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Original Article

Correlation of steatosis degree with metabolic syndrome in patients with fatty liver disease-associated metabolic dysfunction (MAFLD)

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Abstract

Metabolic-associated fatty Liver Disease (MAFLD) is a growing global health concern. Hepatic steatosis is frequently associated with metabolic syndrome, which consists of obesity, insulin resistance, and hypertension. Understanding the correlation between the stages of hepatic steatosis and metabolic syndrome is essential for risk stratification and therapeutic planning. To investigate the correlation between the stages of hepatic steatosis and the presence of metabolic syndrome in MAFLD patients. This study is a cross-sectional analytical study conducted at Dr. Wahidin Sudirohusodo General Hospital, Makassar, Indonesia, from 2024 to 2025. A total of 126 patients with MAFLD were selected. Metabolic parameters data were collected, including BMI, waist circumference, blood pressure, lipid profile, fasting blood glucose, HbA1c, and the presence of metabolic syndrome. Hepatic steatosis stages were assessed via imaging and classified into grades 1 to 3. Statistical associations were evaluated using the Chi-square test with significance at p<0.05. Most of the patients were obese (82.5%), had elevated waist circumference (93.7%), and fulfilled criteria for metabolic syndrome (75.4%). Grade 1 steatosis was most prevalent (79.4%), followed by grades 2 (13.5%) and 3 (7.1%). A statistically significant association was observed between the severity of hepatic steatosis and BMI (p=0.031), hypertension (p=0.011), and metabolic syndrome (p=0.021). No significant associations were found with waist circumference, triglycerides, HDL, fasting blood glucose, diabetes status, or HbA1c. Severe hepatic steatosis is strongly correlated with obesity, hypertension, and metabolic syndrome in MAFLD patients. These results emphasize the essential role of systemic metabolic dysfunction in developing hepatic steatosis and stress the importance of meticulous management of metabolic risk factors in treating MAFLD.

Keywords: fatty liver disease, steatosis, metabolic syndrome

Introduction

Hepatic steatosis or fatty liver is a pathological condition characterized by the intracellular accumulation of lipids, predominantly triglycerides, within hepatocytes. It is defined by intrahepatic fat constituting at least 5% of the liver's weight. It is considered an early indicator of a range of liver pathologies, including non-alcoholic fatty liver disease (NAFLD). Although steatosis is potentially reversible, persistent lipid accumulation can progress to hepatic inflammation (steatohepatitis), fibrosis, cirrhosis, or even hepatocellular carcinoma if not appropriately managed [1].

Metabolic Associated Fatty Liver Disease (MAFLD) a recently adopted nomenclature replacing NAFLD, currently affects approximately 25% of the global adult population and represents a significant clinical and economic burden worldwide [2]. The increasing burden of MAFLD is closely related to rapid changes in lifestyle patterns, including decreased physical activity, high-energy intake, and the overall consumption of nutritionally unbalanced diets [3]. The new definition focuses on inclusive diagnostic criteria based on the presence of metabolic dysfunction, rather than the exclusion of significant alcohol consumption [4].

Data gathered from the Third National Health and Nutrition Examination Survey (NHANES III), encompassing 13,083 participants, indicate that the diagnostic criteria for MAFLD are more practical and demonstrate greater efficacy in identifying individuals at elevated risk than the former NAFLD framework [2, 5]. Furthermore, several large-scale, population-based studies have reported a marked and exponential increase in the prevalence of MAFLD across the Asia-Pacific region over the last three decades [2].

A comprehensive systematic review and meta-analysis including 13,044,518 individuals from Asian populations estimated the overall prevalence of MAFLD to be 29.62%. A complex interplay of genetic factors, dietary patterns, physical activity, lifestyle habits, and behavioral determinants modulates this figure. A meta-analysis of 18 studies estimated an annual MAFLD incidence of 50.9 cases per 1,000 individuals in Asian countries [5]. In Indonesia, prevalence rates have reached as high as 65.8% within specific subpopulations [6].

Research has demonstrated that individuals exhibiting higher grades of hepatic steatosis (grades 2 and 3) are more likely to present with key components of metabolic syndrome, including obesity, hypertension, insulin resistance, and dyslipidemia. Notably, MAFLD shares overlapping clinical characteristics with metabolic syndrome and is increasingly recognized as a condition fundamentally driven by insulin resistance [7]. Jinjuvadia *et al.* reported a 43% increase in NAFLD prevalence among individuals diagnosed with metabolic syndrome [8].

One of the primary challenges in the clinical care of MAFLD is identifying patients at risk for early progression to advanced disease stages. Surmounting this challenge requires comprehensive insight into both intrahepatic and extrahepatic drivers of disease progression, as well as the creation and implementation of valid, non-invasive diagnostic assays with high sensitivity and specificity [4].

Several investigations have consistently identified hepatic steatosis as an independent risk factor for the progression of hepatic fibrosis, a significant prognostic factor in patients with MAFLD. Additionally, the degree of steatosis has been shown to influence treatment outcomes and responses to medical treatment [9].

In light of these findings, it is essential to develop a more nuanced understanding of the relationship between the degree of hepatic steatosis and liver disease progression to inform the development of more effective prevention, diagnostic, and therapeutic strategies [9, 10].

This study aims to investigate the correlation between the stages of hepatic steatosis and metabolic syndrome among patients diagnosed with Metabolic-associated Fatty Liver Disease (MAFLD).

Material and methods

This study employs an analytical correlational design with a cross-sectional approach and was conducted at Dr. Wahidin Sudirohusodo General Hospital, Makassar, Indonesia, from 2024 to 2025. The study population consisted of all patients diagnosed with Metabolic-Associated Fatty Liver Disease (MAFLD) at the institution during the study period. Participants were selected based on the following inclusion criteria: (1) age over 18 years, (2) confirmed diagnosis of hepatic steatosis or fatty liver through imaging or other diagnostic modalities, and (3) fulfillment of the diagnostic criteria for MAFLD. A minimum sample size of 73 individuals was determined, and participants were recruited using a consecutive sampling method, whereby eligible individuals were enrolled continuously until the required sample size was achieved. Data analysis was performed using SPSS version 25.

Results

A total of 126 patients with MAFLD were enrolled in this study, comprising both inpatients and outpatients at Dr. Wahidin Sudirohusodo General Hospital during the period from November 2024 to March 2025. The participants ranged in age from 24 to 78 years, with a mean age of 51. Females comprised 54.8% of the sample, while males accounted for 45.2%. The majority of participants (82.5%) were classified as obese based on Body Mass Index (BMI), and a substantial proportion (93.7%) exhibited elevated waist circumference, indicating central obesity.

Hypertension was observed in 57.9% of participants, while the remaining 42.1% were normotensive. Elevated triglyceride levels were identified in 54% of the subjects, whereas 46% had levels within the normal range. Low levels of HDL cholesterol were observed in 43.7% of participants. Regarding glucose regulation, 45.2% of participants had normal fasting blood glucose, 22.2% were pre-diabetic, and 32.5% were diabetic. Consistently, based on HbA1c values, 41.3% were classified as normal, 15.9% as pre-diabetic, and 42.9% as diabetic. All subjects presented with hepatic steatosis, of which 79.4% had grade 1, 13.5% grade 2, and 7.1% grade 3. Furthermore, 75.4% of participants met the diagnostic criteria for metabolic syndrome, underscoring the comorbidity burden of this cohort (Table 1).

A statistically significant positive association was found between BMI and the severity of hepatic steatosis (p=0.031). Among participants categorized as obese (BMI≥23 kg/m^2), 16.3% had steatosis grade 2, and 8.7% had grade 3. In contrast, all individuals in the non-obese group exhibited only grade 1 steatosis, indicating that a higher BMI is significantly correlated with more severe hepatic steatosis.

Although waist circumference was elevated in most participants, its association with steatosis severity was not statistically significant (p=0.831). Among high-risk individuals based on waist circumference, 13.6% had grade 2 steatosis, and 6.8% had grade 3 steatosis, whereas in the low-risk group, 12.5% had grade 2 and 12.5% had grade 3 steatosis. The lack of statistical significance suggests that waist circumference, when

Table 1: Demographic and clinical characteristics.

Variable		n	%
Sex	Male	57	45.2
	Female	69	54.8
Age	<60-year-old	93	73.8
	≥60-year-old	33	26.2
BMI	Obese	104	82.5
	Non Obese	22	17.5
Waist circumference	High Risk	118	93.7
	Low Risk	8	6.3
Blood pressure	Hypertension	73	57.9
	Non-hypertension	53	42.1
TG	High Risk	68	54.0
	Low Risk	58	46.0
HDL	High Risk	55	43.7
	Low Risk	71	56.3
FBG	Normal	57	45.2
	Pre diabetic	28	22.2
	Diabetic	41	32.5
DM	Yes	59	46.8
	No	67	53.2
HbA1c	Normal	52	41.3
	Pre-diabetic	20	15.9
	Diabetic	54	42.9
Steatosis	Grade 1	100	79.4
	Grade 2	17	13.5
	Grade 3	9	7.1
Metabolic syndrome	Yes	95	75.4
	No	31	24.6

Note: BMI – Body Mass Index; TG – Triglyceride; HDL – High-Density Lipoprotein; FBG – Fasting Blood Glucose; HbA1c – Hemoglobin A1c Hemoglobin A1c.

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considered in isolation, may not reliably predict steatosis progression in this cohort.

We have established a significant relationship between steatosis severity and hypertension incidence (p=0.011). Among the hypertensive subgroup, 16.4% were affected by grade 2 steatosis, while 12.3% were affected by grade 3 steatosis. In contrast, the normotensive group did not have any cases of grade 3 steatosis. This indicates that hypertension can contribute to the development of hepatic steatosis in patients with the MAFLD diagnosis.

There were no statistically significant correlations between the grade of steatosis and levels of triglyceride (p=0.092), HDL cholesterol (p=0.198), fasting blood glucose levels (p=0.575), diabetic status (p=0.538), or HbA1c levels (p=0.690), although subtle trends were apparent.

On the other hand, metabolic syndrome was significantly associated with the severity of steatosis (p=0.021). Metabolic syndrome patients had more significant proportions of grade 2 (16.8%) and grade 3 (9.5%) steatosis than those without the syndrome (3.2% and 0%, respectively). These observations highlight the crucial role of systemic metabolic derangements in the pathogenesis and progression of hepatic steatosis in MAFLD patients (Table 2).

Discussion

MAFLD is closely linked with various metabolic parameters, including BMI, WC, TG, HT, HDL, FBG, HbA1c, and metabolic syndrome [2]. This study employed the Chi-square test to determine associations between these variables and the severity of hepatic steatosis. The resulting p-values offer insight into the strength of these associations [11].

A total of 126 people were included in the assessment. Demographic analysis revealed that 45.2% of the subjects were male and 54.8% were female. While an overall higher prevalence of MAFLD is more often seen in males, the risk rises significantly in females post-menopause. Zheng *et al.* (2020) corroborate the argument that gender distinction is also a firm predictor of the prevalence of NAFLD. The comparatively more significant percentage of females reported in the present study could indicate the effect of hormonal changes, especially on postmenopausal women.

73.8% of the subjects were aged 60 years or less, and 26.2% were 60 years or older. Sánchez-Rangel *et al.* (2021) reported that MAFLD is more prevalent in the younger population. However, hepatic steatosis may be more apparent in elderly individuals due to decreased capacity for lipid metabolism. This aligns with the age distribution observed in the present study [12].

A population-based study in Thailand involving 34,709 individuals found a higher prevalence of non-alcoholic fatty liver disease (NAFLD) in females (22.9%) compared to males (18.3%), with the most significant disparity observed in the 56–60 age group (27.4% in females *versus* 21.2% in males) [13].

Obesity emerged as the most prominent risk factor, with over 80% of the study population classified as obese. This finding reinforces earlier research by Bellentani and Marino (2017), who emphasized the strong association between obesity and hepatic steatosis. Visceral fat accumulation contributes to hepatic injury and metabolic dysregulation [12].

Waist circumference, a marker of visceral adiposity, was elevated in 93.7% of participants, indicating a high risk for hepatic fat accumulation. Ramirez *et al.* have highlighted the critical role of abdominal fat distribution in the pathogenesis of MAFLD [14].

Hypertension was present in 57.9% of participants. As a common component of metabolic syndrome, hypertension may exacerbate MAFLD through mechanisms involving hepatic inflammation and oxidative stress. This is supported by a meta-analysis conducted by Targher *et al.* (2010), which emphasized the synergistic effects of hypertension, dyslipidemia, and diabetes in accelerating hepatic injury [15].

Glycemic abnormalities were common: 22.2% of participants had prediabetes, and 32.5% had diabetes. Additionally, 42.9% exhibited elevated HbA1c levels. These findings are consistent with Bellentani and Marino (2017), who reported a strong association between type 2 diabetes and MAFLD. Chronic hyperglycemia promotes hepatic lipid accumulation, thereby increasing the risk of steatosis and disease progression [3].

Chi-square analysis revealed a statistically significant association between BMI and hepatic steatosis severity (p=0.031), underscoring the central role of obesity in disease development. An elevated BMI is associated with increased hepatic lipogenesis, primarily due to insulin resistance and disruptions in lipid metabolism. Younossi *et al.* (2021) and Stefan *et al.* (2023) reported that obesity induces pro-inflammatory cytokines that exacerbate hepatic inflammation and disease progression [16]. Lim *et al.* (2022) also suggested that visceral fat distribution can be a more accurate predictor of the development of MAFLD than BMI alone [17].

Obesity is also responsible for hypertriglyceridemia, which is capable of accelerating hepatic inflammation

Table 2: Correlation of metabolic component with steatosis.

Variable			Steatosis			Total	Chi-Square test (p)
			Grade 1	Grade 2	Grade 3		
BMI	Obese	n	78	17	9	104	0.031*
		%	75%	16.3	8.7%	100.0%	
	Non-obese	n	22	0	0	22	
		%	100%	0	0	100.0%	
WC	High risk	n	94	16	8	118	0.831
		%	79.7%	13.6%	6.8%	100.0%	
	Low risk	n	6	1	1	8	
		%	75%	12.5%	12.5%	100.0%	
BP	Hypertension	n	52	12	9	73	0.011*
		%	71.2%	16.4%	12.3%	100.0%	
	Non-hypertension	n	48	5	0	53	
		%	90.6%	9.4%	0.0%	100.0%	
TG	High risk	n	51	9	8	68	0.092
		%	75.0%	13.2%	11.8%	100.0%	
	Low risk	n	49	8	1	58	
		%	84.5%	13.8%	1.7%	100.0%	
HDL	High risk	n	47	4	4	55	0.198
		%	85.5%	7.3%	7.3%	100.0%	
	Low risk	n	53	13	5	71	
		%	74.6%	18.3%	7.0%	100.0%	
FBG	Normal	n	47	7	3	57	0.575
		%	82.5%	12.3%	5.3%	100.0%	
	Pre-diabetic	n	22	5	1	28	
		%	78.6%	17.9%	3.6%	100.0%	
	Diabetic	n	31	5	5	41	
		%	75.6%	12.2%	12.2%	100.0%	
DM	Yes	n	48	6	5	59	0.538
		%	81.3%	10.1%	8.6%	100%	
	No	n	52	11	4	67	
		%	77.6%	16.4%	6%	100%	
HbA1c	Normal	n	41	8	3	52	0.690
		%	78.8%	15.4%	5.8%	100.0%	
	Pre-diabetic	n	18	1	1	20	
		%	90.0%	5.0%	5.0%	100.0%	
	Diabetic	n	41	8	5	54	
		%	75.9%	14.8%	9.3%	100.0%	
Metabolic syndrome	Yes	n	70	16	9	95	0.021*
		%	73.7%	16.8%	9.5%	100.0%	
	No	n	30	1	0	31	
		%	96.8%	3.2%	0.0%	100.0%	

Note: BMI– Body Mass Index; WC – Waist Circumference; TG – Triglyceride; HDL – High-Density Lipoprotein; FBG – Fasting Blood Glucose; HbA1c – Hemoglobin A1c; FLI – Fatty Liver Index; *– statistically significant.

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and lipid deposition. This process results in hepatocyte damage and augmented oxidative stress, as Stefan *et al.* (2023) explained [12]. Additionally, systemic inflammation driven by cytokines such as TNF-α and IL-6 further exacerbates hepatic damage and promotes progression to metabolic-associated steatohepatitis (MASH), as demonstrated by Zhang *et al.* (2023) [12].

Despite its known association with visceral adiposity, waist circumference did not show a statistically significant relationship with steatosis severity in this study. Although previous studies (*e.g.*, Kim *et al.*, 2017; Zhao *et al.*, 2019) have found waist circumference to be a reliable indicator of MAFLD risk due to its association with insulin resistance and inflammation, the current findings suggest that waist circumference alone may not be a sufficient predictor of steatosis severity [18]. This discrepancy may be due to confounding factors or the multifactorial nature of hepatic lipid accumulation.

Hypertension, however, had a significant relationship with hepatic steatosis ($p<0.05$). Fu *et al.*'s (2023) research on a population of 4,705 patients showed that hypertension alone increased the risk of hepatic steatosis (OR=1.4; 95% CI: 1.1–1.8). High blood pressure can be a causal factor in hepatic inflammation and fibrosis, underscoring the importance of comprehensive cardiovascular risk management in patients with MAFLD [19, 20].

To our surprise, triglyceride concentrations were not shown to correlate substantially with the degree of steatosis. Despite being a frequent finding among MAFLD patients, the transient nature of increased triglyceride levels could limit their effectiveness in predicting the degree of fat accumulation. The research conducted by Younossi *et al.* (2021) and Than *et al.* (2022) revealed that hypertriglyceridemia was more associated with fibrosis and MASH advancement than with initial fat accumulation [21].

Similarly, no statistically significant associations were found between steatosis severity and low HDL levels ($p=0.198$), fasting blood glucose ($p=0.575$), diabetes status ($p=0.538$), or HbA1c ($p=0.690$). While these factors are central to the pathophysiology of MAFLD, their impact on steatosis severity may be overshadowed by more dominant factors such as obesity and hypertension. Moreover, previous studies (*e.g.*, Petta *et al.*, 2020; Hazlehurst *et al.*, 2016) have suggested that these variables may be more relevant to predicting fibrosis and advanced disease rather than steatosis *per se* [22].

In contrast, metabolic syndrome is significantly associated with steatosis severity ($p=0.021$). This finding is consistent with prior research (Younossi *et al.*, 2021; Than *et al.*, 2022; Stefan *et al.*, 2023), which has established that the cumulative burden of metabolic risk factors—particularly central obesity, hypertension, and dyslipidemia—substantially increases the risk of advanced hepatic steatosis. The presence of metabolic syndrome may reflect a systemic metabolic derangement that directly drives hepatic fat accumulation and disease progression [23].

Conclusion

This study showed that obesity, hypertension, and the presence of metabolic syndrome are significantly linked to the severity of hepatic steatosis in patients with Metabolic-Associated Fatty Liver Disease (MAFLD), but not waist circumference, triglycerides, HDL, fasting blood glucose, diabetes, or HbA1c. The results demonstrate how important systemic metabolic dysfunction is in promoting the development of liver fat accumulation, especially when it comes to excess body weight and high blood pressure. These findings highlight the necessity of thorough metabolic risk management for MAFLD patients in order to prevent the disease from progressing and enhance clinical results.

Conflict of interest

The authors declare no conflict of interest.

Ethics approval

Ethical approval for this study was obtained from the Biomedical Research Ethics Committee on Human Subjects, Faculty of Medicine, Hasanuddin University (approval ID: 930/UN4.6.4.5.31/PP36/2024).

Consent to participate

Written informed consent was obtained from all the participants.

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