

Original Article

Correlation of Structured Lifestyle Modification Versus Standard Care with Progression Markers from Non-Alcoholic Fatty Liver Disease to Decompensated Chronic Liver Disease In Adults Aged 30-60 Years

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Abstract

Objectives: To assess the relationship between well-documented lifestyle changes and non-invasive measures of liver fibrosis and portal hypertension in adults aged 30-60 years who have imaging-proven NAFLD.

Methodology: This comparative cross-sectional study Department of General Medicine, Sandeman Provincial Hospital / Bolan Medical College / Hospital, Quetta. From July 2025 to December 2025. We used consecutive non-probability sampling to recruit 64 adults with an imaging diagnosis of NAFLD, who were randomly allocated to either a structured lifestyle-modification group (SLM; n=32) or a standard-care group (SC; n=32). The program included six months of structured diet counseling, supervised exercise, and behavioral support. We assessed APRI, FIB-4, AST/ALT ratio, platelet count, and ultrasound findings. We analyzed data using SPSS and used $p < 0.05$ as the cut-off.

Results: The overall mean age was 46.1 ± 8.2 years; group means were 45.4 ± 8.0 and 46.8 ± 8.4 years, respectively ($p = 0.74$). Males comprised 39 (60.9%) participants. Compared with standard care, structured lifestyle modification was associated with lower APRI (0.62 ± 0.24 vs. 0.83 ± 0.31 , $p = 0.003$), FIB-4 (1.21 ± 0.48 vs. 1.62 ± 0.59 , $p = 0.005$), and AST/ALT ratio (0.78 ± 0.18 vs. 0.91 ± 0.22 , $p = 0.012$). Platelet count was higher (189.6 ± 42.7 vs. $171.2 \pm 39.8 \times 10^9/L$, $p = 0.087$). Ultrasound evidence of splenomegaly or minimal ascites was less frequent (12.5% vs. 31.3%, $p = 0.048$). After adjustment, structured lifestyle modification remained associated with lower APRI ($p = 0.018$) and FIB-4 ($p = 0.021$).

Conclusion: Structured lifestyle modification was associated with more favorable non-invasive markers of hepatic fibrosis and early portal hypertension than standard care. Prospective studies with longer follow-up are needed to validate these results.

Introduction

Non-alcoholic fatty liver disease (NAFLD) is now one of the most prevalent chronic liver diseases globally and is an emerging cause of liver disease morbidity and mortality. It is defined as an excessive amount of fat buildup in the liver, without significant alcohol use. It includes a wide spectrum of clinical disease from simple hepatic steatosis to non-alcoholic steatohepatitis, to progressive hepatic fibrosis, cirrhosis, and decompensated chronic liver disease. The rise in prevalence of obesity, type 2 diabetes mellitus, metabolic syndrome, and sedentary lifestyle has made a significant contribution to the growing burden of NAFLD. Around 25% of the adult population has NAFLD, making it a public health problem [1]. Metabolic and lifestyle factors play a major role in driving the progression of NAFLD. Obesity, especially central adiposity, is common in people with NAFLD, and type 2 diabetes mellitus is a significant risk factor for disease progression and worsening liver disease. Insulin resistance, lipid metabolism abnormalities, oxidative stress, chronic inflammation, and dysregulated adipokine activity mediate hepatocellular injury and fibrogenesis. Many patients are clinically stable for years, but a subset will develop progressive fibrosis and cirrhosis, ultimately resulting in portal hypertension, ascites, variceal bleeding, hepatic encephalopathy, and other features of decompensated chronic liver disease [2,3]. Therefore, it is important to identify patients at increased risk of progression before it occurs. Biopsy is an important reference test for evaluating liver fibrosis, but it is invasive, costly, variable, and not easily repeated, so it is not used routinely. For this reason, non-invasive markers like the AST-to-Platelet Ratio Index (APRI) and Fibrosis-4 (FIB4) index are now clinically important. These indices rely on readily available clinical and laboratory data to predict the risk of significant or advanced fibrosis. Other useful parameters include the AST/ALT ratio and platelet count, which may indicate hepatic injury and portal-hypertensive changes. Moreover, findings on abdominal ultrasound may be suggestive of early portal hypertension (splenomegaly and minimal ascites) [4,5]. Lifestyle modification is the mainstay of NAFLD management. Current management strategies focus on weight loss, dietary modification, regular physical activity, and control of metabolic risk factors. Even without weight loss, regular aerobic and resistance exercise can improve insulin sensitivity, body composition, liver enzyme levels, and metabolic health. Hypocaloric and Mediterranean dietary patterns may help decrease hepatic fat content and improve metabolic abnormalities. Further dietary and exercise advice and ongoing monitoring could still help increase adherence [6]. However, despite the well-recognized contribution of lifestyle changes to NAFLD management, little clinical evidence shows the effects of a structured, multidisciplinary lifestyle program on objective measures of liver disease progression compared with routine standard care. Typical treatment is mostly a brief verbal counseling session about healthy eating and increased exercise, with no further follow-up, supervised exercise, dietary checks, or behavioral therapy. A structured program, however, offers predetermined diet counseling, supervised exercise, behavioral reinforcement, attendance monitoring, and adherence assessment [7]. The present study was thus aimed at comparing patients receiving structured lifestyle modification with those receiving standard care and at exploring the correlation with non-invasive markers of liver fibrosis and early portal hypertension. The study aims to assess whether the structured lifestyle intervention is associated with more favorable progression markers in adults with NAFLD confirmed by imaging, using APRI, AST/ALT ratio, platelet count, and ultrasound findings. The results could provide clinically relevant evidence for structured lifestyle-based management and early risk assessment in patients at risk of progressive liver disease [8,9].

Materials and Methods

Study Design & Setting

This comparative cross-sectional study Department of General Medicine, Sandeman Provincial Hospital / Bolan Medical College / Hospital, Quetta. From July 2025 to December 2025.

Participants

Sixty-four adults (aged 30–60 years) with imaging-confirmed NAFLD were consecutively sampled. We randomly assigned 32 participants to a structured lifestyle-modification group and 32 to a standard-care group. The structured group received usual clinical lifestyle advice plus dietary counseling, supervised exercise, and behavioral support for at least six months, while the standard-care group received usual clinical lifestyle advice.

Sample Size Calculation

The sample size was determined based on a population mean difference of 0.28 in the ALT/AST ratio; a population mean of 1.18 and a population standard deviation of 0.38 for ALT/AST; and a population mean of 0.90 and a population standard deviation of 0.32 for the final clinical findings. The sample size for each group was determined to be 25, with 80% power at an alpha of 0.05 (two-tailed). We allowed a total of 32 participants per group (20% non-response), for a total of 64 participants across the groups.

Ethical Approval

Ethical approval was obtained from the Research Evaluation Unit, College of Physicians and Surgeons Pakistan (CPSP), under Ref. No. CPSP/REU/MED-2024-001-22782, dated June 16, 2025, prior to study commencement.

Inclusion Criteria

- Adults (30-60 years of age) of both sexes.
- Abdominal ultrasound or transient elastography performed to confirm NAFLD in the last year.
- Moderate amount of alcohol consumption (less than 21 standard drinks per week for men and less than 14 standard drinks per week for women).
- Structured-program participants were enrolled in dietary counseling, supervised exercise, and behavioral support for at least six months.
- Participants in standard care who are given standard clinical care and lifestyle recommendations for at least six months.
- Recent AST, ALT, platelet count, and ultrasound results.
- Capacity and consent to give written informed consent.

Exclusion Criteria

- Viral hepatitis B or C and other chronic liver diseases.
- Autoimmune hepatitis, hemochromatosis, Wilson disease, or alpha-1-antitrypsin (A1AT) deficiency.

- Cirrhosis or decompensated liver disease (ascites, variceal bleeding, or clinical hepatic encephalopathy (HE)).
- Excessive alcohol intake of the above levels.
- End-stage renal disease, severe heart failure, or active malignancy.
- Pregnancy or lactation.
- Impaired cognition or psychiatric disorder that impairs consent or ability to complete questionnaires.
- Inadequate laboratory or imaging information.

Diagnostic and Management Strategy

We diagnosed NAFLD by imaging and evaluated disease progression using APRI, FIB-4, AST/ALT ratio, platelet count, and evidence of splenomegaly or minimal ascites on ultrasound. Structured management included dietary counseling, supervised exercise (at least 150 minutes per week), and behavioral support, while standard care consisted of brief routine lifestyle counseling.

Statistical Analysis

SPSS was used to enter the data and perform the analysis. We reported continuous variables as mean ± standard deviation (SD) and categorical variables as frequencies and percentages. We used the Shapiro-Wilk test and Q-Q plots to assess normality. Appropriate parametric or non-parametric tests were used to compare between groups. We evaluated associations between intervention/adherence and progression markers using Pearson or Spearman correlation—partial correlations controlled for age, gender, BMI, and diabetes. We considered $p < 0.05$ (two-tailed) statistically significant.

Results

We included 64 patients with imaging-confirmed NAFLD: 32 (50.0%) in the structured lifestyle-modification group and 32 (50.0%) in the standard-care group. The overall mean age was 46.1 ± 8.2 years. The mean age was 45.4 ± 8.0 years in the structured-program group and 46.8 ± 8.4 years in the standard-care group, with no statistically significant difference between groups ($p=0.74$). Overall, 39 (60.9%) participants were male, and 25 (39.1%) were female. Patients receiving structured lifestyle modification demonstrated significantly more favorable fibrosis-related markers than those receiving standard care. The mean APRI was 0.62 ± 0.24 in the structured group compared with 0.83 ± 0.31 in the standard-care group ($p=0.003$). Similarly, the mean FIB-4 index was significantly lower among participants receiving structured intervention (1.21 ± 0.48 vs. 1.62 ± 0.59 , $p=0.005$). The AST/ALT ratio was also significantly lower in the structured group than in the standard-care group (0.78 ± 0.18 vs. 0.91 ± 0.22 , $p=0.012$). The mean platelet count was higher among patients receiving structured lifestyle modification ($189.6 \pm 42.7 \times 10^9/L$) than among those receiving standard care ($171.2 \pm 39.8 \times 10^9/L$), although the difference was not statistically significant ($p=0.087$). Ultrasound evidence of splenomegaly or minimal ascites was identified in 12.5% of patients in the structured group compared with 31.3% in the standard-care group ($p=0.048$). Greater adherence to the structured program was associated with progressively lower APRI and FIB-4 values. After adjustment for age, gender, BMI, and diabetes status, structured lifestyle modification remained significantly associated with lower APRI ($p=0.018$) and FIB-4 ($p=0.021$).

Table 1. Demographic and Clinical Characteristics of Study Participants

Variable	Total (n=64)	Structured Lifestyle (n=32)	Standard Care (n=32)	p-value
Age (years), mean ± SD	46.1 ± 8.2	45.4 ± 8.0	46.8 ± 8.4	0.740
Male, n (%)	39 (60.9)	—	—	—
Female, n (%)	25 (39.1)	—	—	—
BMI (kg/m ²), mean ± SD	—	—	—	—
Type 2 diabetes, n (%)	—	—	—	—
Hypertension, n (%)	—	—	—	—
Dyslipidemia, n (%)	—	—	—	—
Duration of NAFLD (months), mean ± SD	—	—	—	—

Data are presented as mean ± standard deviation (SD) for continuous variables and frequency (percentage) for categorical variables. p-values represent comparisons between the structured lifestyle-modification and standard-care groups. Values marked “—” require confirmation from the study dataset.

Table 2. Comparison of Non-Invasive Liver Fibrosis Markers Between Study Groups

Liver-related Marker	Structured Lifestyle (n=32) Mean ± SD	Standard Care (n=32) Mean ± SD	p-value
APRI	0.62 ± 0.24	0.83 ± 0.31	0.003
FIB-4 index	1.21 ± 0.48	1.62 ± 0.59	0.005
AST/ALT ratio	0.78 ± 0.18	0.91 ± 0.22	0.012
Platelet count (×10 ⁹ /L)	189.6 ± 42.7	171.2 ± 39.8	0.087

APRI = AST-to-Platelet Ratio Index; FIB-4 = Fibrosis-4 index; AST = aspartate aminotransferase; ALT = alanine aminotransferase. Values are expressed as mean ± SD. We considered $p < 0.05$ statistically significant.

Table 3. Ultrasound Findings Suggestive of Early Portal Hypertension

Ultrasound Finding	Structured Lifestyle (n=32)	Standard Care (n=32)	p-value
Splenomegaly/minimal ascites, n (%)	4 (12.5)	10 (31.3)	0.048
No ultrasound evidence of portal hypertension, n (%)	28 (87.5)	22 (68.7)	0.048

Ultrasound evidence of early portal-hypertensive change was defined as splenomegaly (>13 cm) or minimal ascites. Data are presented as

frequency and percentage. Differences between groups were statistically significant at $p<0.05$.

Table 4. Association of Structured Lifestyle Modification With Liver Progression Markers After Adjustment

Outcome Marker	Direction of Association	Adjusted p-value	Statistical Interpretation
APRI	Lower with structured lifestyle modification	0.018	Significant
FIB-4 index	Lower with structured lifestyle modification	0.021	Significant
AST/ALT ratio	Lower with structured lifestyle modification	—	Requires SPSS confirmation
Platelet count	Higher with structured lifestyle modification	—	Requires SPSS confirmation
Ultrasound portal-hypertension signs	Less frequent with structured lifestyle modification	—	Requires SPSS confirmation

Partial correlation analysis was adjusted for age, gender, BMI, and diabetes status. APRI = AST-to-Platelet Ratio Index; FIB-4 = Fibrosis-4 index. We considered a two-tailed p -value <0.05 statistically significant. The adjusted p -values shown for APRI and FIB-4 are those reported in the study results; obtain the remaining adjusted values from the final SPSS analysis.

Discussion

This study showed that, among adults with imaging-documented NAFLD, structured lifestyle changes were correlated with better non-invasive measures of hepatic fibrosis and early portal hypertension than standard care. The mean levels of APRI, FIB-4, and AST/ALT ratio were significantly lower in the structured lifestyle group, and the platelet count was numerically higher in this cohort of 64 patients. The structured group also had less splenomegaly and minimal ascites on ultrasound. The results indicated that a multi-component intervention (dietary counseling, supervised exercise, and behavioral support) may be more effective than a short, unstructured lifestyle intervention [10,11]. Clinically important, the structured group had lower APRI and FIB-4 values, as these scores offer easy-to-use strategies for identifying patients at elevated risk of advanced fibrosis. International guidance increasingly supports sequential use of blood-based fibrosis scores, specifically the FIB-4 score, with imaging-based assessment used when indicated. The 2024 EASL-EASD-EASO guidelines also highlight lifestyle changes, weight loss, diet, and exercise as key pillars of metabolic dysfunction-associated steatotic liver disease (MASLD), the new term for most patients formerly labeled as having NAFLD [12]. The results agree with the systematic review and meta-analysis by Fernández et al., which included 30 randomized controlled trials and reported greater decreases in ALT, AST, and HOMA-IR with combined diet and exercise than with either intervention alone. The authors also speculate that supervised,

longer interventions might yield better results. The present study further confirms these findings, showing these differences in not only traditional biochemical markers but also markers more directly involved in fibrosis risk, such as APRI and FIB-4 [13]. In another meta-analysis of randomized trials published in 2021 by Kawaguchi et al., they found a significant decrease in fatty liver index and insulin resistance in randomized trials that used the Mediterranean diet. This supports implementing the dietary component of structured interventions that include a hypocaloric Mediterranean diet. In a 2022 systematic review and meta-analysis of 26 studies and 3037 individuals by Haigh et al., dietary interventions decreased ALT, AST, fatty liver index, hepatic steatosis, and hepatic stiffness. Other advantages of Mediterranean-diet interventions included beneficial effects on ALT, fatty liver index, and liver stiffness [14,15]. Our findings are also supported by evidence relating to physical activity. In a 2021 meta-analysis, Zhang et al. showed that various exercise types improved several hepatic and metabolic parameters in NAFLD. A recent (2022) network meta-analysis by Li et al. also compared different exercise strategies and found positive results for all aerobic, resistance, and combined exercise types. A recent systematic review and network meta-analysis from 2024, which included 43 studies and 2070 patients, also confirmed the positive impact of exercise interventions on liver-related outcomes [16]. However, not all recent evidence shows that exercise can improve fibrosis. A systematic review and meta-analysis published in 2022 examined aerobic exercise and concluded that evidence for its benefits was less clear for advanced fibrosis and NASH. But another meta-analysis of biopsy-proven NAFLD found that exercise alone may not be an effective histological treatment [17]. This separation could explain why the present structured intervention (with both exercise and dietary/behavioral components) had more favorable surrogate markers of fibrosis than standard care. Similarly, Chai et al. reported in their 2023 systematic review that weight loss from lifestyle interventions, mainly dietary changes and exercise, improved hepatic fat and metabolic parameters. Meta-analyses of sedentary-lifestyle modification support the metabolic pathway by which increased physical activity could improve hepatic health, as shown by improvements in ALT, glucose, insulin, and HOMA-IR [18]. Finally, the present AASLD guidelines highlight that weight loss reduces steatosis, NASH, and fibrosis in a dose-dependent manner, and that multidisciplinary approaches can help increase uptake of liver-protective behaviors. The lower incidence of ultrasound findings suggestive of early portal hypertension in the structured group is encouraging. Still, it should be taken with a pinch of salt, as ultrasound is less sensitive than advanced fibrosis assessment, and this study was cross-sectional. The relatively small sample size, non-probability sampling, use of surrogate markers, and lack of histological confirmation limit causal interpretation. However, the consistency of results across APRI, FIB-4, the AST/ALT ratio, and ultrasound suggests a potential correlation between structured lifestyle intervention and improved hepatic disease markers. Further studies with larger numbers of patients, objective measures of weight loss and adherence, elastography, and longer follow-up are needed to determine whether these benefits reduce the incidence of cirrhosis and decompensated chronic liver disease [19,20]. This study has several limitations. Its comparative cross-sectional design makes causal inference and temporal evaluation of disease progression difficult. The sample size was relatively small and may have limited the generalizability of the sample (consecutive non-probability sampling). Liver fibrosis was not determined by liver biopsy or advanced liver elastography but by non-invasive markers and ultrasound. Recall and reporting bias may also occur when subjects report their own symptoms.

Conclusion

Structured lifestyle modification was associated with improved non-invasive markers of hepatic fibrosis and early portal hypertension

compared with standard care among patients with NAFLD. Findings support multidisciplinary nutritional, physical activity and/or behavioral interventions. These associations require confirmation in larger, longer follow-up studies, and the impact on clinical progression needs clarification.

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Conflict of Interest: Nil

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Authors Contributions

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