Research Article

BIOMEDICAL Journal of Scientific & Technical Research

ISSN: 2574 -1241

DOI: 10.26717/BJSTR.2023.49.007840

Atherosclerotic Coronary Artery Disease and Nonalcoholic Fatty Liver Disease: Exploring the Possible Correlation

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ARTICLE INFO

Received: March 30, 2023

Published: April 11, 2023

Citation: Mario Claudio Soares Sturzeneker, Camilla Mattia Calixto, Bruna Karas, Mariana Fonseca, Jaqueline Meert Parlow, Amy Sakakibara, Paola Gonçalves Moreira de Oliveira, Alice Magro Koscianski, Gilberto Baroni and Marcelo de Oliveira Dreweck . Atherosclerotic Coronary Artery Disease and Nonalcoholic Fatty Liver Disease: Exploring the Possible Correlation. Biomed J Sci & Tech Res 49(4)-2023. BJSTR. MS.ID.007840.

ABSTRACT

Introduction: Non-alcoholic fatty liver disease (NAFLD) is the most common cause of chronic liver disease. It is frequently associated with components of metabolic syndrome and cardiovascular diseases, especially atherosclerotic disease.

Objective: Evaluate the relationship between coronary artery disease (CAD) and NAFLD in the context of prevalence and severity.

Method: A pilot cross-sectional study was developed, from January to October 2022, with the inclusion criteria age > 18 years, history of documented significant CAD, coronary artery bypass surgery, or percutaneous coronary intervention. Exclusion criteria were significant alcohol consumption and secondary causes of hepatic steatosis. Thirty-one patients were allocated. CAD was classified as moderate, severe, and very severe. NAFLD was assessed by abdominal ultrasonography and graded as mild, moderate, or severe.

Results: The prevalence of NAFLD was 70.96%, 81.82% with a mild grade, and 18.18% with a moderate grade. Among patients with NAFLD, 22.72% had moderate CAD, 31.81% had severe CAD, and 45.45% had very severe CAD. In those with moderate CAD, 100% had mild-grade NAFLD. In those with severe CAD, 85.71% had mild and 14.28% had moderate NAFLD, and in those with very severe CAD, 70% had mild and 30% had moderate NAFLD. The correlation between the degree of CAD and the presence of NAFLD ($p = 0.9818$) and the correlation between the severity of both diseases ($p = 0.3679$) were not significant.

Conclusion: NAFLD is frequently associated with atherosclerotic coronary artery disease, and there is an apparently significant influence of the presence of CAD on the prevalence of NAFLD.

Keywords: Nonalcoholic Fatty Liver Disease; Coronary Artery Disease; Atherosclerosis

Abbreviations: NAFLD: Non-Alcoholic Fatty Liver Disease; IR: Insulin Resistance; NASH: Non-Alcoholic Steatohepatitis; CVD: Cardiovascular Diseases; MetS: Metabolic Syndrome; CAC: Coronary Artery Calcium; ACS: Acute Coronary Syndrome; SURF CHD: Survey of Risk Factors in Coronary Heart Disease; CABG: Coronary Artery Bypass Surgery; PCI: Percutaneous Coronary Intervention; CAD: Coronary Artery Disease

Introduction

Non-alcoholic fatty liver disease (NAFLD) is the most frequent cause of chronic liver disease, with an estimated prevalence of 25% and progressive incidence [1,2]. It is an increasing cause of end-stage liver disease and primary liver cancer. It is a leading indication for liver transplantation in the United States and the fastest-growing cause of liver-related death worldwide [3,4]. Defined by the presence of steatosis in at least 5% of hepatocytes, evidenced by imaging method or histology, and the absence of significant alcohol consumption, other chronic liver diseases, and competing causes of hepatic steatosis [5]. Noninvasive imaging methods such as ultrasonography, computerized tomography scan, and magnetic resonance imaging can be used to identify NAFLD [5]. The gold standard for determining the stage and grade of liver injury is a biopsy, indicated for patients at high risk of steatohepatitis or advanced fibrosis and in cases where other methods cannot define the etiology [5,6]. Its broad histological spectrum ranges from isolated steatosis to steatohepatitis (steatosis associated with hepatocyte necroinflammation and ballooning), advanced fibrosis, and cirrhosis [5]. It has variable rates of progression as a heterogeneous disease, but in most patients, it presents as a slowly progressive disease [7]. Several metabolic, genetic, and gut microbiota-related factors may be involved in the pathophysiology of NAFLD. However, in many aspects, the mechanisms involved still need to be fully understood [8].

Insulin resistance (IR) has been described as the essential factor for the development of the disease, determining lipid accumulation and, consequently, oxidative stress, lipid peroxidation, and the inflammatory process that culminates in injury, cell death, and fibrotic replacement [9]. The specific pharmacological treatment of NAFLD remains undetermined, and no drug has been cleared by the regulatory agencies for this purpose, with the treatment of frequently associated diseases such as obesity, type 2 diabetes mellitus (DM2), hypertension (SAH), and dyslipidemia being recommended [10]. For biopsy-proven cases of non-alcoholic steatohepatitis (NASH), pioglitazone and vitamin E can be considered in non-diabetics, and pioglitazone in diabetics [5]. In the animal model of NAFLD, the angiotensin 2 AT1 receptor blocker, olmesartan, and the angiotensin-converting enzyme inhibitor, ramipril, widely used in the treatment of NASH, significantly attenuated the development of the full histological spectrum of hypercholesterolemic diet-induced liver disease. It can be inferred that they may have adjuvant action in treating NAFLD in hypertensives [11,12]. Similar to what is recommended for cardiovascular diseases (CVD) in general, adopting a healthy lifestyle, such as a balanced diet and regular physical activity aimed at weight loss, forms the basis of treatment for NAFLD [5]. In a prospective study involving NASH patients, histological improvement was observed in those who achieved at least 5% weight loss, being more significant in individuals who lost 10% or more of their body weight [13].

NAFLD is frequently associated with metabolic syndrome (Mets) components, which has motivated its description as a hepatic manifestation and an emerging component of this syndrome [2]. In a prospective study, 36% of NAFLD patients satisfied the diagnostic criteria for MetS (presence of 3 or more components), and the vast majority had at least one component of the syndrome, 69, 86 and 97% of normal weight, overweight and obese individuals respectively [14]. NAFLD is highly prevalent in obese people, with this prevalence exceeding 95% in grade 3 obese individuals undergoing bariatric surgery [15]. In diabetics, the prevalence has been estimated at 55% [16], and an increased risk of advanced fibrosis and cirrhosis has been reported in these patients [17]. CVD, especially atherosclerotic disease, has been frequently associated with NAFLD [18]. Both share several risk factors, but the mechanisms involved in this relationship are complex and heterogeneous and have not yet been fully elucidated [19]. Genetics, IR, adipose tissue dysfunction, dyslipidemia, oxidative stress, inflammation, and endothelial dysfunction are critical factors of this association [20]. In a systematic review, NAFLD was significantly associated with subclinical atherosclerotic disease, independent of traditional risk factors and MetS [21]. In a meta-analysis of observational studies, the prevalence of CVD in NAFLD-affected people was estimated at 25 to 40%. CVD was the leading cause of death, corresponding to up to 40% of overall mortality [22]. NAFLD has been associated with increased subclinical and clinical manifestations of CVD.

In a recently published systematic review and meta-analysis, NAFLD was related to the more significant progression of coronary artery calcium (CAC) score, an important subclinical marker of coronary artery disease (CAD) [23]. Regarding the correlation between the severity of NAFLD and the severity of CAD, in a cross-sectional study, severe NAFLD was related to the presence of coronary plaques, especially those at high risk, regardless of traditional risk factors [24]. Furthermore, a retrospective study showed a significant relationship between NAFLD and the severity of acute coronary syndrome (ACS), with the results also suggesting a correlation between the severity of both [25]. In a prospective cohort study, NAFLD was related to an increased risk of developing obstructive atherosclerotic lesions >50% and of percutaneous coronary interventions [26]. Results of a prospective study conducted in Japan suggest that the presence of NASH determines a higher risk of coronary lesions compared to NAFLD patients with isolated steatosis [27]. Moreover, in a meta-analysis of observational studies and a recently published cohort study, the risk of fatal and nonfatal cardiovascular events was significantly higher in patients with NAFLD, and this risk was higher in those with more severe liver disease [22,28]. In this sense, the apparent correlation between the severity of NAFLD and CAD, the importance of both in current medical practice and the absence of consistent data necessary to properly understand this relationship justify conducting studies in this context.

Methodology

This is a cross-sectional, descriptive pilot study developed, from January to October 2022, at the Regional University Hospital of Campos Gerais (HU-UEPG). The allocated patients are part of a subgroup of another ongoing study: Survey of Risk Factors in Coronary Heart Disease (SURF CHD) II. Inclusion criteria were age over 18 years, history of documented significant CAD, coronary artery bypass surgery (CABG), or percutaneous coronary intervention (PCI). Exclusion criteria, however, were significant alcohol consumption (≥20 grams of alcohol per day for women and ≥30 grams for men), prolonged use of steatogenic medications, and other secondary causes of steatosis, as well as known chronic hepatopathy. CAD was classified as moderate or grade 1 if only one vessel was approached by PCI, severe or grade 2 if PCI approached two or more vessels, and very severe if there was isolated CABG or CABG associated with PCI. The ultrasonographic examinations were performed by a radiologist certified by Brazilian College of Radiology and Federal Council of Medicine,using a SAMSUNG device, model HS50, convex transducer with a frequency of 4.0 MHz. Standard images in the axial and sagittal planes of the left and right lobes were acquired, totalizing four images per patient. The diagnosis and grading of hepatic steatosis were based on the finding of increased hepatic echogenicity subjectively or in comparison to the right kidney, being stratified into three grades.

1. Grade I (Mild): increased hepatic echogenicity with normal visualization of intrahepatic vascular branches and diaphragmatic facet;

2. Grade II (Moderate): increased hepatic echogenicity with attenuation of the posterior acoustic beam and partial blurring of the intrahepatic vascular branches and diaphragmatic facet;

3. Grade III (Severe): increased hepatic echogenicity with attenuation of the posterior acoustic beam and complete blurring of the diaphragmatic facet [29,30].

The data collected were tabulated and submitted for statistical analysis using the BioEstat software, version 5.3. The results were described as percentages, and to evaluate the relationship between CAD and NAFLD, the Spearman coefficient was used as a prerequisite for linear regression analysis. The Ponta Grossa State University Research Ethics Committee approved the study under protocol number 5.209.153.

Objective

To evaluate the relationship between CAD and NAFLD in the context of prevalence and severity.

Results

Of 50 eligible patients, 16 did not maintain regular outpatient follow-up, and three did not perform abdominal ultrasonography, so 19 patients were excluded, and 31 were allocated (Figure 1). Regarding the characterization of the allocated individuals, 18 (58.06%) were male, and 13 (41.94%) were female. The mean age was 66.93 years, with the extremes of age being 45 years and 90 years. The most prevalent ethnicity was white, with 77.42%. Black and brown corresponded to 6.45% and 16.13%, respectively. All individuals were non-drinkers, and 5 were former drinkers. As for comorbidities, 25.8% were smokers, 45.16% were overweight, 25.80% were obese, 32.26% were diabetic, 93.54% were hypertensive, 35.48% had MS, and all individuals were dyslipidemic (Table 1). CAD classified as moderate or grade 1 was evidenced in 7 patients (22.58%), severe or grade 2 in 10 (32.26%), and very severe CAD or grade 3 in 14 patients, 45.16% (Figure 2). Twenty-two patients had NAFLD (70.96%), 14 were male (63.64%), and 8 (36.36%) were female. Regarding ethnicity, 81.81% were white, 9.09% were brown, and 9.09% were black. Regarding comorbidities, 54.54% were overweight, 18.18% were obese (all grade 1), 100% had hypertension, 100% had dyslipidemia, 40.90% had MS, and 40.9% had DM2 (Table 2). Regarding the severity of NAFLD, 18 patients (81.82%) had grade 1 or mild, and 4 (18.18%) had grade 2 or moderate. No individual presented severe NAFLD. Among the patients with NAFLD, 5 (22.72 %) had grade 1 or moderate CAD, 7 (31.81%) had grade 2 or severe, and 10 (45.45%) had grade 3 or very severe CAD (Table 3).

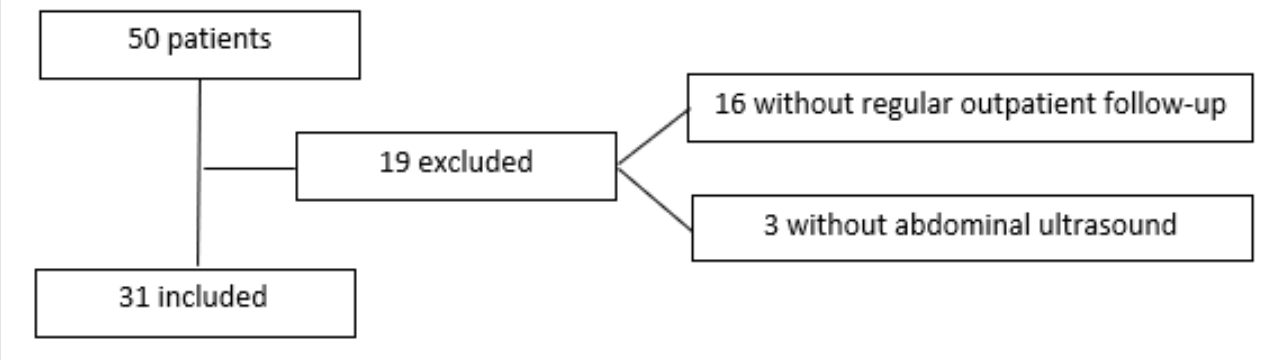

Figure 1: Patients Included and Excluded in the Study.

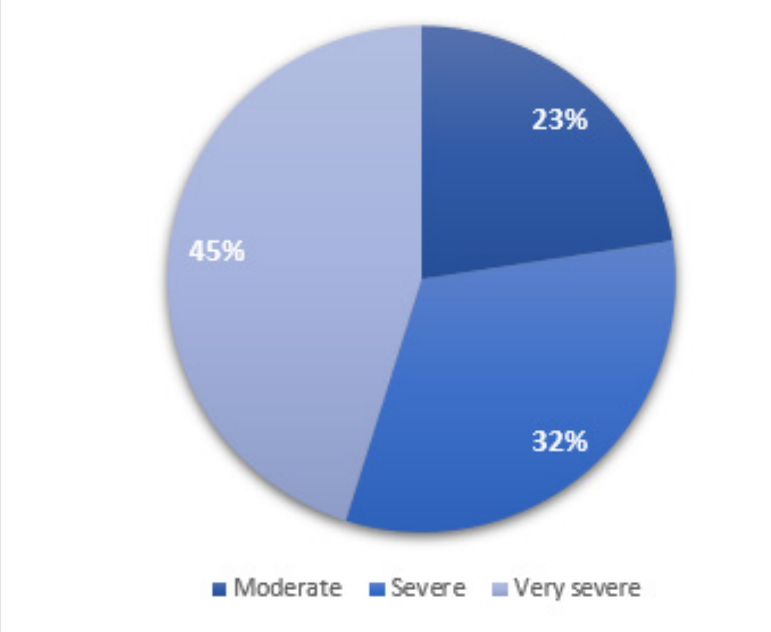

Figure 2: Severity of CAD in the Allocated Population.

Table 1: General Characterization of the Allocated Individuals.

Variables		Number of individuals	Percentage
Sex	Male	18	58,06%
	Female	13	41,94%
Ethnicity	White	24	77,42%
	Brown	5	16,13%
	Black	2	6,45%
Smokers		8	25,8%
BMI	Overweight	14	45,16%
	Obesity	8	25,80%
DM2		10	32,26%
SAH		29	93,54%
Dyslipidemia		31	100%
MetS		11	35,48%

Note: BMI (Body Mass Index), DM2 (type 2 Diabetes Mellitus), SAH (Systemic Arterial Hypertension), MS (Metabolic Syndrome).

Table 2: General Characterization of NAFLD Patients.

Variables		Number of individuals	Percentage
Sex	Male	14	63,64%
	Female	8	36,36%
Ethnicity	White	18	81,81%
	Brown	2	9.09%
	Black	2	9,09%
Smokers		4	18,18%
BMI	Overweight	12	54,54%
	Obesity	4	18,18%
DM2		9	40,90%
SAH		22	100%
Dyslipidemia		22	100%
MetS		9	40,90%

Note: BMI (Body Mass Index), DM2 (Type 2 Diabetes Mellitus), SAH (Systemic Arterial Hypertension), MS (Metabolic Syndrome).

Table 3: Severity of CAD in NAFLD Patients.

CAD	Number of Individuals with NAFLD	Porcentage
Moderate	5	22,72%
Severe	7	31,81%
Very severe	10	45,45%
Total	22	100%

Note: CAD (coronary artery disease).

Concerning the prevalence of NAFLD and its grades in each subgroup according to the classification of CAD, 5 of the seven individuals with moderate or grade 1 CAD had steatosis (71.43%), and all had mild grade (100%). Among the ten individuals with severe or grade 2 CAD, steatosis was detected in 7 (70%), with 6 (85.71%) of mild grade and 1 (14.28%) of moderate grade. Ten (71.43%) of the 14 patients with very severe or grade 3 CAD had steatosis, of which 7 (70%) were mild, and 3 (30%) were moderate (Table 4). The correlation between the degree of CAD and the presence of NAFLD assessed by Spearman's coefficient (Spearman's coefficient: 0.0043, p value=0.9818) and, additionally, by linear regression analysis, was not significant (p = 0.9877). Similarly, the correlation between the degree of CAD and the degree of NAFLD was also not significant (Spearman coefficient: 0.1252, p-value=0.5022), linear regression analysis with p-value=0.3679.

Table 4: Grades of NAFLD According to the Classification of CAD.

CAD	NAFLD Total		NAFLD Grade I		NAFLD Grade II	
	Number of individuals	Porcentage	Number of individuals	Porcentage	Number of individuals	Percentage
Moderate	5	71,43%	5	100%	0	0
Severe	7	70%	6	85,71%	1	14,28%
Very severe	10	71,43%	7	70%	3	30%
Total	22	70,96%	18	58,06%	4	12,90%

Note: NAFLD (Non-Alcoholic Fatty Liver Disease).

Discussion

The frequent, significant, and sometimes independent association between NAFLD and CVD, especially atherosclerotic disease, has been demonstrated in different ways and has reinforced the hypothesis that this liver disease is at least one risk marker for CVD [25]. Among the possible links between CVD and NAFLD is the systemic inflammatory response related mainly to NASH [6]. In the present study, NAFLD was highly prevalent (70.96%), similar to the prevalence reported in a cross-sectional study (71.9%) in the subgroup of CAD patients, who represented 64.8% of the sample consisting of 264 non-diabetic patients [31]. Also similar was the prevalence of 72.7% reported in CAD carriers, who accounted for 70.5% of the sample of a prospective study involving 505 patients [32]. Also, a significant but lower

prevalence of NAFLD was reported in patients with severe CAD without total occlusion (54.5%) and more significant in those with total occlusion (100%). However, those with total occlusion represented 5.5% of the sample, and the diagnosis of CAD was established by angiotomography [33]. Similarly, the estimated prevalence of NAFLD in 110 individuals with documented CAD, characterized by stenosis of one of the main coronary arteries or its branches, was 49%. NAFLD was diagnosed by ultrasonography; however, patients who underwent coronary artery bypass grafting, representing 45.16% of our sample, were excluded [34]. The prevalence of NAFLD in the general population was estimated at 25.24% in a widely cited meta-analysis [2] and more recently estimated at 32.4% [35].

Therefore, even considering potential confounders, there is an apparently significant influence of the presence of CAD on the prevalence of NAFLD. Regarding the prevalence according to the severity of NAFLD, in our study, 81.81% of individuals had mild or grade 1 NAFLD, 18.18% grade 2 or moderate, and none grade 3 or severe. In a previously cited study, the result was similar, 77.7% and 22.2%, respectively, of mild and moderate grades, with the absence of severe grade [34]. In a recently published cross-sectional study, the lower grade of NAFLD was also predominant. However, the sonographic classification of NAFLD was composed only of the two extremes, mild and marked grade, with the criteria for marked or severe grade of NAFLD consisting of the composition of the criteria for grade 2 or moderate and grade 3 or severe NAFLD [36]. Concerning the potential relationship between the severity of both diseases, in the subgroup with CAD classified as moderate (grade 1), 100% of the cases of NAFLD were grade 1 or mild. In those with severe (grade 2) CAD, 85.71% of the cases of NAFLD were grade 1 or mild and 14.28% moderate or grade 2, and in those with very severe (grade 3) CAD, 70% of the cases of NAFLD were grade 1 or mild and 30% grade 2 or moderate. In a cross-sectional study involving patients with suspected CAD, the prevalence of NAFLD was higher in patients with CAD classified as severe. However, the diagnosis of NAFLD was based on the presence of steatosis detected by ultrasound, associated with increased serum alanine aminotransferase levels, the grades of hepatopathy were not estimated, and the CAD patients were divided into two groups, mild and severe [37].

In a previously cited study, the presence of CAD was significantly related to increased prevalence of NAFLD. By the parameters of ultrasound liver elastography, there was no significant difference between the grades of CAD. However, at least one lesion determining stenosis ≥75%, characterized the highest grade of CAD [32]. Therefore, all patients allocated in our study in this proposed classification would have the same severity of CAD. In patients undergoing liver biopsy and coronary artery angiotomography, the calcium score and the degree of stenosis in coronary arteries were significantly related to the degree of ballooning and fibrosis stage but not to the degree of steatosis and lobular inflammation [27]. However, in addition to the known limitation of angiotomography to adequately quantify coronary stenosis, CAD was classified into four grades, and the grade 4 classification criterion was compatible with all grades defined in our study. Despite the limitations, these results suggest a possible correlation between the severity of both diseases or a trend in this direction. Obesity was observed in 18.18% of these patients, all classified as obese grade 1. However, MS was diagnosed in 40.90%, and DM2 was also diagnosed in 40.9% of these patients. In a cross-sectional study involving patients with DM2, the overall prevalence of NAFLD, regardless of BMI, was 50%. However, when these patients were evaluated according to the BMI, the prevalence was higher than 60% in obese patients with grade 2 and higher than 80% in obese patients with grade 3 [38].

Considering the possible potentiation of the prevalence by the association of DM2 and obesity, as well as the percentage of obese in our sample, it can be inferred that CAD is the main factor determining this high prevalence. However, the limited number of patients allocated makes this conclusion difficult. In our study, the correlation between the severity of CAD and the prevalence of NAFLD and between the severity of both was not significant. However, it can be inferred from the results that there is a correlation trend between the severity of both diseases in focus. The limitations imposed by the small sample of patients, the predominance of the mild form of NAFLD in this sample (81.81%), and the absence of the severe form tend to weaken these results. This is a pilot study, so it will be continued with the necessary adjustments so that it can contribute effectively to elucidating this frequent, important, and challenging relationship between CAD and NAFLD.

Conclusion

NAFLD is frequently associated with atherosclerotic coronary artery disease. Therefore, even considering potential confounders, we can consider that there is an apparently significant influence of the presence of CAD on the prevalence of NAFLD. However, this relationship still needs adequate understanding.

Conflicts of Interest

There are no conflicts of interest in the present study.

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ISSN: 2574-1241

DOI: 10.26717/BJSTR.2023.49.007840

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