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Non-alcoholic fatty liver disease: Patient profiles and key contributing factors

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ABSTRACT

BACKGROUND: Non-alcoholic fatty liver disease (NAFLD) is a growing global health concern, strongly associated with obesity, metabolic syndrome, and cardiovascular diseases. Recognizing the key risk factors is essential for early detection and prevention.

OBJECTIVES: This study aims to examine patient profiles and identify the primary contributing factors to NAFLD.

METHODS: This cross-sectional study was conducted in the Gastroenterohepatology Division of Dr. Saiful Anwar Hospital, Malang, from May to October 2022. A total of 31 patients diagnosed with NAFLD based on abdominal ultrasound findings were included. Data were collected through medical history assessments, physical examinations, laboratory tests, and transient elastography. Multivariate logistic regression analysis was performed using SPSS 25 software.

RESULTS: The findings revealed that age ($p = 0.029$), body mass index (BMI) ≥23 kg/m² ($p < 0.001$), abdominal circumference exceeding normal limits ($p < 0.001$), and dyslipidemia ($p < 0.001$) were significantly associated with NAFLD. However, blood pressure, gender, fasting blood sugar, aspartate aminotransferase (AST), and alanine aminotransferase (ALT) did not exhibit significant associations ($p > 0.05$).

CONCLUSION: The study identified age, obesity, abdominal circumference, and dyslipidemia as the main contributing factors to NAFLD. Early detection and targeted intervention for metabolic risk factors are crucial in preventing disease progression.

KEYWORDS: NAFLD; obesity; dyslipidemia; insulin resistance; risk factors.

INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) is an increasing global health challenge due to its association with metabolic disorders and the risk of progression to more severe liver complications.[1] Currently, approximately 25% of the global population is diagnosed with NAFLD, with incidence rates continuing to rise in parallel with the increasing prevalence of obesity, diabetes, and dyslipidemia.[2] One of the primary factors contributing to the development of NAFLD is visceral and subcutaneous adipose tissue, where triglycerides are stored as the main component of fat accumulation in the liver.[3] There are several related mechanisms through which the latter sets in motion risk factors mainly for cardiovascular disease and liver fibrosis.[4-6] The event of accumulation leads to insulin resistance, systemic inflammation, and oxidative stress.[7] While obesity is one recognized factor in NAFLD, studies have suggested that others, especially metabolic ones, are responsible for NAFLD in patients with normal body weight.[8]

A large number of studies pointed out hypertension as a specific risk factor for NAFLD.[9] The primary mechanisms linking hypertension to NAFLD include insulin resistance, oxidative stress, chronic inflammation, endothelial dysfunction, and changes in gut microbiota.[10] Studies have shown that the prevalence of NAFLD among hypertensive individuals was greater than those with normal blood pressure.[11] An aggravating factor by which hypertension might also facilitate NAFLD evolution into liver fibrosis is by means of the increased activity of the renin-angiotensin system and oxidative stress, both invoking inflammation in hepatocytes.[12] However, there exists an inconsistency regarding the findings on the direct relationship between hypertension and NAFLD, requiring investigation of this association with respect to its pathophysiological mechanisms.[13] Thus, this study intends to investigate the association between metabolic factors, including hypertension, and the pathogenesis of NAFLD so as to understand better their role in disease progression.[14] The present study findings are expected to provide a clearer understanding of metabolic risk factors implicated in NAFLD pathogenesis, thereby laying the groundwork for more effective preventive and therapeutic strategies.[14] Should the goals of the study receive validation through the results, then interventions addressing metabolic factors (for example, blood pressure control, dyslipidemia, and lifestyle changes) should be considered as prime strategies in preventing the evolution of NAFLD to a more advanced stage.

METHODS

Design

This study was conducted through an observational method with cross-sectional design taking place at the gastroenterology and hepatology clinic of Saiful Anwar Hospital, Malang. The observation runs from March 2022 to August 2022. The sampling method was sequencing, which allowed for participant selection based on predetermined criteria for inclusion and exclusion for those NAFLD factors that were found to be associated. The research protocol was developed following the guidelines outlined in the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE)

Ethical Approval

This research protocol was deemed ethically appropriate under approval number 400/065/K.3/102.7/2022. The study was conducted by the principles of the Declaration of Helsinki. Before participation, all subjects were provided with a detailed explanation of the study's objectives, potential risks, and benefits, and they voluntarily provided written informed consent. Participants had the right to withdraw from the study without any consequences. No financial incentives were offered to participants.

Participants & eligibility criteria

This study involved 27 respondents selected using a consecutive sampling method, with the minimum sample size determined based on Lemeshow's formula. Sampling was carried out consecutively, meaning that every patient who met the inclusion criteria and did not fall under the exclusion criteria was included until the required sample size was reached. The inclusion criteria consisted of adult patients (>18 years old) diagnosed with NAFLD, confirmed through abdominal ultrasound. The exclusion criteria included patients with a history of alcohol consumption, use of medications that could induce fatty liver, liver infections caused by Hepatitis B or Hepatitis C viruses, hepatocellular carcinoma, congenital liver abnormalities, or autoimmune diseases.

Data collection

This study was conducted in the Gastroenterohepatology Division of Dr. Saiful Anwar General Hospital, Malang, over a period of six months, from May 2022 to October 2022. Data were collected through anamnesis, physical examination, laboratory tests, abdominal ultrasound, and transient elastography. Data collection was carried out by trained medical professionals who had undergone prior training in research procedures. In cases of data inconsistencies or missing information, verification was conducted through medical records or consultations with the attending physician.

Covariates

In this study, each variable was defined and measured according to applicable medical standards. NAFLD diagnosis was confirmed based on the presence of hepatic steatosis detected through abdominal ultrasound, with no other secondary causes identified. Other variables, including blood pressure, body mass index (BMI), abdominal circumference, waist circumference, body fat mass, and muscle mass, were measured using standard clinical instruments. Lipid profile (total cholesterol, HDL, LDL, triglycerides), fasting blood glucose, and liver function markers (ALT, AST) were assessed through laboratory analysis. Transient elastography results were utilized to evaluate the degree of liver fibrosis in patients.

Statistical Analysis

Categorical data in this study were presented as n (%). The Kolmogorov-Smirnov test was used to assess the distribution of numerical data. If the data followed a normal distribution, they were reported as mean ± SD. Conversely, if the distribution was non-normal, data were presented as median (IQR). Baseline data analysis included the chi-square test for categorical variables and either the independent t-test or Mann-Whitney test for numerical data, depending on the normality of distribution. The study outcomes were evaluated using logistic regression, where only variables with $p < 0.25$ were included in multivariable logistic regression. The primary analysis was conducted using stepwise logistic regression, eliminating variables with the highest p-value until only significant variables ($p < 0.05$) remained. Significant variables were further analyzed to assess their impact on NAFLD incidence. All statistical analyses were performed using SPSS 25 software (IBM SPSS Statistics 25, IBM Corp., Armonk, NY, US).

RESULTS

Baseline characteristics of patients included in our study

A total of 31 patients with NAFLD were included in this study after undergoing selection based on inclusion and exclusion criteria. The majority of patients were female (54.8%), with a mean age of 42.5 years. Based on age classification, most patients were in the 30–60 year range (74.1%), while 16.0% were under 35 years old, and 9.6% were over 60 years old. Blood pressure varied among participants, with 48.4% having normal blood pressure, 12.9% classified as high normal, and the remainder diagnosed with stage 1 hypertension (29.0%) or stage 2 hypertension (9.7%). In terms of anthropometry, most patients were obese, with 58.1% classified as grade II obesity, while only 9.7% had a normal body weight. Central obesity, as determined by waist circumference, was present in 93.5% of patients. Lipid profile analysis revealed that the majority had hypercholesterolemia (64.6%) and hypertriglyceridemia (58.1%), while 25.8% had low high-density lipoprotein (HDL) levels and 93.5% had high low-density lipoprotein (LDL) levels. Regarding metabolic parameters, 48.4% of patients had normal fasting blood glucose levels, while 32.2% had impaired fasting glucose, and 19.4% had hyperglycemia. Liver function evaluation showed that 16.1% of patients had

elevated AST levels, while 35.5% had increased alanine aminotransferase (ALT) levels. According to transient elastography, the majority of patients were in the F0-1 category (83.9%), while 9.7% were classified as F2, and 6.5% as F4. Ultrasound findings indicated that 51.6% had severe fatty liver, while 22.6% had mild fatty liver, and 25.8% had moderate fatty liver. The overall baseline characteristics of the patients are summarized in Table 1.

Table 1. Baseline characteristics of patients included in our study

Characteristics	n = 31
Gender	
Male	14 (45.2)
Female	17 (54.8)
Age (years)	42.5 ± 13.6
Age Category	
<35 years	5 (16.0)
30 – 60 years	23 (74.1)
>60 years	3 (9.6)
Blood Pressure (mmHg)	
Normal (≤120/89)	15 (48.4)
High Normal (120-139 / 80-89)	4 (12.9)
Hypertension Stage 1 (140-159 / 90-99)	9 (29.0)
Hypertension Stage 2 (≥160/100)	3 (9.7)
Anthropometry	
Body Mass Index (kg/m²)	
Underweight (<18.5)	0 (0)
Normal (18.5-22.9)	3 (9.7)
Overweight (23-24.9)	3 (9.7)
Obesity Grade I (25-29.9)	7 (22.6)
Obesity Grade II (>29.9)	18 (58.1)
Weist Circumference	
Normal	2 (6.5)
Central Obesity (Male >90 cm, Female >80 cm)	29 (93.5)
Upper Arm Circumference	31.2 ± 5.1
Body Fat Mass	30.5 ± 6.5
Body Muscle Mass	26.5 ± 4.6
Lipid Profile	
Total Cholesterol (mg/dL)	244.2 ± 54.7
Normal,	11 (35.4)
Hypercholesterolemia	20 (64.6)
Triglyceride (mg/dL)	176.9 ± 90.6
Normal	13 (41.9)
Hypertriglyceridemia	18 (58.1)
HDL (mg/dL)	47.5 ± 10.8
Normal	23 (74.2)
Hypo-HDL	8 (25.8)
LDL (mg/dL)	139.2 ± 39.5
Normal	2 (6.5)
Hyper-LDL	29 (93.5)
Fasting Blood Sugar	
Normal (<100 mg/dL)	15 (48.4)
Impaired Fasting Blood Sugar (100-125 mg/dL)	10 (32.2)
Hyperglycemia (>126 mg/dL)	6 (19.4)

Liver Function	
AST (IU/L)	27.68 ± 15.32
Normal (0-40)	26 (83.9)
Increased (>40)	5 (16.1)
ALT (IU/L)	37.56 ± 20.42
Normal (0-40)	20 (64.5)
Increased (>40)	11 (35.5)
Transient Elastography	
F0-1	26 (83.9)
F2	3 (9.7)
F4	2 (6.5)
Ultrasound (USG)	
Mild Fatty Liver	7 (22.6)
Moderate Fatty Liver	8 (2.6)
Severe Fatty Liver	16 (51.6)

Note, Values are mean ± SD or n (%); AST, aspartate aminotransferase; ALT, alanine aminotransferase; BMI, body mass index; HDL, high-density lipoprotein; LDL, low-density lipoprotein; USG, ultrasonography; FBG, glucose fasting blood sugar; F0-1, no to mild fibrosis; F2, significant fibrosis; F4, cirrhosis.

Factors associated with NAFLD

The risk factors associated with non-alcoholic NAFLD in this study were analyzed, revealing several significant associations. The analysis showed that BMI ≥23 kg/m² (OR: 23.516; 95% CI: 0.001-0.002; $p < 0.001$) and waist circumference exceeding normal limits (OR: 23.516; 95% CI: 0.001-0.002; $p < 0.001$) were significantly correlated with the incidence of NAFLD. Additionally, age (OR: 23.290; 95% CI: 0.001-0.002; $p = 0.029$) and dyslipidemia (OR: 27.129; 95% CI: 0.000-0.001; $p < 0.001$) also demonstrated significant associations. Conversely, blood pressure >120/80 mmHg ($p = 0.857$), fasting blood sugar ≥100 mg/dL ($p = 0.867$), AST levels >50 U/L ($p = 0.165$), ALT levels >50 U/L ($p = 0.611$), and gender ($p = 0.178$) did not show significant associations with NAFLD incidence. A summary of the risk factors associated with NAFLD in this study is presented in Table 2.

DISCUSSION

This study demonstrated a significant association between several variables and the incidence of NAFLD, namely age, BMI, abdominal circumference, and dyslipidemia. In contrast, other variables such as blood pressure, gender, fasting blood sugar levels, as well as AST and ALT levels did not show a significant influence. These findings align with previous study indicating that NAFLD is more prevalent in older populations, likely due to the increasing prevalence of metabolic syndrome, diabetes, and hypertension with age.[15] Additionally, the risk of liver fibrosis and non-alcoholic steatohepatitis (NASH) also rises with advancing age.[16] A recent systematic review identified older age as an independent predictor of advanced fibrosis. Therefore, our study aligns with previous findings and supports the role of these contributing factors in the incidence of NAFLD.[17]

Our results indicate that individuals with a high BMI (overweight / obese) have a 23.516-fold higher risk of developing fatty liver compared to the normal population. Several previous studies have also identified BMI as an independent risk factor for NAFLD. Another study found that 74% of patients with NAFLD had an abdominal circumference classified as central obesity.[18] BMI and abdominal circumference serve as linear indicators for determining NAFLD risk; however, these measurements do not

specifically reflect total body fat composition.[19] These findings suggest that weight reduction interventions through healthy dietary habits and physical activity could be an effective strategy for preventing and managing NAFLD.[20]

The underlying theory behind these findings is that NAFLD pathogenesis is closely related to hepatic fat accumulation, which is influenced by an unhealthy lifestyle, metabolic dysfunction, genetic factors, oxidative stress, insulin resistance, and obesity.[21] Insulin resistance leads to an increased release of free fatty acids (FFA) from peripheral adipose tissue, which subsequently results in fat accumulation in hepatocytes.[22] This lipid metabolism dysfunction can cause increased de novo lipogenesis, enhanced fatty acid oxidation, and impaired lipid export, particularly in the form of very low-density lipoproteins (VLDL).[23] Excessive hepatic fat accumulation, along with elevated VLDL production, contributes to atherogenic dyslipidemia, characterized by plasma hypertriglyceridemia, accumulation of triglyceride-rich lipoproteins, an increased number of small, dense LDL (sd-LDL) particles, and reduced HDL levels.[24] These mechanisms align with our study's findings, which highlight dyslipidemia as a key risk factor in the progression of NAFLD.

Table 2. Risk factors associated with non-alcoholic fatty liver disease in our study

Variables	Odds Ratio	95%CI	p
Blood Pressure >120/80 mmHg	0.032	0.010-1.000	0.8570
BMI ≥23 kg/m2	23.516	0.001-0.002	<0.001
Waist circumference	23.516	0.001-0.002	<0.001
Fasting Blood Sugar ≥100	0.034	0.010-1.000	0.8670
Age	23.290	0.001-0.002	0.0290
Dyslipidemia	27.129	0.000-0.001	<0.001
AST >50 U/L	0.577	0.010-1.000	0.1650
ALT >50 U/L	0.682	0.010-1.000	0.6110
Gender	0.290	0.702-0.726	0.1780

Note, BMI, body mass index; AST, aspartate aminotransferase; ALT, alanine aminotransferase.

This study bears multiple implications clinically. First, it underscores the necessity for early identification and management of all the key risk factors for the diseases, including obesity, dyslipidemia, and insulin resistance, so as to prevent disease progression. Second, the findings can indicate that the use of BMI or abdominal circumference as simple parameters can identify the great risk for NAFLD. Third, in addition, this may strengthen the rationale for further lifestyle interventions that improve insulin sensitivity such as putting a person on a low-carbohydrate diet and ensuring regular physical activity as relevant therapeutic lifestyle changes for the manifestation of NAFLD because of the importance of insulin resistance in the pathogenesis of the disease. Fourth, it also indicates that these should be put through further evaluation, such as measurement of the AST and ALT levels, as they are not always revealed in routine laboratory tests among NAFLD patients. Fifth, these points also make possible the ground for headway into subsequent innovative investigations that could further look into NAFLD as an emerging metabolic entity concerning other metabolic traits such as hypertension and body fat distribution.

There were several limitations in this study. First, the cross-sectional design impeded the quest for causality between the risk factors and NAFLD, and future cohort studies could have illuminated these causal associations more clearly. Second, the study sample was relatively small, most likely limiting the generalizability of the findings

across a broader population; indeed, larger studies with more diverse and bigger samples would have been more apt for enhancing the external validity of the results. Third, some patients were excluded from the study due to incomplete data, which may have affected the findings. Future studies could have adopted stricter collection criteria to reduce selection bias. Fourth, fatty liver was not classified according to its severity in this study; hence, the findings could not, by themselves, be viewed as a definitive point of reference for NAFLD risk factors. Advanced imaging modalities such as magnetic resonance imaging (MRI) or computed tomography (CT) would likely have needed to be incorporated into further studies to more accurately classify patients. Fifth, other assessments such as transient elastography (FibroScan) and liver biopsy, which would have provided more specific measures of hepatic fibrosis, were not included in this study. A comprehensive diagnostic method based on the progression of fibrosis and response to treatment should have characterized future studies.

CONCLUSION

This study demonstrates that age, BMI, abdominal circumference, and dyslipidemia have a significant impact on NAFLD. It further emphasizes the role of metabolic disorders, such as hypertension and insulin resistance, in the pathogenesis of NAFLD. These findings highlight the importance of early detection and intervention in managing metabolic risk factors to prevent disease progression. Future study with a longitudinal design and more advanced diagnostic methods is needed to clarify causal relationships and evaluate the effectiveness of NAFLD prevention strategies.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

The protocols of our study have been approved by local ethical committee.

CONFLICTS OF INTEREST

We have no conflict of interest

FUNDING SOURCES

We have no source of funding

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AUTHOR CONTRIBUTION

Conceptualization: SM; Data Curation: FR, IMS, IADS; Formal Analysis: FR, IMS, IADS; Investigation: FR, IMS, IADS; Project Administration: FR, IMS, IADS; Resources: FR, IMS, IADS; Methodology: FR, IMS, IADS; Software: FR, IMS, IADS; Visualization: FR, IMS, IADS; Supervision: SM; Validation: SM; Writing – Original Draft Preparation: FR, IMS, IADS; Writing – Review & Editing: SM. All authors have critically reviewed and approved the final draft and are responsible for the content and similarity index of the manuscript.

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