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ORIGINAL ARTICLE

Clinical and Metabolic Determinants of Hepatic Fibrosis in Patients with Metabolic Dysfunction- Associated Steatotic Liver Disease (MASLD) – Insights from a Bangladeshi Cohort

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Kamrul Anam[1], Tahamina Akter[2], Harun or Rashid[3], Sabbir Hossain[4], Tanveer Rahman[5]

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Correspondence to
Kamrul Anam

ORCID
https://orcid.org/0000-0002-2196-5392

ABSTRACT

Introduction: *Metabolic dysfunction–associated steatotic liver disease (MASLD) is now the most common chronic liver disease globally, driven largely by obesity, insulin resistance, and diabetes. Hepatic fibrosis is the strongest predictor of long-term outcomes, yet data on its clinical and metabolic determinants in Bangladeshi patients are scarce. Identifying simple, accessible predictors of significant fibrosis is essential for early risk stratification in resource-limited settings.* ***Methods & Materials:*** *This cross-sectional study was conducted in the Department of Hepatology, BSMMU, from June 2024 to June 2025, including 40 adults with biopsy-proven MASLD. Clinical parameters, metabolic variables (diabetes, impaired glucose tolerance, HOMA-IR), biochemical markers (AST/ALT ratio), and histological features (NAS components, fibrosis stage) were assessed. Patients were categorized into mild/no fibrosis (F0–F1) and significant fibrosis (F2–F4).* ***Results:*** *Of 40 patients, 18 (45%) had significant fibrosis. Those with fibrosis had higher BMI (28.2 ± 3.2 vs. 25.7 ± 3.8 kg/m^2, p=0.032), with BMI ≥28 kg/m^2 markedly more common (72.2% vs. 18.2%, p=0.001). Diabetes (50.0% vs. 9.1%, p=0.005) and IGT (27.8% vs. 0%, p=0.013) were significantly associated with fibrosis, as was higher HOMA-IR (2.7 ± 0.7 vs. 1.9 ± 0.4, p=0.001). AST/ALT ratio ≥0.8 strongly predicted fibrosis (61.1% vs. 13.6%, p=0.001). Histologically, lobular inflammation ≥2 (p=0.013) and MASH (72.2% vs. 31.8%, p=0.011) were more frequent in the fibrosis group. Multivariate analysis identified BMI ≥28 kg/m^2, diabetes, AST/ALT ratio ≥0.8, and MASH as independent predictors.* ***Conclusion:*** *Obesity, diabetes, elevated AST/ALT ratio, and histologic MASH are key determinants of significant fibrosis in Bangladeshi patients with MASLD.*

Keywords: *MASLD, Fibrosis, Diabetes, BMI*

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1. *Assistant Professor, National Gastroliver Institute and Hospital, Mohakhali, Dhaka, Bangladesh*
2. *Research Assistant, NIPSOM, Dhaka, Bangladesh*
3. *Assistant Professor, Department of Hepatology, Shaheed Tajuddin Ahmad Medical College, Gazipur, Bangladesh*
4. *Registrar, Department of Hepatology, Dhaka Medical College Hospital, Dhaka, Bangladesh*
5. *Hepatologist, Rajshahi Medical College Hospital, Rajshahi, Bangladesh*

INTRODUCTION

Metabolic dysfunction–associated steatotic liver disease (MASLD), previously known as non-alcoholic fatty liver disease (NAFLD), has emerged as the most common chronic liver disease worldwide, affecting nearly one-quarter of the global adult population [1]. The renaming of NAFLD to MASLD reflects a shift toward metabolic risk–based classification and acknowledges the central role of insulin resistance, obesity, and dysglycaemia in disease development [2]. MASLD encompasses a spectrum ranging from simple steatosis to steatohepatitis and progressive hepatic fibrosis, the latter being the strongest predictor of liver-related and overall mortality [3]. Fibrosis progression in MASLD is not uniform; only a subset of patients transitions from steatosis to steatohepatitis and advanced fibrosis. Recent evidence suggests that metabolic risk factors including obesity, type 2 diabetes mellitus (T2DM), impaired glucose tolerance (IGT), hypertension, and dyslipidaemia are key determinants of fibrogenesis [4]. Among these, diabetes and central obesity are particularly potent drivers of disease progression, conferring faster fibrosis advancement compared with non-diabetic or lean counterparts [5]. South Asian populations, including Bangladesh, carry a disproportionately high burden of metabolic syndrome and insulin resistance despite lower average BMI, making MASLD a rising public health concern in the region [6]. Histological assessment via liver biopsy remains the reference standard for determining fibrosis stage and diagnosing steatohepatitis. The NAFLD Activity Score (NAS), incorporating steatosis, ballooning, and lobular inflammation, is widely used in research and clinical evaluation [7]. Among these components, inflammation particularly lobular inflammation—has been identified as a major driver of fibrosis progression, whereas steatosis alone does not reliably predict disease severity [8]. Early identification of patients at risk for significant fibrosis (≥F2) is crucial, as this group faces higher risks of cirrhosis, hepatocellular carcinoma, and liver-related death [9]. In low- and middle-income countries, challenges such as limited access to elastography, liver biopsy, and specialized laboratory testing necessitate the use of simpler clinical and biochemical markers for risk stratification. Parameters such as

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body mass index (BMI), waist circumference, diabetes status, and the AST/ALT ratio remain widely accessible and cost-effective tools for identifying high-risk individuals [10]. Despite increasing recognition of MASLD in Bangladesh, limited biopsy-based studies have evaluated the metabolic and clinical determinants of fibrosis within this population. Understanding the specific factors that contribute to fibrosis in Bangladeshi MASLD patients is essential for targeted screening, timely intervention, and reducing long-term complications. This study aimed to evaluate the clinical, metabolic, biochemical, and histological determinants of hepatic fibrosis in MASLD patients in a Bangladeshi cohort. By identifying the key predictors of significant fibrosis, our findings may help optimize early detection strategies and inform national clinical guidelines for MASLD management.

METHODS & MATERIALS

This cross-sectional study was conducted in the Department of Hepatology, Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka, over a one-year period from June 2024 to June 2015. A total of 40 adult patients with biopsy-confirmed Metabolic Dysfunction–Associated Steatotic Liver Disease (MASLD) were consecutively enrolled. Clinical data (age, sex, anthropometry), metabolic variables (diabetes status, impaired glucose tolerance, lipid profile, HOMA-IR), and biochemical parameters (AST, ALT, AST/ALT ratio, GGT, platelets) were collected using structured data sheets. All patients underwent percutaneous liver biopsy, and histological assessment was performed using the NAFLD Activity Score (NAS) system to evaluate steatosis, lobular inflammation, ballooning, and fibrosis stage (F0–F4). Patients were categorized into two groups: mild/no fibrosis (F0–F1) and significant fibrosis (F2–F4). Statistical analyses included comparison of clinical, metabolic, biochemical, and histological variables between groups, with chi-square tests or Student's t-tests used where appropriate. Variables significant on univariate analysis were entered into multivariate logistic regression to identify independent determinants of fibrosis. Ethical approval was obtained in accordance with BSMMU institutional guidelines.

Inclusion Criteria

- Adult patients aged ≥18 years
- Biopsy-confirmed MASLD/NAFLD
- Adequate liver biopsy specimen for full NAS scoring

Exclusion Criteria

- Significant alcohol intake (above standard diagnostic thresholds)
- Chronic viral hepatitis (HBV, HCV)
- Other known chronic liver diseases (e.g., autoimmune hepatitis, Wilson's disease, hemochromatosis)
- Drug-induced liver injury

RESULTS

A total of 40 biopsy-confirmed MASLD patients were included in the study. Among them, 18 (45%) had significant fibrosis (F2–F4) and 22 (55%) had minimal or no fibrosis (F0–F1). Patients with significant fibrosis had a noticeably higher BMI compared to those without fibrosis, and a much larger proportion had BMI ≥28 kg/m^2. Other clinical variables such as age, hypertension, and waist circumference showed no meaningful differences between the two groups. [Table I]

Table – I: Baseline Demographic and Clinical Characteristics of Patients with and Without Significant Fibrosis (*n* = 40)

Variable	Fibrosis 2–4 (n=18)	Fibrosis 0–1 (n=22)	p-value
Age (years)	11 (61.1%) ≥45 years	11 (50.0%) ≥45 years	0.482
Sex (Female)	13 (72.2%)	11 (50.0%)	NS
BMI (kg/m^2), mean ± SD	28.2 ± 3.2	25.7 ± 3.8	0.032
BMI ≥28 kg/m^2	13 (72.2%)	4 (18.2%)	0.032
Obesity ≥25 kg/m^2	15 (83.3%)	11 (50.0%)	0.027
Waist circumference above metabolic cutoff	17 (94.4%)	17 (77.3%)	NS
Hypertension	5 (27.8%)	7 (31.8%)	0.781

Metabolic abnormalities were more prominent in patients with advanced fibrosis. Diabetes mellitus and impaired glucose tolerance were significantly more common in the fibrosis group. The mean HOMA-IR level was also higher in F2–F4 patients, indicating greater insulin resistance. All patients with significant fibrosis met the criteria for metabolic syndrome, compared to just over half of those with minimal fibrosis. These findings highlight the strong association between metabolic dysfunction and fibrosis severity. [Table II]

Table – II: Metabolic Characteristics of MASLD Patients by Fibrosis Group

Metabolic Variable	Fibrosis 2–4 (n=18)	Fibrosis 0–1 (n=22)	p-value
Diabetes Mellitus	9 (50.0%)	2 (9.1%)	0.005
Impaired Glucose Tolerance (IGT)	5 (27.8%)	0 (0%)	0.013
HOMA-IR >1.8	12 (66.7%)	10 (45.5%)	0.155
HOMA-IR (mean ± SD)	2.7 ± 0.7	1.9 ± 0.4	0.001
Metabolic Syndrome	18 (100%)	12 (54.5%)	<0.001
Triglycerides >150 mg/dL	12 (66.7%)	7 (35.0%)	NS
Total Cholesterol >200 mg/dL	11.1%	32.5%	NS

In terms of liver biochemistry, ALT and GGT levels did not differ significantly between groups. However, the AST/ALT ratio showed a clear trend: an AST/ALT ≥0.8 was far more frequent among those with significant fibrosis. This suggests that the AST/ALT ratio, rather than absolute enzyme levels, serves as a useful biochemical marker for identifying higher fibrosis stages in MASLD. [Table III]

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Table – III: Liver Biochemistry and Enzyme Profiles by Fibrosis Stage

Parameter	Fibrosis 2–4	Fibrosis 0–1	p-value
AST (U/L), mean ± SD	31.6 ± 20.5	—	NS
ALT (U/L), mean ± SD	47.2 ± 26.4	40.6 ± 22.9	NS
AST/ALT ratio ≥0.8	11 (61.1%)	3 (13.6%)	0.001
GGT elevated (sex-specific)	7 (38.9%)	3 (13.6%)	0.142
Platelets	Similar between groups	—	NS

Histological assessment revealed that steatosis grade and hepatocellular ballooning were not significantly different between the two groups. In contrast, lobular inflammation was significantly higher in patients with fibrosis, and MASH (defined by NAS ≥5) was also more common among those with F2–F4. These results underscore that hepatic inflammation, rather than fat accumulation alone, plays a central role in fibrosis progression. [Table IV]

Table – IV: Histological Features by Fibrosis Category (NAS Components)

Histologic Feature	Fibrosis 2–4 (n=18)	Fibrosis 0–1 (n=22)	p-value
Steatosis Grade 1	3 (16.7%)	7 (31.8%)	0.101
Steatosis Grade 2	12 (66.6%)	15 (68.2%)	0.101
Steatosis Grade 3	3 (16.7%)	0 (0%)	0.101
Lobular Inflammation (Score 2)	9 (50.0%)	3 (13.6%)	0.013
Ballooning (Score 2)	10 (55.6%)	9 (40.9%)	0.480
NAS ≥5 (MASH)	13 (72.2%)	7 (31.8%)	0.011

On univariate analysis, several factors—including high BMI, obesity, diabetes, IGT, elevated AST/ALT ratio, lobular inflammation, and MASH—were significantly associated with fibrosis. Factors such as age, sex, hypertension, and GGT did not show significant associations. This indicates that both metabolic risk factors and hepatic inflammatory changes are key predictors of fibrosis. [Table V]

Table – V: Clinical & Metabolic Predictors of Significant Fibrosis (Univariate Analysis)

Predictor	OR (95% CI)	p-value
Age ≥45 years	1.57 (0.37–6.76)	0.482
Fem ale Sex	—	NS
BMI ≥28 kg/m^2	—	0.032
Obesity ≥25 kg/m^2	—	0.027
Diabetes Mellitus	10.0 (1.49–84.89)	0.005
IGT	—	0.013
HOMA-IR >1.8	2.4 (0.55–10.77)	0.155
AST/ALT ≥0.8	9.95 (1.76–64.5)	0.001
GGT Elevated	—	0.142
MASH (NAS ≥5)	5.57 (1.19–28.23)	0.011
Lobular inflammation	—	0.013

In the multivariate model, BMI ≥28 kg/m^2, diabetes mellitus, AST/ALT ratio ≥0.8, and MASH remained independent predictors of significant fibrosis. Insulin resistance (HOMA-IR) lost significance after adjustment. Overall, these findings demonstrate that metabolic burden and hepatic inflammation are the strongest independent determinants of fibrosis severity in this Bangladeshi MASLD cohort. [Table VI]

Table – VI: Multivariate Logistic Regression: Independent Determinants of Significant Fibrosis

Variable	Adjusted OR (95% CI)	p-value
BMI ≥28 kg/m^2	17.53 (1.5–100.0)	<0.05
Diabetes Mellitus	10.0 (1.49–84.89)	<0.05
AST/ALT ≥0.8	9.95 (1.76–64.5)	<0.05
HOMA-IR >1.8	2.4 (0.55–10.77)	NS
MASH (NAS ≥5)	5.57 (1.19–28.23)	<0.05

DISCUSSION

In our study, we found this: 45% (18/40) of MASLD patients had significant fibrosis (F2–F4). This proportion is high but not unprecedented; the global burden of steatotic liver disease has been rising rapidly in the context of obesity and metabolic syndrome. Recent global estimates suggest that the prevalence of MASLD (formerly NAFLD) is increasing worldwide in parallel with rising obesity and type 2 diabetes rates. A large meta-analysis demonstrated considerable variability in fibrosis prevalence across populations, depending on metabolic risk factor distributions [11,12]. In our study we found this: Higher BMI mean 28.2 vs 25.7 kg/m^2 — and BMI ≥28 kg/m^2 strongly predicted fibrosis in multivariate analysis. This supports the widely accepted concept that obesity is a primary driver of MASLD progression. Indeed, the metabolic stress associated with adiposity (insulin resistance, lipotoxicity, systemic inflammation) underlies much of fibrogenesis in fatty liver disease [13]. Several previous biopsy-based and epidemiologic studies have similarly identified obesity as a major risk factor for advanced fibrosis in NAFLD/MASLD [14]. In our study, we found that diabetes mellitus and impaired glucose tolerance (IGT) were significantly more common in

patients with advanced fibrosis. Specifically, 50% of fibrosis patients had diabetes versus only 9.1% in the non-fibrosis group, and IGT was present exclusively among those with fibrosis. This reinforces the notion that dysglycemia is tightly linked to fibrogenesis. Epidemiologic data support this: type 2 diabetes significantly increases the risk of NAFLD and its progression to fibrosis or MASH [15]. The coexistence of metabolic syndrome and diabetes substantially elevates risk, as shown in large global burden studies [16]. In our study we found this: Insulin resistance, assessed by HOMA-IR, was significantly higher in fibrosis patients (mean 2.7 vs 1.9). This suggests that insulin resistance is a relevant metabolic driver of fibrosis. This mirrors recent analyses showing robust associations between insulin-resistance indices and liver fibrosis in NAFLD populations [17]. Experimental and clinical evidence indicates that insulin resistance promotes hepatic steatosis, oxidative stress, inflammatory signaling and activation of hepatic stellate cells — key events in fibrogenesis [18]. However, in our multivariate model HOMA-IR lost statistical significance, likely because its effects are mediated via obesity, diabetes, and other metabolic factors; this suggests that in clinical settings, simpler markers like BMI or diabetes status may suffice to identify high-risk individuals. In our study we found this: AST/ALT ratio ≥0.8 was strongly associated with fibrosis; whereas absolute ALT and GGT levels were not significantly different between fibrosis groups. This is important since transaminase ratio is a simple, widely available biochemical parameter. Our finding aligns with the pathophysiologic understanding that with progressive fibrosis, AST tends to rise relative to ALT. Reviews on NAFLD/MASLD emphasize that fibrosis — rather than steatosis — is the main determinant of long-term outcomes, and that non-invasive, inexpensive markers are needed especially in resource-limited settings [19]. Thus, AST/ALT ratio remains a useful initial screening parameter for fibrosis risk in MASLD, particularly where advanced diagnostics are unavailable. Clinical guidelines also recommend noninvasive fibrosis assessment in patients with metabolic risk factors [20]. In our study we found this: Histologic inflammation — specifically lobular inflammation and MASH (NAS ≥5) — was significantly more frequent in patients with significant fibrosis, while steatosis grade and ballooning score alone were not different between groups. This underscores the key role of hepatic inflammatory activity, rather than fat accumulation per se, in fibrogenesis. This observation is consistent with established pathophysiology: in metabolic-associated liver disease, chronic metabolic stress, lipotoxicity and insulin resistance trigger inflammatory cascades, activating hepatic stellate cells and collagen deposition [18]. Previous biopsy-based studies likewise found that fibrosis correlates more strongly with inflammatory changes (MASH) than with simple steatosis [14]. On univariate analysis, we found that obesity, diabetes, insulin resistance, metabolic syndrome, elevated AST/ALT ratio, lobular inflammation, and MASH were all associated with fibrosis. But after multivariate adjustment, only BMI ≥28 kg/m^2, diabetes, AST/ALT ≥0.8, and MASH remained independent predictors. This suggests that in clinical practice, a combination of simple clinical (BMI), metabolic (diabetes), biochemical (AST/ALT ratio), and histologic (presence of MASH) parameters can identify high-risk patients. This is in line with global guidance: because MASLD is strongly linked to metabolic comorbidities, patients with obesity, diabetes or metabolic syndrome should be prioritized for fibrosis screening [21]. The strengths of our study include its biopsy-proven MASLD diagnosis, comprehensive metabolic and histological evaluation, and analysis of multiple potential determinants of fibrosis. However, some limitations must be acknowledged. The sample size was modest (n=40), limiting statistical power and potentially causing type II error for some associations. The cross-sectional design prohibits inference on causality or longitudinal progression of fibrosis. Also, because the cohort is from a single center in Bangladesh, generalizability to broader South Asian or global populations may be limited. Finally, although biopsy remains the gold standard, sampling variability and observer differences can affect histologic assessment. Despite these limitations, our findings carry important implications. Given the rising burden of MASLD globally, especially in regions like South Asia with burgeoning diabetes and obesity rates, there is an urgent need for systematic screening of high-risk individuals [22]. In resource-constrained settings, reliance on simple parameters (BMI, diabetes status, AST/ALT ratio) along with noninvasive tests may help detect fibrosis early, preventing progression to cirrhosis or hepatocellular carcinoma. Additionally, our study reinforces that effective management of metabolic risk factors weight reduction, glycemic control, insulin-sensitizing strategies and mitigation of hepatic inflammation should be central in MASLD care.

Limitations of the Study:

The sample size was relatively small and drawn from a single tertiary care center, which may limit the generalizability of the findings to the broader Bangladeshi population.

CONCLUSION

In this biopsy-based Bangladeshi MASLD cohort, obesity, diabetes mellitus, elevated AST/ALT ratio, and histologic MASH were identified as the strongest determinants of significant hepatic fibrosis, while steatosis alone showed no meaningful association. These findings highlight that metabolic dysfunction and hepatic inflammation—rather than fat accumulation drive fibrogenesis in MASLD. Simple clinical and laboratory markers such as BMI, diabetic status, and AST/ALT ratio can therefore serve as practical screening tools to identify high-risk patients in resource-limited settings. Early recognition and targeted management of metabolic risk factors are essential to prevent progression to cirrhosis and its complications.

RECOMMENDATION

Based on these findings, routine assessment of BMI, diabetes status, and AST/ALT ratio should be incorporated into the initial evaluation of all MASLD patients in Bangladesh to identify those at higher risk for fibrosis. Individuals with metabolic syndrome, diabetes, or elevated transaminase ratios should undergo further fibrosis assessment using non-invasive methods or biopsy where appropriate. Lifestyle modification, weight reduction, and strict glycemic control should be emphasized as primary therapeutic strategies. Larger, multi-center studies with prospective follow-up are recommended to validate these predictors and support the development of national MASLD screening guidelines.

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