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Non-Alcoholic Fatty Liver Disease and Its Association with Cardiovascular Risk Factors: A Cross-Sectional Study

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ABSTRACT

Introduction: Non-alcoholic fatty liver disease is the most common chronic liver disease worldwide, closely associated with obesity, type 2 diabetes mellitus and metabolic syndrome. Beyond hepatic complications, NAFLD is increasingly recognized as a multisystem disorder with cardiovascular disease being the leading cause of mortality. The present study aimed to evaluate the prevalence of NAFLD and its correlation with cardiovascular risk factors.

Methods: A retrospective cross-sectional analysis was conducted in Stoke-on-Trent, UK on 183 adult patients diagnosed with NAFLD by ultrasonography. Patients with significant alcohol intake, viral hepatitis, autoimmune or metabolic liver disease were excluded. Demographic, clinical, and biochemical data including cardiovascular risk factors were analyzed. Statistical tests included Chi-square, t-test/ANOVA, and binory logistic regression, with $p < 0.05$ considered significant.

Results: The mean age of participants was 44.8 ± 11.2 years, with a male predominance (64.5%). The most prevalent cardiovascular risk factors were obesity (74.9%) and dyslipidemia (66.1%), followed by hypertension (50.3%), diabetes mellitus (42.6%), and smoking (26.8%). Higher NAFLD grades were significantly associated with increased prevalence of hypertension, diabetes, and dyslipidemia. Biochemical parameters including fasting blood sugar, cholesterol, and triglycerides worsened with disease severity, while HDL levels declined (all $p < 0.05$). Obesity, diabetes, and hypertension as independent predictors of advanced NAFLD.

Conclusion: NAFLD is associated with obesity, diabetes, and hypertension, which also predict disease severity. These findings highlight the need for early screening, comprehensive risk assessment, and integrated management strategies to address both hepatic and cardiovascular outcomes in NAFLD patients.

Keywords: Cardiovascular disease, Diabetes mellitus, Dyslipidemia, Hypertension, Metabolic syndrome, Non-alcoholic fatty liver disease, Obesity, Ultrasonography

INTRODUCTION

Metabolic dysfunction-associated steatotic liver disease (MASLD), previously termed non-alcoholic fatty liver disease (NAFLD), has emerged as the most prevalent chronic liver disorder worldwide, reflecting the global rise in obesity, type 2 diabetes mellitus (T2DM), and metabolic syndrome.[1]

MASLD encompasses a disease spectrum ranging from simple hepatic steatosis to steatohepatitis, progressive fibrosis, cirrhosis, and hepatocellular carcinoma.[2] In South Asia and Western countries alike, the burden of fatty liver disease continues to rise, posing a major public health challenge.[3]

In 2023, international hepatology societies proposed a paradigm shift in nomenclature from NAFLD to MASLD to better reflect the central role of metabolic dysfunction in disease pathogenesis and to move away from a diagnosis of exclusion.[4]

MASLD is increasingly recognized as a multisystem disease with important extrahepatic complications. Cardiovascular disease is the leading cause of mortality in these patients, exceeding deaths due to liver failure or hepatocellular carcinoma. Factors such as insulin resistance, chronic inflammation, oxidative stress, and dyslipidemia contribute to this association.[4] Studies have also shown that fatty liver disease is closely linked with hypertension, abnormal lipid levels, impaired glucose metabolism, and increased risk of cardiovascular events.

Despite growing global evidence, data examining the relationship between fatty liver disease severity and cardiovascular risk factors in real-world clinical populations remain limited, particularly in routine care settings. Moreover, few studies have simultaneously evaluated the burden of multiple cardiovascular risk factors and their independent contribution to disease severity.

With this background, the present study was undertaken to assess the distribution and severity of NAFLD and to evaluate its association with cardiovascular risk factors in an outpatient cohort. This may provide insight into the dual burden of liver and cardiovascular disease and show the need for integrated management strategies.

MATERIALS AND METHODS

This study was designed as a retrospective cross-sectional analysis done in the at Stoke on Trent, MRCGP, UK. A total of 183 patients were included in the study based on inclusion and exclusion criteria during study period. The study was carried out after obtaining approval from the Institutional Ethics Committee, and patient confidentiality was strictly maintained.

The medical records of adult patients (≥18 years) who attended the outpatient department or were admitted between January 2022 to December 2023 were reviewed. Patiecnts with evidence of hepatic steatosis on abdominal ultrasonography were considered for inclusion.

The study included patients aged 18 years and above who were diagnosed with non-alcoholic fatty liver disease (NAFLD) based on ultrasonographic findings and had complete clinical as well as biochemical data available. Patients were excluded if they had a history of significant alcohol consumption, defined as more than 30 g/day for men and more than 20 g/day for women, or if they were known cases of chronic viral hepatitis (HBV or HCV), autoimmune liver disease, Wilson's disease, hemochromatosis, or drug-induced liver injury. Individuals with incomplete records or missing essential data were also excluded from the study.

Patient information was retrieved from hospital records and electronic databases. Demographic details such as age and sex were recorded. Clinical variables included history of hypertension, diabetes mellitus, smoking, and obesity. Anthropometric data (weight, height, body mass index [BMI]) were noted. Laboratory parameters including

fasting blood sugar, lipid profile (total cholesterol, HDL, triglycerides) were collected. Blood pressure recordings and details of comorbid conditions were also documented.

Diagnosis of NAFLD was based on ultrasonographic evidence of hepatic steatosis performed by a qualified radiologist. Grading of fatty liver (Grade I-III) was also recorded for all patients.

Cardiovascular risk factors were assessed based on established clinical and biochemical criteria. Hypertension was identified in patients with a documented diagnosis or those receiving antihypertensive medications.[5] Diabetes mellitus was defined by a fasting blood glucose level of ≥126 mg/dl, HbA1c ≥6.5%, or the use of anti-diabetic drugs.[6] Dyslipidemia was diagnosed in cases where total cholesterol was ≥200 mg/dl, triglycerides ≥150 mg/dl, or high-density lipoprotein (HDL) levels were <40 mg/dl in men and <50 mg/dl in women, or if patients were on lipid-lowering therapy.[7] Obesity was determined using the body mass index (BMI), with a cutoff of ≥25 kg/m^2 as per Asian guidelines.[8] Smoking status was recorded based on a current or past history of smoking.

Data were entered into Microsoft Excel and analyzed using Statistical Package for the Social Sciences (SPSS) software version 25. Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were presented as frequency and percentage [n (%)]. Comparisons between groups were performed using Student's t-test or ANOVA for continuous variables and Chi-square test for categorical variables. Logistic regression analysis was used with enter method and Hosmer-Lemeshow test used to check model fit. A p-value of <0.05 was considered statistically significant.

RESULTS

The mean age of the study participants was 44.8 ± 11.2 years, with a male predominance. The majority of patients were overweight or obese. (Table 1)

Obesity and dyslipidemia were the most common cardiovascular risk factors in NAFLD patients, followed by hypertension and diabetes mellitus. (Table 2)

Table 1: Demographic Profile of Study Participants (n = 183)

Variable	Mean ± SD / n (%)
Age (years)	44.8 ± 11.2
Sex (Male)	118 (64.5%)
Sex (Female)	65 (35.5%)
BMI (kg/m^2)	27.4 ± 3.8

Table 2: Prevalence of Cardiovascular Risk Factors in Patients with NAFLD

Risk Factor	n (%)
Hypertension	92 (50.3%)
Diabetes Mellitus	78 (42.6%)
Dyslipidemia	121 (66.1%)
Obesity (BMI ≥25 kg/m^2)	137 (74.9%)
Smoking (current/past)	49 (26.8%)

Among the 183 patients included in the study, Grade I NAFLD was the most common finding, observed in 87 patients (47.5%). Grade II NAFLD was identified in 63 patients (34.4%), while Grade III NAFLD, representing the most severe form, was present in 33 patients (18.0%). The prevalence of hypertension, diabetes, and dyslipidemia significantly increased with higher grades of fatty liver (p <0.05). (Figure 1)

Biochemical parameters worsened with progression of NAFLD grade. Patients with Grade III fatty liver had significantly higher fasting blood sugar, cholesterol, and triglyceride levels, along with lower HDL levels compared to Grades I and II. (Table 3)

Obese patients with NAFLD showed a significantly higher prevalence of hypertension, diabetes, and dyslipidemia compared to non-obese patients (p <0.001). Interestingly, smoking was more frequent in the non-obese group. (Table 4)

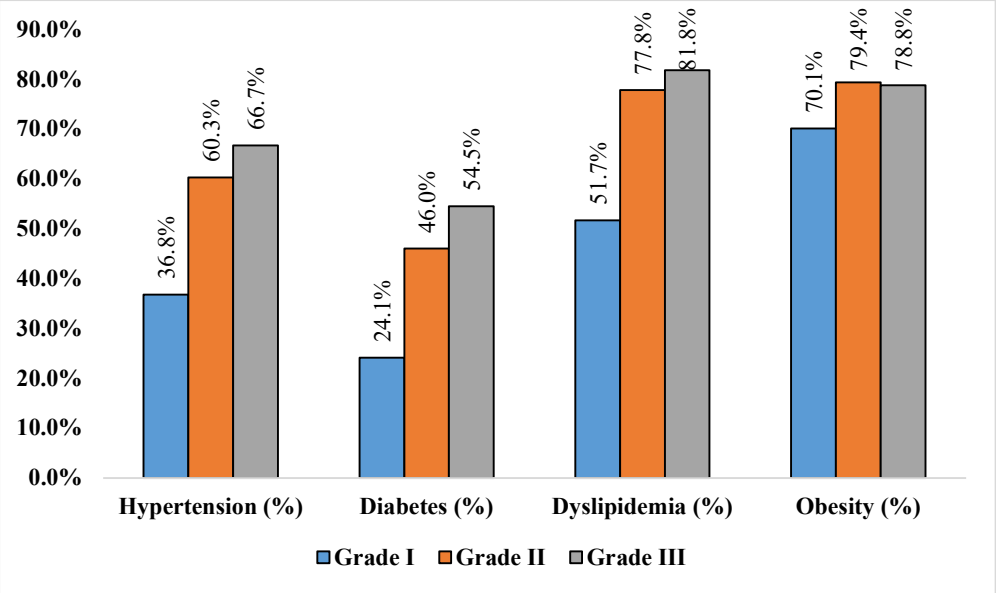

Figure 1: Grading of Fatty Liver on Ultrasonography and Associated Cardiovascular Risk Factors

Table 3: Comparison of Biochemical Parameters Across NAFLD Grades

Parameter	Grade I (n=87) Mean ± SD	Grade II (n=63) Mean ± SD	Grade III (n=33) Mean ± SD	p-value
Fasting Blood Sugar (mg/dl)	109.2 ± 21.5	124.3 ± 26.4	133.6 ± 29.1	<0.001*
Total Cholesterol (mg/dl)	187.6 ± 32.1	202.5 ± 36.7	218.4 ± 41.2	0.002*
Triglycerides (mg/dl)	148.7 ± 44.5	174.9 ± 52.1	196.8 ± 55.6	<0.001*
HDL (mg/dl)	42.5 ± 8.7	38.1 ± 7.4	36.2 ± 6.9	0.01*

*Significant

Table 4: Association of Obesity with Cardiovascular Risk Factors

Variable	Obese (n=137)	Non-obese (n=46)	p-value
Hypertension	81 (59.1%)	11 (23.9%)	<0.001*
Diabetes Mellitus	71 (51.8%)	7 (15.2%)	<0.001*
Dyslipidemia	104 (75.9%)	17 (37.0%)	<0.001*
Smoking	31 (22.6%)	18 (39.1%)	0.04*

*Significant

Table 5: Logistic Regression Analysis of Independent Predictors of NAFLD Severity

Variable	Odds Ratio (OR)	95% CI	p-value
Age >45 years	1.9	1.1-3.4	0.02*
Male sex	1.4	0.8-2.5	0.21
Hypertension	2.1	1.2-3.8	0.01*
Diabetes Mellitus	2.5	1.3-4.6	0.003*
Dyslipidemia	1.8	1.1-3.2	0.04*
Obesity	2.9	1.5-5.4	0.001*

*Significant

Multivariate logistic regression showed that obesity, diabetes, and hypertension were the strongest independent predictors of higher-grade NAFLD. Male sex did not emerge as a significant predictor in this analysis. The Hosmer-Lemeshow test showed a non-significant p-value (>0.05), indicating good model fit. The Nagelkerke R^2 of 0.32 suggests

that approximately 32% of the variability in NAFLD severity is explained by the predictors in the model, with an overall classification accuracy of 76.5%. (Table 5)

DISCUSSION

In our study, the majority of cases fell within the 30-60 years age group as per MASLD criteria which was renamed by Delphi consensus. This finding is consistent with the report by Shil BC et al.[9], who also observed the peak prevalence of NAFLD between 31 and 60 years, with age over 30 years acting as a significant predictive factor along with metabolic risk parameters. Similarly, Lin Y et al.[10] reported that the incidence of NAFLD increased progressively with age, reaching as high as 22.8% in those older than 65 years, while Liu C et al.[11] showed a mean onset age of around 51 years, showing the wide age distribution of NAFLD and its rise with advancing age

Our study male predominance is similar to findings from Nagral A et al.[12], who documented a higher prevalence of NAFLD in men compared to women across most age groups, and from G Saravanan et al.[13], who showed a male predominance (71%) in the younger 18-35 age group. Lonardo A et al.[14] concluded that estrogen is protective during reproductive years, and after menopause, women show a higher risk, which in some cohorts even surpasses men.

In our study majority were BMI more than 25. similarly, Bhaumik R et al.[15] reported that over 60% of NAFLD patients were overweight or obese, with BMI correlating directly with liver enzyme levels. Abangah G et al.[16] further confirmed obesity as one of the strongest predictors of ultrasonographic severity of NAFLD, with over 70% of their patients being overweight or obese.

The prevalence of hypertension in our study (50.3%) was higher than the general population estimates and also somewhat above the 25-40% range reported in previous studies. Ma C et al.[17] reported hypertension prevalence at 39.3% among NAFLD patients, while Ng CH et al.[18] noted rates as high as 45.65%, with uncontrolled hypertension present in 35.12%.

In our study, 42.6% of NAFLD patients had diabetes mellitus, which lies at the lower end of the 40-70% range reported globally. En Li Cho E et al.[19] in a large meta-analysis estimated NAFLD prevalence in T2DM at about 65%, with 31.5% progressing to NASH. Kumar P et al.[20] similarly showed the strong overlap between diabetes and NAFLD.

Chalasani N et al.[21] documented lipid abnormalities in 50-80% of NAFLD patients. Martin A et al.[22] in a meta-analysis reported combined dyslipidemia prevalence around 69%, with hypertriglyceridemia affecting up to 83% of NASH patients. Our results are similar to these findings, showing the central role of dyslipidemia in NAFLD pathogenesis and its link with cardiovascular risk.

In our study, 74.9% of NAFLD patients were obese (BMI ≥25 kg/m^2), confirming obesity as the leading risk factor. Similarly, Teng ML et al.[23] estimated that nearly 40% of overweight and obese individuals have NAFLD, with NASH affecting one-third of this group. Karjoo S et al.[24] further reported high NAFLD prevalence in Asian populations at lower BMI thresholds.

Our results support the evidence that smoking contributes to NAFLD risk and progression, showing the role of cessation as part of preventive strategies. Jang HR et al.[25] in a large Korean cohort showed significantly higher odds of NAFLD in both current and former smokers, with risk increasing with pack-years.

We observed that the prevalence of hypertension progressively increased from 36.8% in Grade I to 60.3% in Grade II and 66.7% in Grade III, with a statistically significant association ($p = 0.003$). This finding is similar to the meta-analysis by Li G et al.[26], which showed a bidirectional relationship between NAFLD and hypertension, showing higher risk and incidence of hypertension with advancing NAFLD severity. Ryoo JH et al.[27] also reported a dose-response pattern, with the incidence of hypertension

increasing from 14.4% in individuals with normal liver to over 30% in moderate-to-severe NAFLD. The shared mechanisms include insulin resistance, systemic inflammation, and altered adipokine signalling, which exacerbate both conditions.

The prevalence of diabetes in our cohort rose significantly with NAFLD grade, from 24.1% in Grade I to 46.0% in Grade II and 54.5% in Grade III ($p = 0.01$). These findings are consistent with Atan NAD et al.[28], who reported that NAFLD severity correlated with worse glycemic control (higher HbA1c and BMI) among diabetic patients. The pathophysiological link lies in worsening insulin resistance and β-cell dysfunction with increasing hepatic steatosis.

Dyslipidemia was present in 51.7% of Grade I patients, increasing significantly to 77.8% in Grade II and 81.8% in Grade III ($p = 0.02$). Martin A et al.[22] also showed that hypertriglyceridemia affects up to 83% of patients with advanced NAFLD/NASH, showing the graded relationship between disease severity and lipid abnormalities. These alterations substantially contribute to the heightened cardiovascular morbidity and mortality observed in NAFLD patients.

In our study shows that while obesity is a major driver of NAFLD, it is already highly prevalent in early stages and does not significantly discriminate across grades. Chen X et al[29] further showed that obesity indices such as BMI, waist circumference, and visceral adiposity index strongly correlate with NAFLD severity, although overlap exists.

We found that 59.1% of obese NAFLD patients had hypertension compared to 23.9% of non-obese, a highly significant association ($p < 0.001$). This is similar to the findings of Chambergo-Michilot D et al.[30], who reported a significant association between hypertension and severe NAFLD, even after adjusting for metabolic syndrome.

Obesity was also significantly associated with diabetes mellitus in our cohort, with 51.8% of obese versus 15.2% of non-obese NAFLD patients being diabetic ($p < 0.001$). This is similar to the study by Suryawanshi NV et al[31], who found that obese diabetics with NAFLD had higher HbA1c and worse glycemic indices compared to non-obese diabetics.

Similarly, dyslipidemia was more prevalent in obese (75.9%) than non-obese NAFLD patients (37.0%, $p < 0.001$). This observation is consistent with Zhu Z et al[32], who noted adverse lipid abnormalities in both obese and non-obese NAFLD, though obesity amplified lipid derangements. Zhang QQ et al.[33] described obesity and dyslipidemia as integral features of the metabolic syndrome contributing to NAFLD pathogenesis.

CONCLUSION

This retrospective cross-sectional study highlighted that non-alcoholic fatty liver disease (NAFLD) is strongly associated with multiple cardiovascular risk factors, particularly obesity, dyslipidemia, hypertension, and diabetes mellitus. The severity of NAFLD, as graded on ultrasonography, highlighted a significant correlation with worsening biochemical parameters and an increasing prevalence of these risk factors. Obesity and diabetes mellitus emerged as the strongest independent predictors of NAFLD severity. These findings underscore the need for early screening and comprehensive management of metabolic risk factors in patients with NAFLD to prevent progression of both liver disease and cardiovascular complications.

Individual Authors' Contributions: UA contributed to the study conception, study design, data collection, data analysis and interpretation, and manuscript preparation. **SM** was involved in the study conception and data collection. **SF** contributed to the study conception, study design, data collection, and manuscript preparation. **FA** participated in the study design and data analysis and interpretation. All authors reviewed and approved the final version of the manuscript.

Availability of Data: The data supporting this study's findings are available upon reasonable request to corresponding author.

Declaration of Non-use of Generative AI: The authors affirm that no generative artificial intelligence tools were utilized in the design, analysis, interpretation of data, or preparation of this manuscript. All content is the result of the authors' original work.

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