebi G, Genc H, Gurel H, Sertoglu E, Kara M, Tapan S, et al. The relationship of circulating fetuin-a with liver histology and biomarkers of systemic inflammation in nondiabetic subjects with nonalcoholic fatty liver disease. Saudi J Gastroenterol 2015;21(3):139.
34. Celikbilek M, Baskol M, Taheri S, Deniz K, Dogan S, Zararsiz G, et al. Circulating microRNAs in patients with non-alcoholic fatty liver disease. World J Hepatol 2014;6(8):613.
35. Celikbilek A, Celikbilek M, Okur A, Dogan S, Borekci E, Kozan M, et al. Non-alcoholic fatty liver disease in patients with migraine. Neurol Sci 2014;35(10):1573-1578.
36. Celikbilek A, Celikbilek M, Bozkurt G. Cognitive assessment of patients with nonalcoholic fatty liver disease. Eur J Gastroenterol Hepatol 2018;30(8):944-950.

37. Celikbilek M, Gürsoy S, Deniz K, Karaman A, Zararsiz G, Yurci A. Mean platelet volume in biopsy-proven non-alcoholic fatty liver disease. Platelets 2013;24(3):194-199.
38. Cengiz C, Ardicoglu Y, Bulut S, Boyacioglu S. Serum retinol-binding protein 4 in patients with nonalcoholic fatty liver disease: does it have a significant impact on pathogenesis? Eur J Gastroenterol Hepatol 2010;22(7):813-819.
39. Cengiz M, Ozenirler S, Elbeg S. Role of serum toll-like receptors 2 and 4 in non-alcoholic steatohepatitis and liver fibrosis. J Gastroenterol Hepatol 2015;30(7):1190-1196.
40. Cengiz M, Ozenirler S, Kocabiyik M. Serum β-trophin level as a new marker for noninvasive assessment of nonalcoholic fatty liver disease and liver fibrosis. Eur J Gastroenterol Hepatol 2016;28(1):57-63.
41. Cetindağlı I, Kara M, Tanoglu A, Ozalper V, Aribal S, Hancerli Y, et al. Evaluation of endothelial dysfunction in patients with nonalcoholic fatty liver disease: Association of selenoprotein P with carotid intima-media thickness and endothelium-dependent vasodilation. Clin Res Hepatol Gastroenterol 2017;41(5):516-524.
42. Colak Y, Senates E, Ozturk O, Yilmaz Y, Coskunpinar E, Kahraman OT, et al. Plasma fibrinogen-like protein 2 levels in patients with non-alcoholic fatty liver disease. Hepatogastroenterology 2011;58(112):2087-2090.
43. Colak Y, Senates E, Coskunpinar E, Oltulu YM, Zemheri E, Ozturk O, et al. Concentrations of connective tissue growth factor in patients with non-alcoholic fatty liver disease: association with liver fibrosis. Dis Markers 2012;33(2):77-83.
44. Colak Y, Karabay CY, Tuncer I, Kocabay G, Kalayci A, Senates E, et al. Relation of epicardial adipose tissue and carotid intima-media thickness in patients with nonalcoholic fatty liver disease. Eur J Gastroenterol Hepatol 2012;24(6):613-618.
45. Colak Y, Senates E, Ozturk O, Doganay HL, Coskunpinar E, Oltulu YM, et al. Association of serum lipoprotein-associated phospholipase A2 level with nonalcoholic fatty liver disease. Metab Syndr Relat Disord 2012;10(2):103-109.
46. Colak Y, Senates E, Ozturk O, Yilmaz Y, Zemheri E, Enc FY, et al. Serum concentrations of human insulin-like growth factor-1 and levels of insulin-like growth factor-binding protein-5 in patients with nonalcoholic fatty liver disease: association with liver histology. Eur J Gastroenterol Hepatol 2012;24(3):255-261.
47. Colak Y, Bozbey G, Erim T, Caklili OT, Ulasoglu C, Senates E, et al. Impaired gallbladder motility and increased gallbladder wall thickness in patients with nonalcoholic fatty liver disease. J Neurogastroenterol and Motil 2016;22(3):470.
48. Demirag MD, Onen HI, Karaoguz MY, Dogan I, Karakan T, Ekmekci A, et al. Apolipoprotein E gene polymorphism in nonalcoholic fatty liver disease. Dig Dis Sci 2007;52(12):3399-3403.
49. Dogan S, Celikbilek M, Yilmaz YK, Sarikaya S, Zararsiz G, Serin HI, et al. Association between liver fibrosis and coronary heart disease risk in patients with nonalcoholic fatty liver disease. Eur J Gastroenterol Hepatol 2015;27(3):298-304.
50. Dogru T, Genc H, Tapan S, Ercin CN, Ors F, Aslan F, et al. Elevated asymmetric dimethylarginine in plasma: an early marker for endothelial dysfunction in non-alcoholic fatty liver disease? Diabet Res Clin Pract 2012;96(1):47-52.
51. Eminler AT, Aygun C, Konduk T, Kocaman O, Senturk O, Celebi A, et al. The relationship between resistin and ghrelin levels with fibrosis in nonalcoholic fatty liver disease. J Res Med Sci 2014;19(11):1058.
52. Emre A, Terzi S, Celiker E, Sahin S, Yazıcı S, Erdem A, et al. Impact of nonalcoholic fatty liver disease on myocardial perfusion in nondiabetic patients undergoing primary percutaneous coronary intervention for ST-segment elevation myocardial infarction. Am J Cardiol 2015;116(12):1810-1814.
53. Ercin CN, Dogru T, Tapan S, Karslioglu Y, Haymana C, Kilic S, et al. Levels of soluble CD40 ligand and P-Selectin in nonalcoholic fatty liver disease. Dig Dis Sci 2010;55(4):1128-1134.
54. Ercin CN, Dogru T, Tapan S, Kara M, Haymana C, Karadurmus N, et al. Plasma apelin levels in subjects with nonalcoholic fatty liver disease. Metabolism 2010;59(7):977-981.
55. Ercin CN, Doğru T, Çelebi G, Gürel H, Genc H, Sertoğlu E, et al. The relationship between blood urea nitrogen levels and metabolic, biochemical, and histopathologic findings of nondiabetic, nonhypertensive patients with nonalcoholic fatty liver disease. Turk J Med Sci 2016;46(4):985-991.
56. Eren F, Kurt R, Ermis F, Atug O, Imeryuz N, Yilmaz Y. Preliminary evidence of a reduced serum level of fibroblast growth factor 19 in patients with biopsy-proven nonalcoholic fatty liver disease. Clin Biochem 2012;45(9):655-658.
57. Eren H, Horsanali MO. The independent association of non-alcoholic fatty liver disease with lower urinary tract symptoms/benign prostatic hyperplasia and erectile function scores. BJU Int 2019;124(2):329-335.
58. Erkan G, Sayın I, Polat FB, Çorakçı A, Ataç GK, Değertekin H. The relationship between insulin resistance, metabolic syndrome and nonalcoholic fatty liver disease in non-obese non-diabetic Turkish individuals: A pilot study. Turk J Gastroenterol 2014;25(Suppl 1):63-68.
59. Erkan G, Muratoglu S, Ercin U, Bilgihan A. Angiopoietin-like protein 2 and angiopoietin-like protein 6 levels in patients with nonalcoholic fatty liver disease. Arch Med Sci 2018;14(4):781.
60. Fotbolcu H, Yakar T, Duman D, Ozden K, Karaahmet T, Tigen K, et al. Aortic elastic properties in nonalcoholic fatty liver disease. Blood Pressure Monit 2010;15(3):139-145.
61. Fotbolcu H, Yakar T, Duman D, Karaahmet T, Tigen K, Cevik C, et al. Impairment of the left ventricular systolic and diastolic function in patients with non-alcoholic fatty liver disease. Cardiol J 2010;17(5):457-463.
62. Gökmen FY, Ahbab S, Ataoğlu HE, Türker BÇ, Çetin F, Türker F, et al. FT3/FT4 ratio predicts non-alcoholic fatty liver disease independent of metabolic parameters in patients with euthyroidism and hypothyroidism. Clinics 2016;71(4):221-225.
63. Gulsen M, Yesilova Z, Bagci S, Uygun A, Ozcan A, Ercin CN, et al. Elevated plasma homocysteine concentrations as a predictor of steatohepatitis in patients with non-alcoholic fatty liver disease. J Gastroenterol Hepatol 2005;20(9):1448-1455.
64. Kara M, Genc H, Tapan S, Meral C, Ercin C, Erdal M, et al. Alpha fetoprotein levels and its relationship with histopathological findings in patients with non-alcoholic fatty liver disease. Eur Rev Med Pharmacol Sci 2013;17(11):1536-1541.
65. Kara M, Dogru T, Genc H, Sertoglu E, Celebi G, Gurel H, et al. Neutrophil-to-lymphocyte ratio is not a predictor of liver histology in patients with non-alcoholic fatty liver disease. Eur J Gastroenterol Hepatol 2015;27(10):1144-1148.
66. Karabay CY, Kocabay G, Kalayci A, Colak Y, Oduncu V, Akgun T, et al. Impaired left ventricular mechanics in nonalcoholic fatty liver disease: a speckle-tracking echocardiography study. Eur J Gastroenterol Hepatol 2014;26(3):325-331.
67. Karakurt F, Carlioglu A, Koktener A, Ozbek M, Kaya A, Uyar M, et al. Relationship between cerebral arterial pulsatility and carotid intima media thickness in diabetic and non-diabetic patients with non-alcoholic fatty liver disease. J Endocrinol Invest 2009;32(1):63-68.
68. Kargili A, Cipil H, Karakurt F, Kasapoglu B, Koca C, Aydn M, et al. Hemostatic alterations in fatty liver disease. Blood Coagul Fibrinolysis 2010;21(4):325-327.
69. Kasapoglu B, Turkay C, Yalcin K, Carlioglu A, Sozen M, Koktener A. Low vitamin D levels are associated wıth increased risk for fatty liver disease among non-obese adults. Clin Med 2013;13(6):576.
70. Kasapoglu B, Turkay C, Yalcin KS, Kosar A, Bozkurt A. MTHFR 677C/T and 1298A/C mutations and non-alcoholic fatty liver disease. Clin Med 2015;15(3):248.
71. Kasapoglu B, Turkay C, Yalcın KS, Boga S, Bozkurt A. Increased microalbuminuria prevalence among patients with nonalcoholic fatty liver disease. Renal Failure 2016;38(1):15-19.

72. Kasapoglu B, Turkay C, Yalcın KS, Carlioglu A, Koktener A. Role of γ-glutamyl transferase levels in prediction of high cardiovascular risk among patients with non-alcoholic fatty liver disease. Indian J Med Res 2016;143(1):30.
73. Keskin M, Hayıroğlu Mİ, Uzun AO, Güvenç TS, Şahin S, Kozan Ö. Effect of nonalcoholic fatty liver disease on in-hospital and long-term outcomes in patients with ST–segment elevation myocardial infarction. Am J Cardiol 2017;120(10):1720-1726.
74. Kilciler G, Genc H, Tapan S, Ors F, Kara M, Karadurmus N, et al. Mean platelet volume and its relationship with carotid atherosclerosis in subjects with non-alcoholic fatty liver disease. Upsala J Med Sci. 2010;115(4):253-259.
75. Koplay M, Gulcan E, Ozkan F. Association between serum vitamin B12 levels and the degree of steatosis in patients with nonalcoholic fatty liver disease. Journal of Investigative Medicine (Baltimore) 2011;59(7):1137-1140.
76. Korkmaz H, Unler GK, Gokturk HS, Schmidt WE, Kebapcilar L. Noninvasive estimation of disease activity and liver fibrosis in nonalcoholic fatty liver disease using anthropometric and biochemical characteristics, including insulin, insulin resistance, and 13C-methionine breath test. Eur J Gastroenterol Hepatol 2015;27(10):1137-1143.
77. Küçükazman M, Ata N, Dal K, Yeniova AÖ, Kefeli A, Basyigit S, et al. The association of vitamin D deficiency with non-alcoholic fatty liver disease. Clinics 2014;69(8):542-546.
78. Kucukazman M, Ata N, Yavuz B, Dal K, Sen O, Deveci OS, et al. Evaluation of early atherosclerosis markers in patients with nonalcoholic fatty liver disease. Eur J Gastroenterol Hepatol 2013;25(2):147-151.
79. Kutlu O, Altun Ö, Dikker O, Aktaş Ş, Özsoy N, Arman Y, et al. Serum adropin levels are reduced in adult patients with nonalcoholic fatty liver disease. Med Princ Pract 2019;28(5):463-469.
80. Oğuz D, Ünal HÜ, Eroğlu H, Gülmez Ö, Çevik H, Altun A. Aortic flow propagation velocity, epicardial fat thickness, and osteoprotegerin level to predict subclinical atherosclerosis in patients with nonalcoholic fatty liver disease. Anatol J Cardiol 2016;16(12):974.
81. Onat A, Can G, Kaya A, Akbaş T, Özpamuk-Karadeniz F, Şimşek B, et al. Fatty liver disease: Disparate predictive ability for cardiometabolic risk and all-cause mortality. World J Gastroenterol 2015;21(48):13555.
82. Oral A, Sahin T, Turker F, Kocak E. Relationship between serum uric acid levels and nonalcoholic fatty liver disease in non-obese patients. Medicina 2019;55(9):600.
83. Oral A, Sahin T, Turker F, Kocak E. Evaluation of plateletcrit and platelet distribution width in patients with non-alcoholic fatty liver disease: A retrospective chart review study. Med Sci Monit 2019;25:9882.
84. Ozturk K, Uygun A, Guler AK, Demirci H, Ozdemir C, Cakir M, et al. Nonalcoholic fatty liver disease is an independent risk factor for atherosclerosis in young adult men. Atherosclerosis 2015;240(2):380-386.
85. Ozturk K, Dogan T, Celikkanat S, Ozen A, Demirci H, Kurt O, et al. The association of fatty pancreas with subclinical atherosclerosis in nonalcoholic fatty liver disease. Eur J Gastroenterol Hepatol 2018;30(4):411-417.
86. Ozveren O, Dogdu O, Sengul C, Cinar V, Eroglu E, Kucukdurmaz Z, et al. Deterioration of heart rate recovery index in patients with non-alcoholic fatty liver disease (NAFLD). Med Sci Monit 2014;20:1539.
87. Ozveren O, Izgi C, Eroglu E, Simsek MA, Turer A, Kucukdurmaz Z, et al. Doppler tissue evaluation of atrial conduction properties in patients with non-alcoholic fatty-liver disease. Ultrason Imaging 2016;38(3):225-235.
88. Purnak T, Beyazit Y, Ozaslan E, Efe C, Hayretci M. The evaluation of bone mineral density in patients with nonalcoholic fatty liver disease. Wien Klin Wochenschr 2012;124(15-16):526-531.
89. Sapmaz F, Uzman M, Basyigit S, Ozkan S, Yavuz B, Yeniova A, et al. Steatosis grade is the most ımportant risk factor for development of endothelial dysfunction in NAFLD. Medicine (Baltimore) 2016;95(14):e3280.
90. Sargin H, Sargin M, Gozu H, Orcun A, Baloglu G, Ozisik M, et al. Is adiponectin level a predictor of nonalcoholic fatty liver disease in nondiabetic male patients? World J Gastroenterol 2005;11(37):5874.
91. Saricam T, Kircali B, Koken T. Assessment of lipid peroxidation and antioxidant capacity in non-alcoholic fatty liver disease. Turk J Gastroenterol 2005;16(2):65-70.
92. Senates E, Yilmaz Y, Colak Y, Ozturk O, Altunoz ME, Kurt R, et al. Serum levels of hepcidin in patients with biopsy-proven nonalcoholic fatty liver disease. Metab Syndr Relat Disord 2011;9(4):287-290.
93. Şenateş E, Colak Y, Yeşil A, Coşkunpinar E, Şahin Ö, Kahraman Ö, et al. Circulating resistin is elevated in patients with non-alcoholic fatty liver disease and is associated with steatosis, portal inflammation, insulin resistance and nonalcoholic steatohepatitis scores. Minerva Med 2012;103(5):369-376.
94. Senturk O, Kocaman O, Hulagu S, Sahin T, Aygun C, Konduk T, et al. Endothelial dysfunction in Turkish patients with non-alcoholic fatty liver disease. Intern Med J 2008;38(3):183-189.
95. Sertoglu E, Ercin CN, Celebi G, Gurel H, Kayadibi H, Genc H, et al. The relationship of serum uric acid with non-alcoholic fatty liver disease. Clin Biochem 2014;47(6):383-388.
96. Sunbul M, Agirbasli M, Durmus E, Kivrak T, Akin H, Aydin Y, et al. Arterial stiffness in patients with non-alcoholic fatty liver disease is related to fibrosis stage and epicardial adipose tissue thickness. Atherosclerosis 2014;237(2):490-493.
97. Sunbul M, Kivrak T, Durmus E, Akin H, Aydin Y, Ergelen R, et al. Nonalcoholic steatohepatitis score is an independent predictor of right ventricular dysfunction in patients with nonalcoholic fatty liver disease. Cardiovasc Ther 2015;33(5):294-299.
98. Tekatas DD, Bahcecioglu IH, Ispiroglu M, Sahin A, Ilhan N, Yalniz M, et al. Role of renin-angiotensin-converting enzyme level and ACE gene polymorphism in patients with nonalcoholic fatty liver disease. Euroasian J Hepatogastroenterol 2016;6(2):137-142.
99. Tok D, Ekiz F, Basar O, Coban S, Ozturk G. Serum endocan levels in patients with chronic liver disease. Int J Clin Exper Med 2014;7(7):1802.
100. Türkay C, Özol D, Kasapoğlu B, Kirbas İ, Yıldırım Z, Yiğitoğlu R. Influence of obstructive sleep apnea on fatty liver disease: role of chronic intermittent hypoxia. Respir Care 2012;57(2):244-249.
101. Uygun A, Ozturk K, Demirci H, Oztuna A, Eren F, Kozan S, et al. The association of nonalcoholic fatty liver disease with genetic polymorphisms: a multicenter study. Eur J Gastroenterol Hepatol 2017;29(4):441-447.
102. Yalniz M, Bahcecioglu IH, Ataseven H, Ustundag B, Ilhan F, Poyrazoglu OK, et al. Serum adipokine and ghrelin levels in nonalcoholic steatohepatitis. Mediators Inflamm 2006;2006(6):34295.
103. Yaman H, Cakır E, Ozcan O, Yesilova Z, Ozcan A, Akgul EO, et al. Elevated urine neopterin levels in nonalcoholic steatohepatitis. Clin Biochem 2005;38(2):187-190.
104. Yesilova Z, Ozata M, Oktenli C, Bagci S, Ozcan A, Sanisoglu SY, et al. Increased acylation stimulating protein concentrations in nonalcoholic fatty liver disease are associated with insulin resistance. Am J Gastroenterol 2005;100(4):842-849.
105. Yilmaz Y, Ulukaya E, Atug O, Dolar E. Serum concentrations of human angiopoietin-like protein 3 in patients with nonalcoholic fatty liver disease: association with insulin resistance. Eur J Gastroenterol Hepatol 2009;21(11):1247-1251.
106. Yilmaz Y, Yonal O, Kurt R, Oral AY, Eren F, Ozdogan O, et al. Serum levels of osteoprotegerin in the spectrum of nonalcoholic fatty liver disease. Scand J Clin Lab Invest 2010;70(8):541-546.
107. Yilmaz Y, Kurt R, Yonal O, Polat N, Celikel CA, Gurdal A, et al. Coronary flow reserve is impaired in patients with nonalcoholic fatty liver disease: association with liver fibrosis. Atherosclerosis 2010;211(1):182-186.
108. Yilmaz Y, Yonal O, Kurt R, Alahdab YO, Eren F, Ozdogan O, et al. Serum levels of omentin, chemerin and adipsin in patients with biopsy-proven nonalcoholic fatty liver disease. Scand J Gastroenterol 2011;46(1):91-97.
109. Yilmaz Y, Eren F, Yonal O, Kurt R, Aktas B, Celikel CA, et al. Increased serum FGF21 levels in patients with nonalcoholic fatty liver disease. Eur J Clin Invest 2010;40(10):887-892.

110. Yilmaz Y, Eren F, Kurt R, Yonal O, Polat Z, Senates E, et al. Serum galectin-3 levels in patients with nonalcoholic fatty liver disease. Clin Biochem 2011;44(12):955-958.
111. Yilmaz Y, Eren F, Yonal O, Polat Z, Bacha M, Kurt R, et al. Serum progranulin as an independent marker of liver fibrosis in patients with biopsy-proven nonalcoholic fatty liver disease. Dis Markers 2011;31(4):205-210.
112. Yilmaz Y, Eren F, Ayyildiz T, Colak Y, Kurt R, Senates E, et al. Serum pigment epithelium-derived factor levels are increased in patients with biopsy-proven nonalcoholic fatty liver disease and independently associated with liver steatosis. Clin Chim Acta 2011;412(23-24):2296-2299.
113. Yilmaz Y, Yonal O, Kurt R, Alahdab YO, Ozdogan O, Celikel CA, et al. Circulating levels of vascular endothelial growth factor A and its soluble receptor in patients with biopsy-proven nonalcoholic fatty liver disease. Arch Med Res 2011;42(1):38-43.
114. Yilmaz Y, Kurt R, Gurdal A, Alahdab YO, Yonal O, Senates E, et al. Circulating vaspin levels and epicardial adipose tissue thickness are associated with impaired coronary flow reserve in patients with nonalcoholic fatty liver disease. Atherosclerosis 2011;217(1):125-129.
115. Yilmaz Y, Kurt R, Eren F, Imeryuz N. Serum osteocalcin levels in patients with nonalcoholic fatty liver disease: association with ballooning degeneration. Scand J Clin Lab Invest 2011;71(8):631-636.
116. Yilmaz Y, Eren F, Colak Y, Senates E, Celikel CA, Imeryuz N. Hepatic expression and serum levels of syndecan 1 (CD138) in patients with nonalcoholic fatty liver disease. Scand J Gastroenterol 2012;47(12):1488-1493.
117. Yilmaz Y, Eren F. Serum biomarkers of fibrosis and extracellular matrix remodeling in patients with nonalcoholic fatty liver disease: association with liver histology. Eur J Gastroenterol Hepatol 2019;31(1):43-46.
118. Yilmaz H, Yalcin KS, Namuslu M, Celik HT, Sozen M, Inan O, et al. Neutrophil-lymphocyte ratio (NLR) could be better predictor than C-reactive protein (CRP) for liver fibrosis in non-alcoholic steatohepatitis (NASH). Ann Clin Lab Sci 2015;45(3):278-286.
119. Yilmaz Y, Ozturk O, Alahdab YO, Senates E, Colak Y, Doganay HL, et al. Serum osteopontin levels as a predictor of portal inflammation in patients with nonalcoholic fatty liver disease. Dig Liv Dis 2013;45(1):58-62.
120. Bayrak M. Non-invasive diagnosis of nonalcoholic fatty liver disease: impact of age and other risk factors. Aging Male 2020;23:1275-1282.
121. Delik A, Akkız H, Dinçer S. The effect of PNPLA3 polymorphism as gain in function mutation in the pathogenesis of non-alcoholic fatty liver disease. Indian J Gastroenterol 2020;39:84-91.
122. Akkiz H, Taskin E, Karaogullarindan U, Delik A, Kuran S, Kutlu O. The influence of RS738409 I148M polymorphism of patatin-like phospholipase domain containing 3 gene on the susceptibility of non-alcoholic fatty liver disease. Medicine (Baltimore) 2021;100(19):e25893.
123. Arıkan D, Önmez A, Aksu E, Taşdemir N. Predictivity of fatty liver index for non-alcoholic fatty liver disease in lean females with polycystic ovary syndrome. Afr Health Sci 2022;22(1):648-656.
124. Ekinci I, Dumur S, Uzun H, Anataca G, Yalcinkaya I, Buyukkaba M, et al. Thrombospondin 1 and nuclear factor kappa b signaling pathways in non-alcoholic fatty liver disease. J Gastrointestin Liver Dis 2022;31(3):309-316.
125. Karaoğullarindan Ü, Üsküdar O, Odabaş E, Saday M, Akkuş G, Delik A, et al. Is mean platelet volume a simple marker of non-alcoholic fatty liver disease? Indian J Gastroenterol 2023;42(2):219-225.
126. Vural Keskinler M, Mutlu HH, Sirin A, Erkalma Senates B, Colak Y, Tuncer I, et al. Visceral adiposity index as a practical tool in patients with biopsy-proven nonalcoholic fatty liver disease/nonalcoholic steatohepatitis. Metab Syndr Relat Disord 2021;19(1):26-31.
127. Sonmez A, Dogru T, Ercin CN, Genc H, Celebi G, Gurel H, et al. Betatrophin levels are related to the early histological findings in nonalcoholic fatty liver disease. Metabolites 2021;11(7):425.
128. Ulasoglu C, Tekin ZN, Akan K, Yavuz A. Does nonalcoholic pancreatic steatosis always correlate with nonalcoholic fatty liver disease? Clin Exp Gastroenterol 2021;14:269-275.
129. Yilmaz M, Odabas O, Karaaslan M, Guler OF, Toprak T, Bicer S, et al. Predicting risk of erectile dysfunction in patients with nonalcoholic fatty liver disease. Andrologia 2021;53(7):e14091.
130. Yozgat A, Ekmen N, Kasapoglu B, Unsal Y, Sokmen FC, KEKiLLi M. Monocyte/HDL ratio in non-alcoholic hepatic steatosis. Arq Gastroenterol 2021;58:439-442.
131. Gassaway BM, Petersen MC, Surovtseva YV, Barber KW, Sheetz JB, Aerni HR, et al. PKCε contributes to lipid-induced insulin resistance through cross talk with p70S6K and through previously unknown regulators of insulin signaling. Proceed Nat Acad Sci 2018;115(38):E8996-E9005.
132. Tilg H, Effenberger M. From NAFLD to MAFLD: when pathophysiology succeeds. Nat Rev Gastroenterol Hepatol 2020;17(7):387-388.
133. Eslam M, Sanyal AJ, George J, Sanyal A, Neuschwander-Tetri B, Tiribelli C, et al. MAFLD: a consensus-driven proposed nomenclature for metabolic associated fatty liver disease. Gastroenterology 2020;158(7):1999-2014. e1.
134. Huang DQ, Noureddin N, Ajmera V, Amangurbanova M, Bettencourt R, Truong E, et al. Type 2 diabetes, hepatic decompensation, and hepatocellular carcinoma in patients with non-alcoholic fatty liver disease: an individual participant-level data meta-analysis. Lancet Gastroenterol Hepatol 2023;8(9):829-836.
135. Ling Q, Chen J, Liu X, Xu Y, Ma J, Yu P, et al. The triglyceride and glucose index and risk of nonalcoholic fatty liver disease: A dose–response meta-analysis. Front Endocrinol 2023;13:1043169.
136. Yilmaz Y. The heated debate over NAFLD renaming: An ongoing saga. Hepatol Forum 2023;4(3):89-91.