



Evaluation of the Frequency of Non-Alcoholic Fatty Liver Disease Among Coronary Heart Disease Patients

Abeer H. Abdelkader¹, Naglaa A. Abdelwahab¹, Hisham S Roshdy², Doaa I. Elsayed^{1*}, Moustafa H. Elshamy¹

¹Department of Gastroenterology, Hepatology and Infectious Diseases, Faculty of Medicine, Zagazig University, Egypt.

²Department of Cardiology, Faculty of Medicine, Zagazig University, Egypt

*Corresponding author:

Doaa I. Elsayed

Email:

Khaleddoaa095@gmail.com

Submit Date 19-01-2025

Accept Date 29-01-2025

ABSTRACT

Background: Nonalcoholic fatty liver disease (NAFLD) is the most common form of chronic liver disease and cardiovascular complications account for about 40% of total deaths in NAFLD. Evaluation of the link between NAFLD and ischaemic heart disease helps in better management of NAFLD patients and reduction of coronary heart disease complication. This study targets detection of the link between NAFLD and coronary heart diseases, also provides better management of non-alcoholic fatty liver disease patients and helps reduction of cardiovascular complications in these patients.

Methods: 84 patients with coronary heart disease were evaluated with routine investigation (CBC, liver function tests, ESR. Lipid profile), BMI, abdominal ultrasound, echocardiography with estimation of wall motion score index of left ventricle, and fib 4 evaluation of liver fibrosis. **Results:** The severity of cardiac hypokinesia and liver fibrosis were directly proportionate to the severity of liver steatosis.

Conclusion: NAFLD is commonly associated with coronary heart disease and the severity of NAFLD detected by ultrasonography is strongly related to the severity of coronary arteries obstruction.

Keywords: NAFLD; coronary heart disease; wall motion score index

INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) is one of the most prevalent chronic hepatic disorders influencing about 15–30% of the general population [1]. Environmental changes and adverse feeding practices made the prevalence of these disorders rising globally [2].

Simple steatosis, nonalcoholic steatohepatitis (NASH) and fibrosis are all included in the broad category of nonalcoholic fatty liver disease (NAFLD) [3].

Even though at least 40% of mortality in NAFLD are attributable to cardiovascular disease (CVD), liver-related complications are important causes of death in this condition. NAFLD, irrespective of conventional risk factors, is a risk factor for CVD [4].

The leading cause of death worldwide is coronary artery disease (CAD), which includes myocardial infarction, unstable angina, and stable angina. Numerous risk factors, including age, gender, diabetes, hypertension, obesity, hyperlipidemia, and tobacco use, have been identified. Every risk factor is commonly linked to metabolic syndrome [5,6].

Type 2 diabetes mellitus, obesity, dyslipidemia, hypertension, and insulin resistance are all linked to steatotic liver disease, it represents a new metabolic syndrome component [7].

The implications of nonalcoholic fatty liver disease and nonalcoholic steatohepatitis are important because of their high frequency in the general population and their contribution to the risk of coronary artery disease [8].

METHODS

Between December 2022 and June 2024, 84 patients with coronary heart disease who were inpatients and outpatients in the cardiology, hepatology, gastroenterology, and infectious diseases departments of Zagazig University Hospitals in Zagazig, Egypt, were presented in this cross-sectional study. Every patient provided written informed permission, and Zagazig University IRB #9587/ 14-6-2022 approved the study.

Inclusion criteria were the study comprised adult patients with coronary heart disease who had been diagnosed by history, clinical examination, electrocardiogram, echocardiography, and coronary angiography. Exclusion criteria were patients with hepatocellular cancer, heart failure, alcoholic hepatitis, autoimmune hepatitis, or persistent HBV and HCV were not included.

Patients' assessments

Every patient in the current study underwent a thorough clinical examination and medical history. (CBC), biochemistry (ALT, AST albumin, bilirubin, prothrombin time), random, fasting, postprandial blood glucose, and glycosylated hemoglobin (HbA1C) are examples of routine laboratory tests.

The lipid profile is composed of triglycerides, total cholesterol, low-density lipoprotein and high-density lipoprotein .

Body mass index; weight in kilograms by height in square meters. Overweight when BMI was ≥ 25 and $< 30 \text{ kg/m}^2$, and Obesity was diagnosed when BMI was $\geq 30 \text{ kg/m}^2$ [9] (Table 1).

Scoring of fibrosis by FiB4 [11] (Table 2).

$$\text{FIB 4} = \frac{\text{age (years)} \times \text{AST (U/L)}}{\text{platelet count (10}^9\text{/L)} \times \sqrt{\text{ALT (U/L)}}$$

Echocardiography and calculation of wall motion score index

As part of the standard of care, a transthoracic echocardiography was conducted by a cardiac

specialist. A 17-segment model was used to analyze the left ventricular (LV) regional wall motion scores (12) Segmental investigation of wall motion score using transthoracic echocardiography. The following are the LV segments: 1 stands for the basal anterior, 2 for the basal anterior septum, 3 for the inferior septum, 4 for the basal inferior, 5 for the basal inferolateral, 6 for the basal anterolateral, 7 for the mid-anterior, 8 for the mid-anterior septum, 9 for the mid-inferior septum, 10 for the mid-inferior, 11 for mid-inferolateral, 12 for the mid-anterolateral, 13 is the anterior apex, 14 is the septal apex, 15 is the inferior apex, 16 is the lateral apex and 17 is the apex.

Individual segments were scored as follows

1= normal or hyperkinetic

2= hypokinesia (reduced thickening)

3= akinesia absent or negligible thickening, e.g., scar)

4= dyskinesia (systolic thinning or stretching, e.g., aneurysm)

The wall motion score index (WMSI)

summation of the scores of all individual segments and the result is divided by the number of segments. The final result represents the average score for all left ventricular segments.

Statistical analysis

SPSS (Statistical Package for Social Science) version 24 was used to computerize and statistically analyze the gathered data. For numerical data, the mean and standard deviation (mean \pm SD) were employed. Non-numerical (qualitative) data were described using frequency and percentage. The relationship between the qualitative factors was examined using chi-square. To compare quantitative data, use a one-way ANOVA. Spearman correlation tests for non-parametric variable correlation and post hoc testing for multiple comparisons. $P < 0.05$ was used to denote significance

Table 1: Grading of liver steatosis by ultrasound [10]

Grades	Sonographic appearance (descriptive criteria)
Grade 0: absent steatosis	When the liver has been normal echotexture
Grade 1: Mild steatosis	When the liver has a slight diffuse increase in echogenicity with normal visualization of the diaphragm and wall of the portal vein
Grade 2: moderate steatosis	Moderate steatosis when the liver has moderately increased echogenicity with the slightly impaired appearance of the diaphragm and wall of the portal vein.
Grade 3: Severe steatosis	When the liver has markedly increased echogenicity with poor or no visualization of the diaphragm, portal vein wall, and posterior part of the right lobe of the liver

Table 2: Fib 4 value interpretation

Fib 4 score	Interpretation
<1.3	Mild fibrosis
1.3- 2.67	Indeterminate
>2.67	Severe

RESULTS

Patients' characteristics

Patients with coronary heart disease in this study aged between 35 and 89 years old and the majority of them were female (67.9%). Half of them had diabetes (53.6%), more than half had hypertension (58.3%), and obese patients were only one-fifth (20 %.) (Table 3).

In the abdominal ultrasound grading of (NAFLD), approximately one-third (29.8%) had grade 0 with normal echogenicity, over half (58.3%) had grade 1, and only 11.9% had grade 2, (Table 3). Half of the study participants (52.4%) had an elevated risk of developing advanced liver fibrosis, according to FiB 4 estimation.

Wall motion score index

Transthoracic echocardiography was used to measure the Wall Motion Score Index, which revealed that half of the patients had mild hypokinesia and only 17.9% had moderate hypokinesia in the left ventricle (Table 3). When Wall Motion Score Index compared to the ultrasound grading of NAFLD, it was found that 90% of cases with moderate ultrasound (grade 2) had moderate hypokinesia, while nearly two-thirds of cases (77.6%) with grade 1 group had mild hypokinesia. This difference was statistically significant (Table 4).

Comparing the Wall Motion Score Index and FIB 4 within the study group revealed a statistically significant difference because all cases with severe FIB 4 exhibited moderate hypokinesia, while the majority of cases (80%) with moderate FIB4 are classified as mild hypokinesia (Table 4).

Regarding age, in the older age group nearly two-thirds (61.4%) had mild hypokinesia, and (29.5%) of those old age group had moderate hypokinesia. Additionally, there was a statistically significant correlation ($p < 0.05$) between diabetes mellitus and the Wall Motion Score Index, with 28.9% of the diabetic cases having moderate hypokinesia and 48.9% of the cases having mild hypokinesia. Additionally, there was a statistically significant correlation ($p = 0.000$) between the wall motion score index and BMI, with obese individuals with higher BMIs experiencing higher degree of hypokinesia (Table 4).

When correlation analysis was performed, as (Table 5) illustrates. The Wall Motion Score Index was found to be positively and significantly correlated with each of the following: age of cases, diabetes, FIB4, and ultrasound-graded NAFLD.

Table 3: Demographic and imaging of studied patients.

Variables	N= 48
Age (years)	
35- 60	40 (47.6%)
> 60	44 (52.4 %)
Gender	
Male	27 (32.1 %)
Female	57 (67.9 %)
Diabetes	45 (53.6 %)
Hypertension	49 (58.3 %)
BMI (weight in kilograms by height in square meters).	
Less than 30	67 (79.8%)
Equal or more than 30	17 (20.2%)
Grading of NAFLD by ultrasound	
Grade 0	25 (29.8 %)
Grade 1	49 (58.3 %)
Grade 2	10 (11.9 %)
Wall motion score index by echocardiography	
Normal	27 (32.1%)
Mild hypokinesia	42 (50%)
Moderate hypokinesia	15 (17.9%)
FIB 4	
Mild risk of fibrosis (less than 1.3	40 (47.6 %)
Intermediate risk (1.3 - 2.67)	35 (41.7 %)
Severe high risk (more than 2.67)	9 (10.7 %)

Table 4: Comparing wall motion score index with another parameter (FIB 4 estimation and abdominal ultrasound grading of NAFLD.

Variables	Wall Motion Score Index			Tests	
	Normal group N=27	Mild hypokinesia N = 42	Moderate hypokinesia N= 15	Tests	P value
FIB 4					
Mild	26 (65%)	14 (35%)	0	79.427 (x ²)	<0.00 1*
Moderate	1 (2.9)	28 (80 %)	6 (17.1 %)		
Severe	0	0	9 (100 %)		
Grading of steatosis by Ultrasound					
Grade 0	21 (84%)	3 (12 %)	1 (4%)	81.466 (x ²)	<0.00 1*
Grade 1	6 (12.2 %)	38 (77.6%)	5 (10.2 %)		
Grade 2	0	1 (10%)	9 (90%)		
BMI					
Normal	26 (38.8%)	36 (53.7%)	5 (7.5%)	25.525 (x ²)	.000
Obese	1 (5.9%)	6 (35.3%)	10 (58.8%)		
Diabetes					
Yes	10 (22.2%)	22 (48.9%)	13 (28.9%)	9.597 (x ²)	0.008 *
No	17 (43.6%)	20 (51.3%)	2.5 (1%)		
Age					<0.00

Variables	Wall Motion Score Index			Tests	
	Normal group N=27	Mild hypokinesia N = 42	Moderate hypokinesia N= 15	Tests	P value
Middle age (less than 60 Old (more than 60years)	23 (57.5%) 4 (9.1%)	15 (37.5%) 27 (61.4%)	2 (5%) 13 (29.5%)	24.731 (χ^2)	1*

χ^2 =Chi square test

Table 5: Correlation between Wall motion Score Index, fib 4 estimation, Grading ultrasound in NAFLD, age, and diabetes among the studied participants

Variables	Wall motion score index	
	R	P value
Grading of steatosis by ultrasound	0.744	0.000
FIB 4	0.786	0.000
Age	0.533	0.000
Diabetes	0.253	0.020

P=Sig. (2-tailed), r=Pearson Correlation

DISCUSSION

Risk factors for CAD and NAFLD are similar, including obesity, insulin resistance, metabolic syndrome, and dyslipidemia. The substantial morbidity and mortality associated with the two disorders, highlights the significance of early management of both. For instance, individuals with NASH should be screened for CAD if a strong correlation is discovered [13]. Numerous indicators of subclinical atherosclerosis, including increase coronary calcification, reduced flow, vasodilation of arteries and increased intimal-medial thickness of arteries, were linked to NAFLD [14].

In order to improve patient care and lower cardiovascular consequences, this study sought to make to determine the prevalence of NAFLD and its correlation with CAD.

The study sample included more than half (67.9%) females with coronary heart disease with a mean age of 60.68 ± 15.96 years and a range of ages from 35 to 89 years.

Increased proportion of women suffered coronary heart disease could be explained by the fact that females had one or more risk factors, including menopause, diabetes, obesity, and high blood pressure.

According to a study by Namakchian et al., men made up 61.5% (224) of the patients with coronary heart disease [15].

According to the current study's FIB4 index, 9 patients (10.7%) had severe fibrosis, 40 patients (47.5%) had mild fibrosis, and 35 cases (41.7%) had intermediate fibrosis.

All cases with severe FIB 4 exhibited moderate hypokinesia and the majority of cases (80%) with moderate FIB4 are classified as mild hypokinesia, our current data unequivocally demonstrated that there was a direct correlation between the wall motion score index and FIB 4. Abdu et al. reported that 10% of patients showed severe fibrosis ($\geq F3$), which was consistent with our findings [16].

According to a study by Namakchian et al., patients with CAD had a considerably higher FIB4 score, with 54.1% (125) having a high risk of fibrosis and 12.6% (29) having a low risk [15].

Tsai et al. found that FIB4 was higher in patients with atherogenic plaque on coronary CT angiography [17].

Additionally, Jin et al. demonstrated a positive correlation between the FIB4 index and the quantity of diseased arteries in CAD patients [18].

Higher baseline liver fibrosis scores were found to be strongly linked to the risk of cardiovascular events in a study conducted by Liu et al [19]. Baratta et al claim that the risk of cardiovascular events was doubled in people

with NAFLD and four times higher for people with liver fibrosis [20].

According to a different study, there was no correlation between the risk of CAD and an elevated FIB-4 score in the general population or in individuals with chronic liver disease [21].

The group with advanced fibrosis had a considerably higher FIB-4 score than the group with mild-to-moderate fibrosis. This was similar to a study by Ballestri et al. [22] that discovered FIB-4 has a lot of potential for identifying NAFLD and viral hepatitis-related liver fibrosis [22]. Similar results were found by Amernia et al. [23], they came to the conclusion that FIB-4 is one of the best indicators for assessing liver fibrosis in NAFLD patients [23].

According to our current findings, half of the cases (50%) had mild hypokinesia, whereas normal-kinetic cases (32.1%) and moderate hypokinesia (17.9%) using wall motion score index.

We found a statistically significant correlation between the Wall Motion Score Index and BMI, diabetes, and old age group. This is consistent with the findings of Ahmed et al. who demonstrated that diabetes mellitus was present in over 70% of individuals with coronary heart disease [24].

The study also supports the findings of Liu et al. (2008), who found that diabetes mellitus was the most important risk factor for coronary heart disease, with about one-fourth of individuals having DM [25].

When comparing the wall motion score index and NAFLD grades by abdominal ultrasound in this study, we discovered statistically significant correlation because nearly two-thirds of cases (77.6%) with grade 1 had mild hypokinesia, whereas the majority of cases (90%) with grade 2 had moderate hypokinesia. Our results were almost identical to those of Gholoobi et al. It was determined that 63.2% of the patients with NAFLD had CADs, and the prevalence and severity of CAD were highly correlated with the severity of NAFLD [26]. A study by Lerchbaum et al. found a substantial and independent correlation between higher degrees of the fatty liver and cardiovascular mortality [27].

NAFLD representing the hepatic component of metabolic syndrome which is multiple-system illness and raises the risk of CAD [28].

Although a direct link between NAFLD and CAD has not yet been established, the primary cause of death for those with non-alcoholic fatty liver disease is cardiovascular event through a variety of likely underlying mechanisms, including altered lipid metabolism, systemic inflammation, insulin resistance, oxidative stress, endothelial dysfunction, and plaque formation [29].

Wong et al. concluded that 58.2% (n=356) of patients with coronary heart disease had live steatosis on sonographic evaluation [30].

Montemezzo et al. discovered that 56.4% of patients without NAFLD and 93.4% of patients with NAFLD had CAD, which is in line with our findings. The prevalence of CAD was also linked to the severity of NAFLD. Additionally, they noted that CAD was significantly and independently predicted by NAFLD [31].

The link between NAFLD and CAD in patients with type 2 diabetes was also corroborated by Idilman et al. [32].

Numerous studies have looked into the connection between CAD and NAFLD. According to Baharvand-Ahmadi's et al. 47% of CAD patients had fatty liver disease, indicating a strong correlation between CAD and NAFLD [33].

The steatotic liver releases procoagulant and inflammatory chemicals throughout the body so NAFLD has a role in the pathophysiology of CVD rather than just being a marker for it. Peripheral insulin resistance causes increased adipose tissue lipolysis and increased triglyceride production in the liver. The liver's insulin signaling pathways are noticeably disrupted as a result of aberrant fat deposition in the hepatocytes [31].

NAFLD is thought to be a marker and an early mediator of atherosclerosis, and patients with the condition are known to have more complicated coronary artery disease on angiography. Furthermore, coronary artery disease becomes more severe when NAFLD is present [34].

What part does NAFLD play in the risk of CVD? Patients suffering from non-alcoholic fatty liver disease (NAFLD) often have many

cardiovascular disease risk factors, including obesity, insulin resistance, hypertension, and dyslipidemia [35].

Other studies showed that patients with NAFLD had elevated levels of coagulation factors, suggesting an increased risk of thrombosis, in addition to showing that systemic inflammation predisposed individuals to endothelial dysfunction and thrombosis [36, 37].

According to a different study, coagulation factor VII, von Willebrand factors, fibrinogen and C-reactive protein were all greater in NAFLD patients. These variables are all associated with a higher risk of thrombosis [38].

Conclusion

Patients with acute coronary syndromes frequently have NAFLD, and the degree of coronary artery obstruction is closely correlated with the degree of NAFLD identified by ultrasonography.

Conflict of interest

The authors declared that they have no conflicts of interest with respect to authorship and/or publication of this article.

Financial disclosures

This study was not supported by any source of funding.

REFERENCES

1. Lerchbaum E, Pilz S, Grammer TB, Boehm BO, Stojakovic T, Obermayer-Pietsch B et al. The fatty liver index is associated with increased mortality in subjects referred to coronary angiography. *Nutrition, Metabolism Cardiovasc Dis.* 2013 Dec 1; 23(12): 1231-8.
2. Mitra S, De A, Chowdhury A. Epidemiology of non-alcoholic and alcoholic fatty liver diseases. *Transl Gastroenterol Hepatol.* 2020; 5: 16.
3. 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. *Hepatol.* 2016 Jul 1;64(1):73-84.
4. Przybyszewski EM, Targher G, Roden M, Corey KE. Nonalcoholic fatty liver disease and cardiovascular disease. *Clin Liver Dis.* 2021 Jan 1;17(1):19-22.
5. Ong JP, Pitts A, Younossi ZM. Increased overall mortality and liver-related mortality in non-alcoholic fatty liver disease. *J Hepatol.* 2008 Oct;49(4):608-12.
6. Stepanova M, Younossi ZM. Independent association between nonalcoholic fatty liver disease and cardiovascular disease in the US population. *Clin Gastroenterol Hepatol.* 2012 Jun 1;10(6):646-50.
7. Angulo. Nonalcoholic fatty liver disease. *N Engl J Med.* 2002; 346(16):1221-31.
8. Caldwell S, Argo C. The natural history of non-alcoholic fatty liver disease. *Dig Dis.* 2010; 28:162–8.
9. Cichoż-Lach H, Celiński K, Prozorow-Król B. The BARD score and the NAFLD fibrosis score in the assessment of advanced liver fibrosis in nonalcoholic fatty liver disease. *Med Sci Monit: Int Med J experiment and Clin Res.* 2012;18(12), CR735.
10. Ferraioli G, Soares Monteiro L .Ultrasound-Based Techniques for the diagnosisof liver steatosis . *World J Gastroenterol.* 2019; 25(4); 6053-62.
11. Vallet-Pichard A, Mallet V, Nalpas B, Verkarre V. FIB-4: an inexpensive and accurate marker of fibrosis in HCV infection. Comparison with liver biopsy and fibrotic. *Hepatology (Baltimore, Md.).* 2007; 46(1), 32-6.
12. Lang RM, Badano LP, Mor-Avi. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. *Eur Heart J Cardiovasc Imaging.* 2015, 16(3):233–71.
13. Abosheishaa H, Hussein M, Ghallab M, Abdelhamid M, Balassiano N, Ahammed MR et al. Association between non-alcoholic fatty liver disease and coronary artery disease outcomes: A systematic review and meta-analysis. *Diabetes Metab Syndr.* 2024; 18(1):102938.
14. Kim S, Park H, Lee H, Jung K, Lee M, Jhee J, et al. Association between non-alcoholic fatty liver disease and coronary calcification depending on sex and obesity. *Sci Rep.* 2020; 10(1), 1025.
15. Namakchian M, Rabizadeh S, Seifouri S, Asadigandomani H, Bafrani MA, Seifouri K, et al. A. Fibrosis score 4 index has an independent relationship with coronary artery diseases in patients with metabolic-associated fatty liver disease. *Diabetol Metab Syndr.* 2023; 15(1):57.
16. Abdu S, Abdulaziz B, EL Kharbotly E, Nassief A. Assessment of liver fibrosis in non-alcoholic fatty liver disease using real-time elastography. *Benha Med J.* 2020 Nov 1; 37(Special issue (Internal medicine and Hepatology)):1-1.
17. Tsai TY, Hsu PF, Wu CH, Huang SS, Chan WL, Lin SJ et al. Association between Coronary Artery Plaque Progression and Liver Fibrosis Biomarkers in Population with Low Calcium Scores. *Nutrients.* 2022; 30;14(15):3163.
18. Jin J, Zhang H, Cao Y, Liu H, Hua Q, Li Y, et al. Liver fibrosis scores and coronary atherosclerosis: novel findings in patients with stable coronary artery disease. *Hepatol Int.* 2021;15(2):413–23.
19. Liu H, Cao Y, Jin J, Hua Q, Li Y, Guo Y, et al. Liver fibrosis scoring systems as novel tools for predicting cardiovascular outcomes in patients following elective percutaneous coronary intervention. *J Am Heart Assoc.* 2021; 10(3):e018869.

20. Baratta F, Pastori D, Angelico F, Balla A, Paganini A, Cocomello N, et al. Nonalcoholic fatty liver disease and fibrosis associated with increased risk of cardiovascular events in a prospective study. *Clin Gastroenterol Hepatol*. 2020; 18(10): 2324–31.
21. Loosen S, Luedde M, Demir M, Luedde T, Kostev K, Roderburg C. An elevated FIB-4 score is not associated with cardiovascular events: a longitudinal analysis from 137 842 patients with and without chronic liver disease. *Eur J Gastroenterol Hepatol*. 2022; 34(6):717–23.
22. Ballestri S, Mantovani A, Baldelli E, Lugari S, Maurantonio M, Nascimbeni F et al. Liver fibrosis biomarkers accurately exclude advanced fibrosis and are associated with higher cardiovascular risk scores in patients with NAFLD or viral chronic liver disease. *Diagnostics*. 2021;11(1), 98.
23. Amernia B, Moosavy SH, Banookh F, Zoghi G. FIB-4, APRI, and AST/ALT ratio compared to FibroScan for the assessment of hepatic fibrosis in patients with non-alcoholic fatty liver disease in Bandar Abbas, Iran. *BMC gastroenterol*. 2021 Dec; 21:1-7.
24. Ahmed N, Kazmi S, Nawaz H, Javed M, Anwar SA, Alam MA. “Frequency of Diabetes Mellitus in Patients with Acute Coronary Syndrome.” *J Ayub Med Coll Abbottabad*. 2014; 26(1): 57–60.
25. Liu J, Zhao D, Liu Q, Wang W, Sun JY, Wang M et al. “[Study on the Prevalence of Diabetes Mellitus among Acute Coronary Syndrome Inpatients in a Multiprovincial Study in China].” *Zhonghua liu xing bing xue za zhi = Zhonghua liu xing bing xue za zhi*. 2008; 29(6): 526–29.
26. 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 May;6(5):330-7.
27. Lerchbaum E, Pilz S, Grammer TB, Boehm BO, Stojakovic T, Obermayer-Pietsch B et al. The fatty liver index is associated with increased mortality in subjects referred to coronary angiography.
- 37.
38. Çınar T, Şaylık F, Akbulut T, Asal S, Selçuk M, Çiçek V et al. The association between whole blood viscosity and high thrombus burden in patients with non-ST elevation myocardial infarction. *Kardiol Pol*. 2022; 80(4):429–35.
- Nutrition, Metabolism Cardiovasc Dis. 2013 Dec 1;23(12):1231-8.
28. Kasper P, Martin A, Lang S, Kütting F, Goeser T, Demir M et al. NAFLD and cardiovascular diseases: a clinical review. *Clin Res Cardiol*. 2021; 110 (7):921–37
29. Stahl EP, Dhindsa DS, Lee SK, Sandesara PB, Chalasani NP, Sperling LS. Nonalcoholic fatty liver disease and the heart: JACC State-of-the-Art Review. *J Am Coll Cardiol*. 2019; 73(8):948–63.
30. Wong V, Wong G, Yip G, Lo A, Limquiacco J, Chu W, et al. Coronary artery disease and cardiovascular outcomes in patients with non-alcoholic fatty liver disease. *Gut*. 2011; 60(12):1721-7.
31. Montemezzo M, Alturki A, Stahlschmidt F, Olandoski M, Rodrigo Tafarel J, Precoma DB. Nonalcoholic fatty liver disease and coronary artery disease: big brothers in patients with acute coronary syndrome. *Scientific World J*. 2020; 2020(1): 8489238.
32. Idilman IS, Akata D, Hazirolan T, Doganay Erdogan B, Aytemir K, Karcaaltincaba M. Nonalcoholic fatty liver disease is associated with significant coronary artery disease in type 2 diabetic patients: a computed tomography angiography study 2. *J. Diabetes*. 2015; 7: 279–86.
33. Baharvand-Ahmadi B, Sharifi K, Namdari M. Prevalence of non-alcoholic fatty liver disease in patients with coronary artery disease. *ARYA atheroscler*. 2016; 12(4), 201.
34. Ling S, Shu-zheng LÜ. Association between non-alcoholic fatty liver disease and coronary artery disease severity. *Chin Med J*. 2011 Mar 1;124(6):867-72.
35. Than NN, Newsome PN. A concise review of non-alcoholic fatty liver disease. *Atherosclerosis*. 2015 Mar 1; 239(1):192-202.
36. Gidaro A, Manetti R, Delitala A, Salvi E, Bergamaschini L, Vidili G, et al. Prothrombotic and inflammatory markers in elderly patients with non-alcoholic hepatic liver disease before and after weight loss: a pilot study. *J Clin Med*. 2021; 25, 10(21): 4906.
39. Hörber S, Lehmann R, Stefan N, Machann J, Birkenfeld AL, Wagner R, et al. Hemostatic alterations linked to body fat distribution, fatty liver, and insulin resistance. *Mol Metab*. 2021; 53:101262.

Citation

Abdelkader, A., Abdelwahab, N., Roshdy, H., Elsayed, D., Elshamy, M. Evaluation of the Frequency of Non-Alcoholic Fatty Liver Disease Among Coronary Heart Disease Patients. *Zagazig University Medical Journal*, 2025; (1431-1438): -. doi: 10.21608/zumj.2025.353867.3804