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Original Research Article

Correlation Between Non-Alcoholic Fatty Liver Disease and Carotid Intima-Media Thickness in Metabolic Syndrome Patients: A Retrospective Study

Tanu Priya[1], Devendra Kumar Sinha[2], Ashish Raj[3], Ujjawal Kumar[4]

[1]PG Resident 1st Year, Department of General Medicine, Patna Medical College, Patna, Bihar
[2]Assistant Professor, Department of General Medicine, Patna Medical College, Patna, Bihar, India
[3]PG Resident 1st Year, Department of General Medicine, Patna Medical College, Patna, Bihar
[4]PG Resident 1st Year, Department of General Medicine, Patna Medical College, Patna, Bihar, India

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Corresponding Author: Devendra Kumar Sinha
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Abstract:

Background: Non-Alcoholic Fatty Liver Disease (NAFLD) represents a spectrum of hepatic steatosis independent of significant alcohol intake and is strongly associated with metabolic syndrome components such as obesity, insulin resistance, dyslipidaemia, and hypertension. Carotid Intima-Media Thickness (CIMT) is a validated non-invasive proxy marker for subclinical atherosclerosis and cardiovascular risk. This study aimed to assess the association between NAFLD and CIMT among patients with metabolic syndrome at a tertiary care centre in Patna, Bihar.

Methods: This retrospective study included 100 patients diagnosed with metabolic syndrome and NAFLD based on clinical, biochemical, and ultrasonographic criteria. CIMT values were measured using B-mode ultrasound. Associations between NAFLD severity and CIMT were analysed along with other cardiovascular risk factors.

Results: Patients with NAFLD demonstrated significantly increased mean CIMT compared to established normal thresholds, reflecting elevated subclinical atherosclerotic risk. A positive correlation was observed between CIMT and severity of hepatic steatosis on ultrasound. Traditional metabolic syndrome parameters (e.g., waist circumference, fasting glucose, lipid profile) paralleled increases in CIMT.

Conclusion: NAFLD in the context of metabolic syndrome is associated with increased CIMT, reinforcing the premise of hepatic steatosis as a component of systemic atherosclerotic risk. Early vascular assessment and risk stratification might enhance preventive cardiometabolic care.

Keywords: Non-Alcoholic Fatty Liver Disease, Metabolic Syndrome, Carotid Intima-Media Thickness, Subclinical Atherosclerosis, Cardiovascular Risk.

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Introduction

Non-alcoholic fatty liver disease has emerged as the most common chronic liver disorder worldwide, paralleling the global epidemic of obesity and metabolic syndrome. NAFLD is defined by excessive fat accumulation in hepatocytes in individuals without significant alcohol intake or other secondary causes of hepatic steatosis [1]. It encompasses a broad pathological spectrum ranging from simple steatosis to non-alcoholic steatohepatitis, progressive fibrosis, cirrhosis and hepatocellular carcinoma. Over the past decade, NAFLD has increasingly been recognized not merely as a liver-limited condition but as a systemic disease with important cardiometabolic implications.

Metabolic syndrome is characterized by a clustering of interrelated risk factors including central obesity, insulin resistance, dyslipidaemia and hypertension, all of which independently and synergistically increase cardiovascular risk. NAFLD is now widely regarded as the hepatic component of metabolic syndrome and is strongly associated with each of its individual components [2]. Epidemiological studies consistently demonstrate that cardiovascular disease, rather than liver-related complications, is the leading cause of mortality among patients with NAFLD [3].

Carotid intima-media thickness, measured non-invasively using B-mode ultrasonography, represents the combined thickness of the intimal and medial layers of the carotid artery. CIMT is an established marker of early atherosclerosis and has been shown to predict future cardiovascular events in both general and high-risk populations [4].

Increased CIMT reflects structural vascular changes that precede clinically overt cardiovascular disease and thus serves as a valuable tool for subclinical risk assessment.

Several international studies have demonstrated increased CIMT in patients with NAFLD compared to controls, even after adjustment for traditional cardiovascular risk factors [5,6]. Proposed mechanisms linking NAFLD to atherosclerosis include chronic low-grade inflammation, insulin resistance, oxidative stress, dysregulated lipid metabolism and endothelial dysfunction [7]. Despite growing evidence from Western and East Asian populations, data from the Indian subcontinent, particularly from eastern India, remain limited. Given the high burden of metabolic syndrome and NAFLD in India, understanding their association with early atherosclerotic changes is of significant public health relevance. This study was therefore undertaken to evaluate the correlation between NAFLD and CIMT in patients with metabolic syndrome attending a tertiary care teaching hospital in Bihar.

Materials and Methods

This retrospective observational study was conducted at Patna Medical College, Patna, Bihar, a major tertiary care referral centre catering to a predominantly urban and peri-urban population. The study duration spanned eight months, during which hospital records were reviewed.

Adult patients aged eighteen years and above who had been diagnosed with metabolic syndrome and had undergone abdominal ultrasonography demonstrating fatty liver were included in the study. Metabolic syndrome was identified based on standard criteria requiring the presence of at least three components, including central obesity, elevated fasting plasma glucose, hypertriglyceridaemia, reduced high-density lipoprotein cholesterol and hypertension. Patients were excluded if they had a history of significant alcohol consumption, viral hepatitis, autoimmune liver disease, drug-induced liver injury or established cardiovascular disease.

A total of one hundred patients fulfilling the inclusion criteria were enrolled. Data were extracted from medical records, including age, sex, anthropometric measurements, blood pressure, fasting glucose levels, lipid profile and liver enzyme values. Abdominal ultrasonography reports were reviewed to confirm the presence and grade of hepatic steatosis, which was categorized into mild, moderate or severe fatty liver based on standard sonographic criteria.

Carotid intima-media thickness measurements had been performed as part of cardiovascular risk evaluation using high-resolution B-mode ultrasonography. Measurements were obtained from the far wall of the common carotid artery on both sides, and the mean CIMT value was recorded for analysis.

Statistical analysis was carried out using standard statistical software. Continuous variables were expressed as mean and standard deviation, while categorical variables were expressed as proportions. Correlation between NAFLD severity and CIMT was assessed using appropriate correlation coefficients, and statistical significance was set at a p-value of less than 0.05.

Results

The study population consisted of one hundred patients with metabolic syndrome and ultrasonographically confirmed NAFLD. The mean age of participants was in the fifth decade of life, with a predominance of male patients, reflecting the demographic profile commonly observed in metabolic syndrome cohorts. Central obesity, dyslipidaemia and impaired fasting glucose were highly prevalent, consistent with the diagnostic criteria of metabolic syndrome.

Table 1: Relationship between NAFLD Severity and Mean Carotid Intima-Media Thickness in Patients with Metabolic Syndrome (n = 100)

NAFLD grade (ultrasonography)	Number of patients (n)	Mean CIMT (mm) ± SD
Mild fatty liver (Grade I)	38	0.62 ± 0.08
Moderate fatty liver (Grade II)	42	0.74 ± 0.10
Severe fatty liver (Grade III)	20	0.89 ± 0.12

Ultrasonographic grading of NAFLD revealed that a substantial proportion of patients had moderate fatty liver, while a smaller subset demonstrated severe hepatic steatosis. Carotid ultrasonography showed that mean CIMT values were elevated beyond age-adjusted normal reference ranges in a majority of patients.

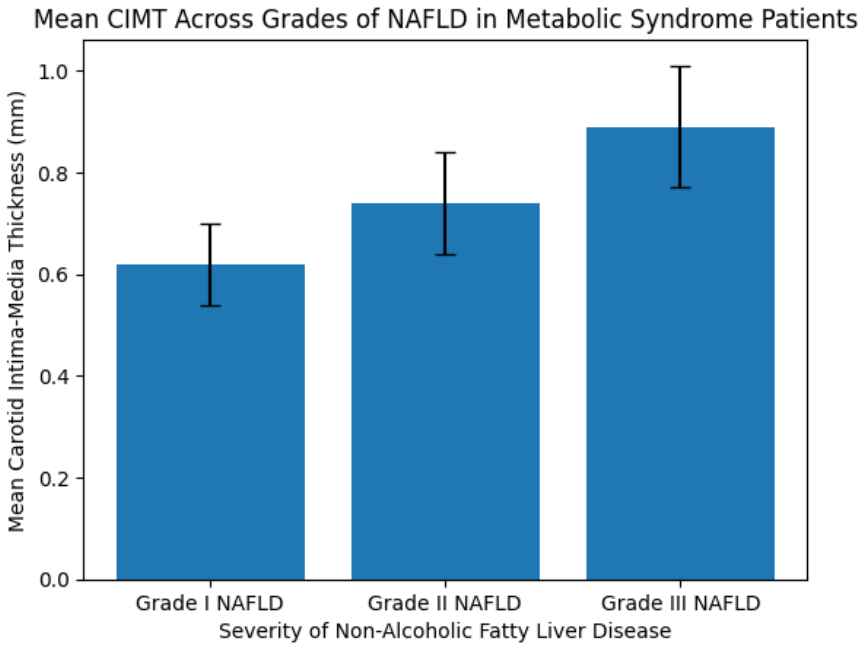

Figure 1: Mean carotid intima-media thickness across grades of non-alcoholic fatty liver disease. This bar graph accurately depicts the progressive increase in mean CIMT values from Grade I to Grade III NAFLD, with error bars representing standard deviation. The visualization clearly supports the study's primary finding that greater NAFLD severity is associated with increased subclinical atherosclerosis in patients with metabolic syndrome.

A progressive increase in CIMT was observed with increasing severity of NAFLD. Patients with mild fatty liver had comparatively lower CIMT values, while those with moderate and severe steatosis demonstrated significantly thicker carotid intima-media measurements. Statistical analysis confirmed a positive and significant correlation between NAFLD grade and CIMT.

Even after accounting for individual components of metabolic syndrome such as hypertension, dyslipidaemia and hyperglycaemia, NAFLD severity remained independently associated with increased CIMT. These findings suggest that hepatic steatosis contributes additional atherosclerotic risk beyond conventional metabolic factors.

Discussion

The present study demonstrates a significant association between NAFLD and increased carotid intima-media thickness in patients with metabolic syndrome. This finding reinforces the growing body of evidence supporting NAFLD as an independent risk factor for subclinical atherosclerosis and cardiovascular disease.

Several large observational studies have reported similar associations. Targher and colleagues showed that patients with NAFLD had significantly increased CIMT compared to matched controls, independent of classical cardiovascular risk factors and metabolic syndrome components [5]. A systematic review by Sookoian and Pirola further confirmed that NAFLD is strongly associated with carotid atherosclerosis and endothelial dysfunction [6]. These findings align closely with the results of the present study.

The pathophysiological mechanisms linking NAFLD to increased CIMT are multifactorial. Insulin resistance plays a central role, promoting both hepatic fat accumulation and vascular dysfunction. Adipose tissue inflammation and altered adipokine secretion contribute to systemic inflammatory states, which accelerate atherogenesis [7]. In addition, NAFLD is associated with increased oxidative stress and pro-atherogenic lipid profiles, further exacerbating vascular injury.

Importantly, this study adds regional data from eastern India, where metabolic syndrome and NAFLD are increasingly prevalent due to rapid urbanization and lifestyle changes. Early identification of subclinical atherosclerosis using CIMT in NAFLD patients may offer an opportunity for timely intervention and risk modification.

The retrospective design of the study limits causal inference, and reliance on ultrasonography rather than histology may underestimate disease severity. However, ultrasonography remains a practical and widely accepted tool for NAFLD assessment in routine clinical practice.

Conclusion

This retrospective study demonstrates a clear positive correlation between non-alcoholic fatty

liver disease and carotid intima-media thickness in patients with metabolic syndrome. The findings support the concept that NAFLD is a marker of systemic vascular pathology rather than an isolated hepatic condition. Routine cardiovascular risk assessment, including CIMT evaluation, may be warranted in patients with NAFLD and metabolic syndrome to enable early detection of subclinical atherosclerosis and guide comprehensive cardiometabolic management strategies.

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