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Liver Stiffness Measurement by Transient Elastography as a Predictor of Disease Progression in NAFLD: Findings from a Two-Year Prospective Cohort

Dr. Kranthi Kumar Pasupulati[1], Dr. Rohit Kumar Bandari[2*]

[1]Assistant Professor in Department of General Medicine in Mallareddy Medical college for women, Hyderabad, Telangana, India- 500055
[2]Assistant Professor in Department of Paediatrics, Mallareddy Medical college for women, Hyderabad, Telangana, India- 500055

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***Corresponding author:** Dr. Rohit Kumar Bandari
Assistant Professor in Department of Paediatrics, Mallareddy Medical college for women, Hyderabad, Telangana, India- 500055

Abstract **Original Research Article**

Background: Non-alcoholic fatty liver disease (NAFLD) is a prevalent chronic liver condition with a spectrum ranging from simple steatosis to non-alcoholic steatohepatitis (NASH), fibrosis, and cirrhosis. Transient elastography (TE) via FibroScan is a non-invasive tool used to quantify liver stiffness measurement (LSM), which correlates with the degree of hepatic fibrosis. ***Objective:*** This prospective cohort study aimed to evaluate the utility of LSM by transient elastography as a predictor of disease progression in patients with NAFLD over a two-year follow-up period at a tertiary care institution in Hyderabad, Telangana, India. ***Methodology:*** Thirty adult patients diagnosed with NAFLD were enrolled from the Department of General Medicine, Mallareddy Medical College for Women, between January 2020 and January 2022. Baseline LSM was performed using FibroScan (Echosens, Paris) and repeated at 12 and 24 months. Clinical, biochemical, and imaging parameters were recorded at each visit. Disease progression was defined as a ≥20% increase in LSM or histopathological progression on repeat biopsy. ***Results:*** Mean baseline LSM was 8.4 ± 4.7 kPa. At 24 months, mean LSM increased to 10.9 ± 5.8 kPa. Disease progression was observed in 11 of 30 patients (36.7%). A baseline LSM >9.5 kPa was significantly associated with progression ($p<0.05$). Patients with advanced fibrosis (F3-F4) at baseline exhibited the greatest increase in LSM over the study period. Elevated ALT, BMI >30 kg/m², and the presence of type 2 diabetes mellitus were identified as significant co-predictors of progression. ***Conclusion:*** Liver stiffness measurement by transient elastography is a reliable, non-invasive predictor of disease progression in NAFLD. Baseline LSM values, particularly above 9.5 kPa, effectively stratify patients at higher risk for progression, supporting its use in routine clinical surveillance of NAFLD patients.

Keywords: Non-alcoholic fatty liver disease, NAFLD, transient elastography, liver stiffness measurement, FibroScan, fibrosis progression, prospective cohort, hepatic steatosis, NASH, non-invasive markers.

1. INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) has emerged as the most common cause of chronic liver disease globally, affecting approximately 25% of the world's adult population [1]. In India, the prevalence of NAFLD has been reported between 9% and 32%, with a rising trajectory paralleling the epidemic of metabolic syndrome, obesity, and type 2 diabetes mellitus [2]. The disease encompasses a histological spectrum from simple hepatic steatosis to non-alcoholic steatohepatitis (NASH), progressive fibrosis, cirrhosis, and ultimately hepatocellular carcinoma (HCC). Given its largely asymptomatic course in early stages, NAFLD represents a significant diagnostic and therapeutic challenge for clinicians.

The natural history of NAFLD is variable and not uniformly progressive; however, a subset of patients with NASH and significant fibrosis are at highest risk for adverse outcomes including liver-related morbidity and mortality [3]. The stage of hepatic fibrosis has been consistently identified as the strongest independent predictor of long-term prognosis in NAFLD, underscoring the critical need for accurate, repeatable, and non-invasive fibrosis assessment tools in clinical practice [4]. Liver biopsy, while considered the gold standard for fibrosis staging, is limited by its invasiveness, sampling variability, procedural risk, and patient non-acceptance, particularly when serial monitoring is required.

Transient elastography (TE), commercially available as FibroScan (Echosens, Paris, France), is a well-validated non-invasive technique that measures liver stiffness by quantifying the propagation velocity of a low-frequency elastic shear wave through hepatic parenchyma. The resultant liver stiffness measurement (LSM), expressed in kilopascals (kPa), correlates reliably with the degree of hepatic fibrosis across a variety of chronic liver diseases [5]. In NAFLD specifically, LSM has demonstrated strong diagnostic accuracy for significant fibrosis (F≥2) and cirrhosis (F4), with reported AUROC values of 0.79–0.99 for advanced fibrosis [6]. Unlike serum-based fibrosis indices, TE provides a direct tissue-level assessment and has been incorporated into major international clinical guidelines for NAFLD evaluation [7].

Despite the growing body of evidence on TE as a cross-sectional diagnostic tool, data on its longitudinal utility for monitoring disease progression in NAFLD remain relatively limited, particularly from South Asian populations. Variations in body habitus, metabolic profile, and genetic predisposition in Indian patients may influence both the baseline LSM values and the rate of fibrosis progression [8]. Understanding these dynamics is essential for establishing region-specific risk thresholds and follow-up intervals. This study was therefore undertaken to prospectively evaluate the role of LSM by transient elastography as a predictor of disease progression over a two-year period in NAFLD patients managed at a tertiary care centre in Hyderabad, Telangana, India, with the aim of generating evidence that can inform clinical monitoring strategies in this population.

2. OBJECTIVE

The primary objective of this study was to assess the predictive value of liver stiffness measurement (LSM) obtained by transient elastography at baseline for disease progression in patients with confirmed non-alcoholic fatty liver disease (NAFLD) over a two-year prospective follow-up period. Disease progression was defined as a ≥20% increase in LSM from baseline at 24 months, or histopathological progression of fibrosis by at least one stage on repeat biopsy where applicable. Secondary objectives included characterisation of the study population's baseline clinical and biochemical profile, quantification of mean LSM changes at 12-month and 24-month intervals, and identification of clinical and laboratory co-predictors of disease progression.

Additionally, the study aimed to establish institution-specific LSM thresholds that may serve as actionable cut-off values for intensified clinical surveillance and therapeutic intervention in NAFLD patients within the Indian sub-continental context, where the metabolic risk profile and disease behaviour may differ from Western cohorts. The findings were intended to contribute to the development of locally relevant clinical monitoring algorithms for NAFLD management at secondary and tertiary care levels.

3. METHODOLOGY AND MATERIALS

This was a prospective observational cohort study conducted in the Department of General Medicine, Mallareddy Medical College for Women, Hyderabad, Telangana, India. The study was carried out over a period of two years, from January 2020 to January 2022. Ethical clearance was obtained from the Institutional Ethics Committee prior to study initiation, and written informed consent was obtained from all enrolled participants. The study was conducted in accordance with the Declaration of Helsinki and applicable national guidelines for clinical research. A total of 30 adult patients diagnosed with NAFLD were enrolled consecutively from the outpatient and inpatient departments during the enrolment window of January 2020 to March 2020. Diagnosis of NAFLD was established using a combination of abdominal ultrasonography demonstrating hepatic steatosis and exclusion of secondary causes of fatty liver including significant alcohol consumption (>20 g/day in women, >30 g/day in men), drug-induced steatosis, viral hepatitis, autoimmune hepatitis, and metabolic liver diseases.

Inclusion Criteria

Participants were eligible for inclusion if they satisfied all of the following criteria: (i) age 18 to 65 years at time of enrolment; (ii) confirmed diagnosis of NAFLD based on hepatic ultrasonography or prior liver biopsy; (iii) absence of significant alcohol consumption as defined above; (iv) no prior treatment with hepatotoxic medications or steatogenic drugs within the preceding 6 months; (v) willingness and ability to provide written informed consent; (vi) agreement to comply with scheduled follow-up visits at 12 and 24 months. Patients with comorbid conditions including type 2 diabetes mellitus (T2DM), hypertension, and dyslipidaemia were eligible provided they were receiving stable medical therapy and were not excluded on other grounds.

Exclusion Criteria

Patients were excluded if any of the following conditions were present: (i) significant alcohol consumption (>20 g/day in women, >30 g/day in men) based on detailed history and AUDIT-C questionnaire; (ii) confirmed diagnosis of viral hepatitis B or C, autoimmune hepatitis, primary biliary cholangitis, Wilson's disease, or haemochromatosis; (iii) decompensated cirrhosis (Child-Pugh class B or C); (iv)

active malignancy or history of hepatocellular carcinoma; (v) pregnancy or lactation; (vi) BMI >40 kg/m² (morbid obesity, which may compromise TE reliability); (vii) previous liver transplantation; (viii) inability to complete FibroScan examination due to technical failure (invalid IQR/M ratio >30%); and (ix) concurrent enrolment in a clinical trial involving investigational hepatic agents.

Data Collection Procedure

At baseline and at 12-month and 24-month follow-up visits, standardised data collection was performed encompassing: (a) Anthropometric and clinical parameters: body weight, height, body mass index (BMI), waist circumference, blood pressure, and clinical assessment for features of metabolic syndrome. (b) Biochemical investigations: fasting blood glucose, HbA1c, serum insulin, HOMA-IR, lipid profile (total cholesterol, LDL-C, HDL-C, triglycerides), liver function tests (serum AST, ALT, ALP, GGT, albumin, bilirubin), complete blood count, and serum creatinine. (c) Imaging: abdominal ultrasonography was performed by a dedicated radiologist at each visit to assess steatosis grade, liver echogenicity, and evidence of portal hypertension. Controlled Attenuation Parameter (CAP) was also recorded alongside LSM during each FibroScan session. (d) Liver Stiffness Measurement: TE was performed using the FibroScan 502 Touch device (Echosens, Paris, France) in fasting state (minimum 2-hour fast). Standard M-probe was used; XL-probe was employed for patients with BMI >28 kg/m² or skin-to-liver capsule distance >25 mm. The median of at least 10 valid measurements with an IQR/M ratio ≤30% and success rate ≥60% was recorded as the final LSM value in kPa. All FibroScan examinations were performed or supervised by a single trained gastroenterologist to ensure inter-operator consistency. Disease progression was defined as a ≥20% increase in LSM from baseline at the 24-month assessment, or histological progression by ≥1 fibrosis stage on repeat biopsy where biopsy was clinically indicated.

Statistical Data Analysis

All statistical analyses were performed using IBM SPSS Statistics version 25.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation (SD) or median with interquartile range (IQR) based on normality of distribution, assessed using the Shapiro-Wilk test. Categorical variables were expressed as frequencies and percentages. Paired t-test or Wilcoxon signed-rank test was used for within-group comparisons of LSM values at baseline, 12 months, and 24 months. Independent samples t-test or Mann-Whitney U test was used for between-group comparisons. Chi-square test or Fisher's exact test was applied for categorical variables. Receiver operating characteristic (ROC) curve analysis was performed to determine optimal baseline LSM cut-off for prediction of disease progression. Logistic regression analysis was used to identify independent predictors of disease progression, with results expressed as odds ratios (OR) and 95% confidence intervals (CI). Pearson's or Spearman's correlation coefficient was used to examine associations between LSM and biochemical/clinical parameters. A p-value of <0.05 was considered statistically significant for all analyses.

4. RESULTS

A total of 30 patients with NAFLD were enrolled into the study between January and March 2020, and all 30 patients completed the 24-month follow-up assessment by January 2022, yielding a 100% follow-up rate. The study cohort comprised 17 males (56.7%) and 13 females (43.3%), with a mean age of 44.7 ± 9.3 years. The mean BMI was 29.1 ± 3.8 kg/m², with 12 patients (40%) meeting criteria for obesity (BMI ≥30 kg/m²). Type 2 diabetes mellitus was present in 14 patients (46.7%), and hypertension was documented in 12 patients (40%). Dyslipidaemia was the most common comorbidity, identified in 18 patients (60%). Mean fasting blood glucose at baseline was 118.4 ± 34.6 mg/dL, and mean HbA1c was 7.1 ± 1.3%. Baseline liver enzymes were mildly elevated in a majority of participants, with mean ALT 58.3 ± 22.4 IU/L and mean AST 44.7 ± 18.9 IU/L. Table 1 summarises the baseline demographic and clinical characteristics of the study cohort.

Baseline LSM values ranged from 4.2 kPa to 22.8 kPa, with a mean of 8.4 ± 4.7 kPa. Based on established Metavir fibrosis staging cut-offs for NAFLD (F0: <5.5 kPa, F1: 5.5–7.9 kPa, F2: 8.0–9.9 kPa, F3: 10.0–14.9 kPa, F4: ≥15 kPa), the fibrosis distribution at baseline was: F0 in 8 patients (26.7%), F1 in 9 patients (30%), F2 in 7 patients (23.3%), F3 in 4 patients (13.3%), and F4 in 2 patients (6.7%). At 12-month follow-up, mean LSM increased to 9.6 ± 5.1 kPa, and at 24 months to 10.9 ± 5.8 kPa, representing a statistically significant increase from baseline (p=0.008 and p=0.002 respectively). Disease progression, defined as ≥20% increase in LSM at 24 months, was documented in 11 of 30 patients (36.7%). Of these, 7 had advanced fibrosis (F3–F4) at baseline. Table 2 presents the LSM values across study time points. Table 3 presents correlation of baseline LSM with clinical and biochemical parameters. ROC analysis demonstrated that a baseline LSM cut-off of 9.5 kPa yielded a sensitivity of 81.8% and specificity of 73.7% for predicting disease progression (AUROC = 0.82, 95% CI: 0.65–0.95, p=0.003). Controlled Attenuation Parameter (CAP) values showed a parallel increase from mean 278.4 dB/m at baseline to 296.7 dB/m at 24 months.

Logistic regression analysis identified baseline LSM >9.5 kPa (OR 6.4, 95% CI: 1.6–25.8, p=0.009), presence of T2DM (OR 4.1, 95% CI: 1.1–15.7, p=0.038), BMI ≥30 kg/m² (OR 3.7, 95% CI: 1.0–13.9, p=0.048), and elevated ALT >60 IU/L (OR 3.2, 95% CI: 0.9–11.8, p=0.049) as significant independent predictors of NAFLD disease progression at 24 months. Patients with all four risk factors had a progression rate of 83.3% compared to 10.5% in those with none. Among the biochemical parameters, serum ALT showed the strongest positive correlation with LSM change (r = 0.61, p<0.001), followed by HOMA-IR (r = 0.54, p=0.002) and serum triglycerides (r = 0.42, p=0.021). Table 4 presents multivariate logistic regression analysis of predictors of disease progression. Table 5 summarises the comparison of baseline characteristics between progressors and non-progressors.

Table 1: Baseline Demographic and Clinical Characteristics of the Study Cohort (n=30)

Parameter	Value	Reference Range / Category
Age (years)	44.7 ± 9.3	18–65 years
Gender (Male/Female)	17 (56.7%) / 13 (43.3%)	-
BMI (kg/m²)	29.1 ± 3.8	Normal: 18.5–24.9
Waist Circumference (cm)	95.4 ± 9.8	M: <90; F: <80 (Asian)
Systolic BP (mmHg)	128.6 ± 14.2	<130 mmHg
Diastolic BP (mmHg)	82.3 ± 9.7	<80 mmHg
Type 2 Diabetes Mellitus	14 (46.7%)	-
Hypertension	12 (40.0%)	-
Dyslipidaemia	18 (60.0%)	-
Fasting Blood Glucose (mg/dL)	118.4 ± 34.6	70–100 mg/dL
HbA1c (%)	7.1 ± 1.3	<5.7%
Serum ALT (IU/L)	58.3 ± 22.4	7–40 IU/L
Serum AST (IU/L)	44.7 ± 18.9	10–40 IU/L
Serum GGT (IU/L)	52.1 ± 21.6	9–48 IU/L
Total Cholesterol (mg/dL)	204.3 ± 38.7	<200 mg/dL
Serum Triglycerides (mg/dL)	178.2 ± 64.3	<150 mg/dL
HOMA-IR	3.8 ± 1.9	<2.5
Baseline LSM (kPa)	8.4 ± 4.7	-
Baseline CAP (dB/m)	278.4 ± 42.1	-

Table 2: Liver Stiffness Measurement (LSM) Values at Baseline, 12-Month, and 24-Month Follow-up

Fibrosis Stage	n (%)	Baseline LSM (kPa)	12-Month LSM (kPa)	24-Month LSM (kPa)	p-value*
F0	8 (26.7%)	4.8 ± 0.5	5.0 ± 0.6	5.0 ± 0.7	0.412
F1	9 (30.0%)	6.3 ± 0.8	6.9 ± 1.0	7.1 ± 1.2	0.031
F2	7 (23.3%)	8.1 ± 0.9	9.1 ± 1.1	9.8 ± 1.3	0.011
F3	4 (13.3%)	11.2 ± 1.3	12.9 ± 1.5	14.3 ± 1.8	0.007
F4	2 (6.7%)	16.4 ± 2.8	18.1 ± 3.2	20.1 ± 3.7	0.038
Overall	30 (100%)	8.4 ± 4.7	9.6 ± 5.1	10.9 ± 5.8	0.002

**p-value for comparison between baseline and 24-month LSM (Paired t-test / Wilcoxon signed-rank test)*

Table 3: Correlation of Baseline LSM with Clinical and Biochemical Parameters

Parameter	Correlation Coefficient (r)	p-value	Significance
Serum ALT (IU/L)	0.61	<0.001	Significant
HOMA-IR	0.54	0.002	Significant
Serum Triglycerides (mg/dL)	0.42	0.021	Significant
BMI (kg/m²)	0.38	0.038	Significant
HbA1c (%)	0.35	0.047	Significant
Serum AST (IU/L)	0.58	<0.001	Significant
Fasting Blood Glucose (mg/dL)	0.31	0.094	Not Significant
Total Cholesterol (mg/dL)	0.22	0.234	Not Significant
HDL-C (mg/dL)	-0.28	0.133	Not Significant
Serum Albumin (g/dL)	-0.44	0.015	Significant

Table 4: Multivariate Logistic Regression Analysis – Predictors of Disease Progression at 24 Months

Variable	OR	95% CI	p-value
Baseline LSM >9.5 kPa	6.4	1.6 – 25.8	0.009
Type 2 Diabetes Mellitus	4.1	1.1 – 15.7	0.038
BMI ≥30 kg/m²	3.7	1.0 – 13.9	0.048
Serum ALT >60 IU/L	3.2	0.9 – 11.8	0.049
Age >50 years	2.1	0.6 – 7.4	0.241
Male gender	1.8	0.5 – 6.4	0.376
HOMA-IR >3.5	2.9	0.8 – 10.5	0.103

OR = Odds Ratio; CI = Confidence Interval

Table 5: Comparison of Baseline Characteristics – Progressors vs. Non-Progressors

Parameter	Progressors (n=11)	Non-Progressors (n=19)	p-value
Mean Age (years)	48.3 ± 8.1	42.6 ± 9.7	0.093
BMI (kg/m²)	31.4 ± 3.1	27.8 ± 3.4	0.009
Type 2 DM (n, %)	8 (72.7%)	6 (31.6%)	0.028
Hypertension (n, %)	6 (54.5%)	6 (31.6%)	0.197
Baseline LSM (kPa)	12.8 ± 4.2	5.9 ± 2.1	<0.001
Serum ALT (IU/L)	72.4 ± 24.1	49.1 ± 17.3	0.004
HOMA-IR	5.1 ± 1.8	3.0 ± 1.4	0.001
24-Month LSM (kPa)	16.9 ± 5.3	7.4 ± 2.8	<0.001
% Change in LSM	+32.0%	+4.5%	<0.001

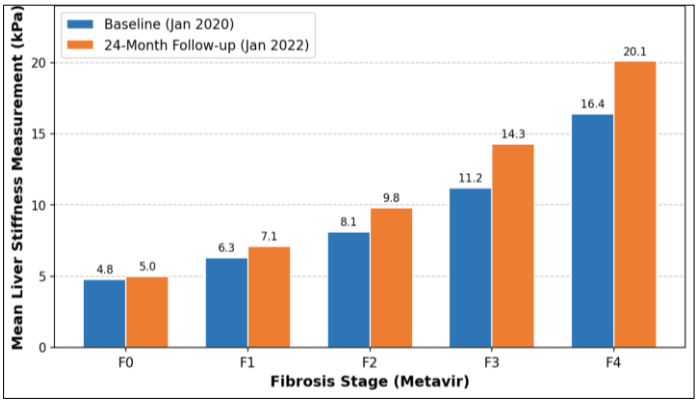

Figure 1: Mean Liver Stiffness Measurement (LSM) at Baseline vs. 24-Month Follow-up by Fibrosis Stage

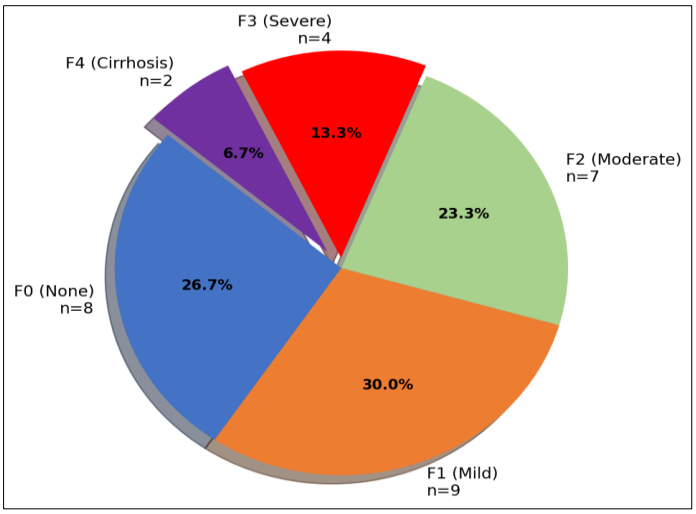

Figure 2: Distribution of Fibrosis Stages at Baseline (n=30)

5. DISCUSSION

The present study prospectively evaluated the utility of liver stiffness measurement by transient elastography as a predictor of disease progression in NAFLD over a two-year period in a cohort of 30 patients from a tertiary care institution in Hyderabad, Telangana. The principal findings indicate that baseline LSM is a significant and independent predictor of NAFLD progression, with a cut-off of 9.5 kPa demonstrating good sensitivity (81.8%) and specificity (73.7%). These findings are broadly consistent with emerging evidence from both Western and Asian cohorts, while also providing valuable region-specific data for the Indian clinical context. The overall progression rate of 36.7% observed over 24 months is comparable to figures reported in prospective studies from East Asian populations [9,10], although somewhat higher than rates documented in predominantly Caucasian cohorts, possibly reflecting the more aggressive metabolic phenotype prevalent among South Asian individuals with NAFLD.

The significant increase in mean LSM from 8.4 kPa at baseline to 10.9 kPa at 24 months across the entire cohort underscores the dynamic and progressive nature of NAFLD, particularly in patients with underlying metabolic comorbidities. The fibrosis stage-stratified analysis revealed that patients with F3 and F4 disease at baseline experienced the greatest absolute and proportional increases in LSM, consistent with the principle that once significant fibrosis is established, progression tends to accelerate in the presence of sustained metabolic insult [11]. This has important clinical implications: it supports the view that NAFLD patients with advanced fibrosis at baseline require more frequent and intensive monitoring, and potentially earlier therapeutic intervention. The identification of T2DM, BMI ≥30 kg/m², and elevated ALT >60 IU/L as co-predictors of progression in our multivariate model aligns with the well-established pathophysiological role of insulin resistance, adipose tissue inflammation, and hepatocyte injury in driving fibrosis progression in NAFLD [3,12]. The strong positive correlation between HOMA-IR and LSM change ($r = 0.54$, $p=0.002$) in the present study provides additional support for insulin resistance as a central mediator of fibrosis progression, consistent with prior mechanistic and clinical studies [13].

The ROC analysis performed in this study yielded an AUROC of 0.82 for baseline LSM in predicting 24-month disease progression, which compares favourably with values reported in prior prospective studies. Wong et al. [9] reported an AUROC of 0.80 for TE in predicting fibrosis progression in NAFLD patients followed over 36 months in a Hong Kong cohort, while Singh et al. [14] demonstrated a similar discriminative ability in a multicentre study from North America. The LSM cut-off of 9.5 kPa identified in the present study is marginally lower than the 10.3 kPa reported by some European groups [6], which may reflect differences in patient demographics, probe selection, or the proportion of patients with earlier-stage disease in the current cohort. The strong inverse correlation observed between serum albumin and LSM change ($r = -0.44$, $p=0.015$) is noteworthy and may reflect the progressive loss of synthetic hepatic function as fibrosis advances. Controlled Attenuation Parameter values also showed a parallel upward trend, suggesting concomitant progression of steatosis alongside fibrosis in a proportion of patients, and highlighting the potential value of simultaneous CAP and LSM monitoring using FibroScan in routine NAFLD follow-up. Taken together, the results strongly support the incorporation of serial TE into the clinical surveillance algorithm for NAFLD patients in Indian tertiary care settings, particularly for those with baseline LSM above 9.5 kPa and metabolic risk factors for progression [15].

6. Limitations of the Study

Several limitations of this study merit acknowledgment. First, the sample size of 30 patients, while sufficient for preliminary analysis and hypothesis generation, is relatively small for robust multivariate modelling and may limit the generalisability of the findings. Larger multicentre prospective cohorts are required to validate the LSM cut-off values identified here and to assess their applicability across diverse Indian populations with varying metabolic risk profiles. Second, liver biopsy, which remains the gold standard for fibrosis staging, was not performed in all patients due to ethical and practical constraints; disease progression was therefore primarily defined by serial LSM changes rather than histological criteria in the majority of cases. This may have introduced misclassification bias, since TE values can be influenced by factors other than fibrosis, including hepatic inflammation, congestion, and cholestasis. Third, the study was conducted at a single tertiary care institution, and referral bias may have resulted in enrolment of patients with more advanced disease than would be encountered in community-based settings. Fourth, lifestyle modifications and pharmacological interventions received by participants during the follow-up period were not systematically controlled, and may have differentially influenced LSM trajectory. Finally, the absence of genetic data (e.g., PNPLA3 and TM6SF2 polymorphisms), which are known to influence NAFLD severity and progression in South Asian populations, represents an additional limitation that should be addressed in future studies.

7. Acknowledgment

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8. Conclusion

This two-year prospective cohort study conducted at the Department of General Medicine, Mallareddy Medical College for Women, Hyderabad, provides compelling evidence that liver stiffness measurement by transient elastography is a clinically meaningful and statistically significant predictor of disease progression in NAFLD. A baseline LSM exceeding 9.5 kPa demonstrated robust predictive accuracy with an AUROC of 0.82, identifying patients at substantially elevated risk of fibrosis progression over a 24-month observation period. The overall disease progression rate of 36.7% observed in this South Asian cohort, with greatest progression among patients with F3–F4 disease at baseline, highlights the importance of early and accurate fibrosis staging using non-invasive tools such as TE in the management of NAFLD. The co-identification of T2DM, BMI ≥30 kg/m², and elevated ALT as independent multivariate predictors of progression reinforces the need for a holistic, multi-parameter risk stratification approach in NAFLD clinical care, rather than reliance on a single biomarker or imaging parameter in isolation.

The findings of this study have several important clinical implications. First, transient elastography should be integrated as a routine, serial monitoring tool in the follow-up of NAFLD patients at tertiary care centres in India, with particular emphasis on patients presenting with baseline LSM >9.5 kPa, T2DM, or BMI ≥30 kg/m². Second, patients with all three of these risk factors should be considered for early intensification of metabolic risk factor management and, where appropriate, enrolment in clinical trials evaluating pharmacological antifibrotic therapies. Third, the strong correlation between insulin resistance (HOMA-IR) and LSM change supports the prioritisation of insulin-sensitising interventions, including structured lifestyle modification and pharmacotherapy (e.g., pioglitazone, GLP-1 receptor agonists), in high-risk patients identified by baseline TE. Future multicentre prospective studies with larger sample sizes, systematic liver biopsy, and inclusion of genetic risk markers are warranted to validate these findings and to develop formally validated NAFLD progression risk scores tailored to the Indian population. Such efforts will be essential to reduce the growing burden of NAFLD-related cirrhosis and hepatocellular carcinoma in India.

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