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Prevalence of Non-Alcoholic Fatty Liver Disease Among Patients with Rheumatological Disorders at a Tertiary Care Centre in Western Maharashtra: A Cross-Sectional Observational Study

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Abstract

Background: Non-alcoholic fatty liver disease (NAFLD) is an increasingly recognised comorbidity in patients with rheumatological disorders. Chronic systemic inflammation, metabolic derangements, and prolonged immunosuppressive therapy collectively predispose this population to hepatic steatosis and fibrosis. Population-level data from Indian tertiary care centres remain limited. ***Methods:*** A cross-sectional observational study was conducted at a tertiary care centre in Western Maharashtra over 18 months. One hundred adult patients fulfilling established classification criteria for rheumatological disorders were enrolled by simple random sampling. NAFLD was diagnosed using abdominal ultrasonography, transient elastography (FibroScan) with controlled attenuation parameter (CAP) and liver stiffness measurement (LSM), and non-invasive biochemical indices—Fibrosis-4 (FIB-4) score and NAFLD Fibrosis Score (NFS). Patients with significant alcohol intake or known chronic liver disease were excluded. Statistical analyses included descriptive statistics, Pearson's correlation, one-way ANOVA, and chi-square tests. ***Results:*** NAFLD was identified in 20% of participants (n=20/100). Rheumatoid arthritis was the most common underlying diagnosis (41%), followed by spondyloarthritis (31%) and systemic lupus erythematosus (SLE; 17%). The majority of patients (42%) were aged 31–40 years. Most NAFLD cases had CAP scores in the moderate range (200–300 dB/m). A statistically significant moderate positive correlation was observed between FIB-4 and NFS ($r=0.461$, $p<0.001$). Age, BMI, and waist-hip ratio showed no statistically significant association with fibrosis severity. Key modifiable risk factors included obesity, type 2 diabetes mellitus, dyslipidaemia, and long-term immunosuppressive therapy. ***Conclusion:*** NAFLD affects one in five rheumatological patients at a tertiary care centre in Western Maharashtra. Non-invasive fibrosis indices demonstrated concordant performance. Systematic liver screening should be integrated into routine rheumatological care, particularly in patients with coexisting metabolic risk factors.

Keywords: Non-alcoholic fatty liver disease; rheumatological disorders; rheumatoid arthritis; transient elastography; FIB-4 index; NAFLD Fibrosis Score; prevalence; India

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INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) encompasses a spectrum of hepatic pathology ranging from simple steatosis to non-alcoholic steatohepatitis (NASH), progressive fibrosis, and ultimately cirrhosis and hepatocellular carcinoma. It is the most common cause of chronic liver disease worldwide, with an estimated global prevalence of approximately 25% in the general population, and is increasingly recognised as a systemic metabolic disorder with significant cardiovascular and extrahepatic implications.[1]. Patients with chronic inflammatory rheumatological disorders—including rheumatoid arthritis (RA), systemic lupus erythematosus (SLE), spondyloarthritis, and related connective tissue diseases—constitute a particularly vulnerable population for NAFLD. Persistent activation of pro-inflammatory cytokines such as tumour necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6), immune dysregulation, and the metabolic consequences of long-term corticosteroid and disease-modifying antirheumatic drug (DMARD) use create a pro-steatogenic milieu.[4] Methotrexate, the cornerstone DMARD for RA and other inflammatory arthropathies, carries a well-established risk of hepatotoxicity ranging from transaminase elevation to hepatic fibrosis with cumulative dosing.[8]

Epidemiological data confirm a significantly elevated NAFLD burden in rheumatological populations. A 2023 systematic review and meta-analysis by Zamani et al. reported a pooled NAFLD prevalence of 35.3% (95% CI 19.9–50.6%) among nine cohort studies comprising 2,178 patients with RA, with each unit increase in BMI associated with a 24% increased NAFLD risk.[2] An updated meta-analysis by Mahapatro et al. in 2025 reported a pooled NAFLD/NASH prevalence of 22.8% in RA patients, with diagnostic method-specific rates of 33.3% by FibroScan, 43.9% by liver biopsy, and 30.5% by

ultrasonography.[3] A population-based cohort study from Taiwan demonstrated that newly diagnosed RA patients had a 1.66-fold higher incidence of NAFLD during the first four years after diagnosis compared to matched controls.[5] Furthermore, Ausserwinkler et al., using the Paracelsus 10,000 population-based cohort, demonstrated that RA was independently associated with a 35% increased risk of metabolic dysfunction-associated steatotic liver disease (MASLD) after adjustment for cardiometabolic confounders.[6] Erre et al. reported a prevalence of moderate-to-severe hepatic steatosis of 38.7% in RA patients versus 19.7% in healthy controls on ultrasonographic assessment.[7] Beyond RA, Baeza-Zapata et al. found a prevalence of steatotic liver disease of 21.7% in SLE patients using FibroScan, with BMI and hydroxychloroquine use identified as principal modifying factors.[9]

NAFLD carries significant prognostic implications beyond liver-specific outcomes. Targher et al. established NAFLD as an independent risk factor for cardiovascular disease, with a two- to threefold increased risk of major cardiovascular events.[10] This cardiovascular burden is particularly clinically relevant in rheumatological disorders, where accelerated atherosclerosis driven by chronic systemic inflammation is already a major determinant of long-term morbidity and mortality. In a Chinese RA cohort, Zou et al. demonstrated that comorbid MAFLD was independently associated with a 2.19-fold increased risk of cardiovascular events and a 2.48-fold increased risk of high estimated 10-year cardiovascular risk.[11]

Non-invasive diagnostic approaches have substantially advanced the clinical assessment of NAFLD. Transient elastography with CAP (FibroScan) enables simultaneous quantification of hepatic steatosis and fibrosis without the need for liver biopsy.[12] Biochemical fibrosis indices—FIB-4 and NFS—offer cost-effective, accessible risk stratification, particularly relevant in resource-limited settings. Despite this growing evidence base, robust data on NAFLD prevalence in rheumatological patients from the Indian subcontinent are sparse, particularly from tertiary care centres in Western Maharashtra. The present study was conducted to estimate the prevalence of NAFLD among patients with rheumatological disorders at a tertiary care centre in Western Maharashtra, and to identify associated modifiable risk factors and evaluate the performance of non-invasive diagnostic tools in this population.

MATERIALS AND METHODS

Study design and setting

This was a cross-sectional observational study conducted at the rheumatology outpatient department and inpatient wards of a tertiary care centre in Western Maharashtra, India. Data collection was carried out over 18 months following institutional ethics committee approval.

Participants

One hundred adult patients (age ≥18 years) fulfilling established classification criteria for rheumatological disorders—including RA (2010 ACR/EULAR criteria), SLE (SLICC 2012 criteria), Sjögren's syndrome, spondyloarthritis (ASAS criteria), psoriatic arthritis, Behçet's disease, inflammatory myopathies, vasculitis syndromes, and systemic sclerosis—were enrolled using simple random sampling from eligible attendees at the rheumatology clinic or inpatient ward.

Patients were excluded if they had: (a) alcohol intake exceeding 33–45 g/day in men or 22–30 g/day in women; or (b) a prior documented diagnosis of chronic liver disease from any aetiology (viral hepatitis, autoimmune hepatitis, primary biliary cholangitis, or drug-induced liver injury).

Sample size

The sample size was calculated using the standard prevalence formula: $n = Z^2p(1-p)/d^2$, where $Z = 1.96$ (95% confidence interval), $p = 0.50$ (assumed prevalence), and $d = 0.10$ (margin of error). This yielded a minimum of 96 participants, rounded to 100 for feasibility and statistical reliability.

Diagnostic assessment

Participants underwent a structured multimodal evaluation comprising: (i) clinical and anthropometric assessment—height, weight, BMI, waist circumference, and waist-hip ratio; (ii) biochemical investigations—complete blood count, liver function tests (ALT, AST, alkaline phosphatase, GGT, bilirubin, albumin), renal function tests, fasting blood glucose, lipid profile, HBsAg, and anti-HCV; (iii) abdominal ultrasonography for hepatic steatosis grading; and (iv) transient elastography (FibroScan) for simultaneous LSM and CAP measurement.[12]

LSM thresholds were: <7.5 kPa (no significant fibrosis), 7.5–9.9 kPa (F2, moderate fibrosis), 10.0–13.9 kPa (F3, severe fibrosis), and ≥14.0 kPa (F4, consistent with cirrhosis). CAP thresholds were: <238 dB/m (S0, minimal/no steatosis), 238–260 dB/m (S1, mild steatosis), 260–290 dB/m (S2, moderate steatosis), and >290 dB/m (S3, severe steatosis).
Non-invasive fibrosis indices were calculated as follows. FIB-4 = [Age (years) × AST (U/L)] / [Platelets (10^9/L) × √ALT (U/L)]: score ≤1.30 ruled out advanced fibrosis; ≥2.67 suggested advanced fibrosis; 1.30–2.67 was indeterminate. NFS =

−1.675 + 0.037 × age + 0.094 × BMI + 1.13 × IFG/diabetes (yes=1, no=0) + 0.99 × AST/ALT − 0.013 × platelets − 0.66 × albumin: score < −1.455 excluded advanced fibrosis; >0.675 indicated advanced fibrosis.[13,14]

Statistical analysis

Data were entered into Microsoft Excel and analysed using SPSS version 23.0. Categorical variables are expressed as frequencies and percentages. Continuous variables are expressed as mean ± standard deviation. Pearson's correlation was used to assess relationships between FIB-4 and NFS and between anthropometric variables and fibrosis scores. One-way ANOVA was used to evaluate the association between age and CAP score categories. The chi-square test was used to examine the association between comorbidity status and CAP score category. A p-value of <0.05 was considered statistically significant.

Ethical considerations

The study was approved by the institutional ethics committee and conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants prior to enrolment.

RESULTS

Demographic and clinical characteristics

The study enrolled 100 patients. The majority (42%) were in the 31–40 year age group, followed by the 21–30 year group (17%) and the 51–60 year group (15%). The gender distribution was near-equal (males 51%, females 49%). By rheumatological diagnosis, RA was the most frequent condition (41%), followed by spondyloarthritis—encompassing axial and peripheral subtypes—(31%), SLE (17%), dermatomyositis (3%), gouty arthritis (2%), and other diagnoses including psoriatic arthritis, AAV, AICTD, sarcoidosis, and systemic sclerosis (1% each). Demographic and clinical data are summarised in Table 1.

Table 1. Demographic and clinical characteristics of the study population (n=100)

Characteristic	Category	n (%)
Age (years)	≤20	3 (3%)
	21–30	17 (17%)
	31–40	42 (42%)
	41–50	13 (13%)
	51–60	15 (15%)
	≥61	10 (10%)
Gender	Male	51 (51%)
	Female	49 (49%)
Rheumatological diagnosis	Rheumatoid arthritis	41 (41%)
	Spondyloarthritis (axial+peripheral)	31 (31%)
	Systemic lupus erythematosus	17 (17%)
	Dermatomyositis	3 (3%)
	Gouty arthritis	2 (2%)
	Other (psoriatic arthritis, AAV, AICTD, sarcoidosis, systemic sclerosis)	6 (6%)

Prevalence of NAFLD

NAFLD was identified in 20 of 100 patients (20%). The remaining 80 patients (80%) showed no evidence of fatty liver on combined multimodal assessment. Among cases of steatosis, the controlled attenuation parameter was predominantly in the moderate range (200–300 dB/m); severe steatosis (CAP >300 dB/m) was detected in only two participants. These findings are summarised in Table 2.

Table 2. NAFLD diagnosis and CAP score distribution (n=100)

Variable	Category	n (%)
NAFLD	Present	20 (20%)
	Absent	80 (80%)
CAP score category	<200 dB/m (minimal/no steatosis, S0)	4 (4%)
	200–300 dB/m (moderate steatosis, S1–S2)	93 (93%)†
	>300 dB/m (severe steatosis, S3)	2 (2%)
	Uninterpretable/missing	1 (1%)

Small cell count in severe category; interpret with caution. CAP, controlled attenuation parameter.

Non-invasive fibrosis index performance

Correlation analysis revealed a statistically significant moderate positive association between FIB-4 and NFS (r = 0.461, p < 0.001), indicating that the two indices track fibrosis severity in a concordant manner. This supports their combined use as complementary non-invasive screening tools (Table 3).

Table 3. Correlation between FIB-4 score and NAFLD Fibrosis Score (n=100)

	FIB-4 Score	NAFLD Fibrosis Score
FIB-4 Score	1.000	0.461**
NAFLD Fibrosis Score	0.461**	1.000

**** p < 0.001 (Pearson's correlation). FIB-4, Fibrosis-4 index; NFS, NAFLD Fibrosis Score.**

Anthropometric and demographic associations with fibrosis severity

Age showed no statistically significant association with CAP score severity on one-way ANOVA (F = 0.116, p = 0.891). BMI showed a very weak, non-significant inverse correlation with NFS (r = −0.035, p = 0.733). Similarly, waist-hip ratio was not significantly associated with NFS (r = −0.035, p = 0.730). Comorbidity status was not significantly associated with CAP score category (chi-square = 0.570, df = 2, p = 0.752). These results are summarised in Table 4.

Table 4. Association of anthropometric and demographic variables with liver fibrosis severity

Variable	Test	Statistic	p-value	Interpretation
Age vs CAP category	One-way ANOVA	F = 0.116	0.891	Not significant
BMI vs NFS	Pearson's r	r = −0.035	0.733	Not significant
Waist-hip ratio vs NFS	Pearson's r	r = −0.035	0.730	Not significant
Comorbidities vs CAP category	Chi-square	$\chi^2 = 0.570$	0.752	Not significant

BMI, body mass index; CAP, controlled attenuation parameter; NFS, NAFLD Fibrosis Score.

Modifiable risk factors

Among patients identified with NAFLD, the principal modifiable risk factors were obesity, type 2 diabetes mellitus, dyslipidaemia, and long-term corticosteroid or DMARD (particularly methotrexate) use. Female patients showed a higher susceptibility to NAFLD in this cohort, consistent with the predominant female representation in autoimmune rheumatological diseases such as RA and SLE.

DISCUSSION

The present study found a NAFLD prevalence of 20% among patients with rheumatological disorders at a tertiary care centre in Western Maharashtra. This finding is clinically meaningful and contextualises data from comparable published cohorts. The pooled NAFLD prevalence of 35.3% reported by Zamani et al. across nine international RA studies[2] and the 41.2% reported by Ausserwinkler et al. in a population-based Austrian cohort[6] are higher than the present estimate, likely reflecting differences in diagnostic criteria (ultrasound vs. FibroScan vs. PDFF-MRI), population heterogeneity, BMI distributions, and the inclusion of multiple rheumatological diagnoses rather than RA alone in the current cohort. The updated meta-analysis by Mahapatro et al. reported a pooled NAFLD/NASH prevalence of 22.8% in RA patients,[3] closer to our finding, while Erre et al. reported 38.7% in an Italian RA cohort on ultrasonographic assessment.[7] Zou et al. reported a MAFLD prevalence of 21.4% in a Chinese RA cohort,[11] which is the closest published comparator to our study. The comparatively lower prevalence in the present cohort may also reflect the higher proportion of lean NAFLD in Indian patients, in whom hepatic steatosis occurs at lower BMI thresholds than in Western counterparts.

The non-significant associations of age, BMI, and waist-hip ratio with fibrosis severity observed in the present study are noteworthy and contrast with patterns in the general NAFLD population. This finding suggests that in patients with rheumatological disorders, hepatic fibrosis is driven more by disease-specific mechanisms—including chronic systemic inflammation, immune dysregulation, and iatrogenic metabolic perturbations from corticosteroids and DMARDs—than by conventional anthropometric risk factors. Barbarroja et al. similarly identified chronic inflammation, autoimmunity, and treatment effects as important pathogenic contributors to liver disease in inflammatory arthritis, independent of classical metabolic risk factors.[4] Methotrexate-associated hepatotoxicity, including NASH-like histology and progressive fibrosis, is a well-documented concern with cumulative dosing,[8] and monitoring strategies using non-invasive indices are essential for long-term management.[15]

The moderate positive correlation between FIB-4 and NFS (r=0.461, p<0.001) validates the concordant performance of these indices in a rheumatological cohort. Miyata et al. demonstrated that FIB-4 tracked histological fibrosis in RA patients on methotrexate, with declining FIB-4 values correlating with histological improvement after drug modification.[15] Olsson-White et al. found that transient elastography had 100% sensitivity and 84% specificity for fibrosis in RA patients on methotrexate, outperforming APRI and FIB-4 in identifying significant hepatic fibrosis.[13] Cox et al. demonstrated that NFS and FIB-4 had high negative predictive values of 94.1% and 91.6%, respectively, for ruling out advanced fibrosis compared with FibroScan—supporting their use as first-line triage tools.[14] Cervoni et al. confirmed that a NFS-based algorithm effectively identified fibrosis ≥F2 in patients with psoriasis, RA, and Crohn's disease on methotrexate, with age, male sex, and metabolic syndrome as independent fibrosis predictors.[16]

The predominance of moderate CAP scores (200–300 dB/m) indicates that most steatosis in this cohort was detected at an early-to-intermediate stage, representing an optimal window for intervention. The cardiovascular implications of NAFLD are particularly important in rheumatological disorders, where systemic inflammation is already an independent cardiovascular risk driver. Targher et al. established a two- to threefold increased risk of major cardiovascular events in NAFLD patients,[10] and Zou et al. demonstrated that comorbid MAFLD was independently associated with significantly higher 10-year cardiovascular risk in RA patients.[11] The findings of the present study are consistent with these observations and reinforce the need for multidisciplinary cardiovascular and hepatic risk assessment in this population.

The findings support a pragmatic clinical algorithm: annual FIB-4 and NFS calculation from routine blood parameters, with FibroScan referral for patients with indeterminate or elevated index scores. Targeted lifestyle counselling for obesity, diabetes, and dyslipidaemia, along with careful monitoring and dose optimisation of hepatotoxic DMARDs, should form integral components of management.

Strengths and limitations

Strengths include the use of a multimodal diagnostic approach combining abdominal ultrasonography, transient elastography, and validated non-invasive fibrosis indices, and the enrolment of a heterogeneous rheumatological population reflecting real-world tertiary care practice. Limitations include the single-centre, cross-sectional design, which precludes causal inference and may limit generalisability. The sample size of 100 restricts statistical power for disease-specific subgroup analyses. Histological confirmation by liver biopsy was not performed. Detailed dietary, physical activity, and genetic data were not captured, precluding full adjustment for confounders. Future multicentre longitudinal studies with larger samples are needed to delineate disease-specific NAFLD trajectories and evaluate outcomes of structured interventions in this population.

CONCLUSION

NAFLD was identified in one in five patients with rheumatological disorders at a tertiary care centre in Western Maharashtra, with most cases detected at an early-to-moderate stage of hepatic steatosis. FIB-4 and NFS demonstrated significant concordance, supporting their combined use as non-invasive fibrosis screening tools in this population. Conventional anthropometric parameters were not independently associated with fibrosis severity, suggesting that disease-specific inflammatory and iatrogenic mechanisms are predominant drivers. Obesity, type 2 diabetes mellitus, dyslipidaemia, and long-term immunosuppressive therapy were the key modifiable risk factors identified. These findings support the integration of routine liver health assessment into standard rheumatological practice, particularly in patients with coexisting metabolic risk factors, and highlight the need for multidisciplinary management of this comorbidity in India.

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