Ann Mil Health Sci Res. 2026 June; 24(2): e172370 https://doi.org/10.69107/amh-172370

Published Online: 2026 August 8 **Research Article**

Intermittent fasting and its association with metabolic, inflammatory, and fibrosis-related outcomes in adults with non-alcoholic fatty liver disease: Findings from a randomized clinical trial

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Received: 30 May, 2026; **Revised:** 27 June, 2026; **Accepted:** 2 August, 2026

Abstract

Background: Non-alcoholic fatty liver disease (NAFLD) is a leading cause of chronic liver dysfunction worldwide and commonly coexists with obesity, insulin resistance, and other metabolic disturbances. Lifestyle-based strategies remain central to disease management, and intermittent fasting has recently emerged as a potentially beneficial nutritional intervention.

Objectives: This study aimed to evaluate whether intermittent fasting affects anthropometric measures, inflammatory biomarkers, fibrosis-related indices, and ultrasonographic manifestations of hepatic steatosis in individuals with NAFLD.

Methods: This randomized clinical trial included 102 adults with ultrasonography-confirmed NAFLD who were randomly assigned in a 1:1 ratio to either a 16:8 intermittent fasting intervention or a control group for 12 weeks. Clinical and laboratory evaluations were performed at baseline and after the intervention. Outcomes included anthropometric measures, inflammatory markers, liver-related laboratory parameters, noninvasive fibrosis-related indices (FIB-4, APRI, NAFLD Fibrosis Score, and BARD score), and an ultrasonographic assessment of hepatic steatosis.

Results: Participants assigned to the intermittent fasting regimen showed significant reductions in body weight, body mass index (BMI), and waist circumference compared with controls (all $P < 0.001$). Marked reductions were also observed in inflammatory markers, particularly the erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP). In addition, significant improvements were observed across all evaluated non-invasive fibrosis indices, including FIB-4, APRI, NAFLD Fibrosis Score, and BARD score. Ultrasonographic evaluation demonstrated a greater reduction in hepatic steatosis severity among participants undergoing intermittent fasting than among controls.

Conclusions: Intermittent fasting appears to be a feasible and potentially beneficial dietary strategy for patients with NAFLD, with favorable effects on noninvasive, fibrosis-related surrogate markers. Further long-term studies incorporating histological endpoints are needed to clarify its role in preventing or delaying fibrosis progression.

Keywords: Intermittent Fasting, NAFLD, FIB-4, APRI, Hepatic Steatosis, Metabolic Health

1. Background

Metabolic dysfunction-associated steatotic liver disease (MASLD), previously known as non-alcoholic fatty liver disease (NAFLD), is a leading cause of chronic liver disease worldwide. Because this study was designed before the adoption of the MASLD nomenclature, the term NAFLD is used throughout this manuscript for consistency.

NAFLD affects approximately one-quarter of the adult population and encompasses a spectrum ranging from simple steatosis to non-alcoholic steatohepatitis (NASH),

How to Cite: Aghajanpour Pasha M, Mousavi V, Saidi S, Baloochi K, Fathipour N. Intermittent fasting and its association with metabolic, inflammatory, and fibrosis-related outcomes in adults with non-alcoholic fatty liver disease: Findings from a randomized clinical trial. Ann Mil Health Sci Res. 2026;24(2):e172370. doi: https://doi.org/10.69107/amh-172370

fibrosis, cirrhosis, and hepatocellular carcinoma (1, 2). Disease progression is closely associated with obesity, insulin resistance, type 2 diabetes, and other metabolic abnormalities (3).

Hepatic fibrosis is the strongest predictor of liver-related complications and mortality in NAFLD (4). Because no pharmacological therapy has been universally accepted, lifestyle modification remains the cornerstone of management. Sustained weight loss through dietary intervention and physical activity improves hepatic steatosis and may slow fibrosis progression; however, long-term adherence to conventional calorie-restricted diets remains challenging (5-7).

Intermittent fasting (IF) has emerged as a promising dietary strategy for improving metabolic health. Recent studies have demonstrated beneficial effects on body weight, hepatic fat accumulation, liver enzymes, and metabolic parameters in patients with NAFLD, with efficacy comparable to or greater than continuous calorie restriction in some settings (8-13). However, evidence regarding its effects on hepatic fibrosis, particularly when assessed using validated non-invasive fibrosis indices, remains limited.

Several validated non-invasive scoring systems, including the Fibrosis-4 (FIB-4) index, AST-to-Platelet Ratio Index (APRI), NAFLD Fibrosis Score (NFS), and BARD score, are widely used to estimate fibrosis risk without liver biopsy. Nevertheless, randomized trials that simultaneously evaluate the effects of intermittent fasting on metabolic parameters, inflammatory markers, imaging findings, and fibrosis-related indices remain scarce.

2. Objectives

This randomized controlled trial aimed to evaluate the effects of a 12-week 16:8 intermittent fasting regimen on anthropometric measures, inflammatory biomarkers, non-invasive fibrosis indices (FIB-4, APRI, NAFLD Fibrosis Score, and BARD score), and hepatic steatosis assessed by ultrasonography in adults with NAFLD.

3. Methods

3.1. Study Design and Participants

This 12-week prospective randomized controlled trial included 102 adults with ultrasonography-confirmed NAFLD.

Eligible participants were men and women aged 18 - 50 years with Grade 1 or 2 hepatic steatosis. Exclusion criteria included diabetes mellitus, hypertension, dyslipidemia, Grade ≥ 3 steatosis, significant alcohol intake, viral or autoimmune liver disease, malignancy, chronic cardiovascular or renal disease, previous bariatric surgery, recent use of antibiotics, corticosteroids, nonsteroidal anti-inflammatory drugs (NSAIDs), hormonal or hepatotoxic medications, thyroid medications, vegetarian or weight-loss diets, recent major weight change (>10% loss or > 5% gain within 6 months), and other causes of secondary hepatic steatosis.

3.2. Sample Size

The sample size was calculated using G*Power. Assuming an effect size of 0.5, $\alpha = 0.05$, and 80% power, 51 participants per group were required (total n = 102).

3.3. Baseline Assessment

Baseline evaluation included demographic characteristics, medical history, anthropometric measurements (weight, height, BMI, and waist circumference), and fasting laboratory tests, including aspartate aminotransferase (AST), alanine aminotransferase (ALT), platelet count, fasting glucose, albumin, ESR, and CRP.

For each participant, fibrosis risk was assessed using four validated non-invasive surrogate indices: the FIB-4 index, APRI, the NAFLD Fibrosis Score, and the BARD score. These scoring systems were selected because they are widely recommended, readily applicable in routine clinical practice, and provide validated estimates of fibrosis risk without the need for liver biopsy.

3.4. Ultrasonography

Abdominal ultrasonography was performed at baseline and after 12 weeks by experienced radiologists at the same imaging center, using standardized criteria. Hepatic steatosis was graded as mild (Grade 1) or moderate (Grade 2) based on liver echogenicity, visualization of intrahepatic vessels, and posterior acoustic attenuation.

3.5. Randomization and Intervention

Eligible participants were recruited consecutively from the outpatient clinic after confirmation of eligibility and provision of written informed consent. Participants were then randomized in a 1:1 ratio to the intermittent fasting group or the control group using a lottery-based allocation procedure. Group assignments were prepared in advance and placed in sealed opaque envelopes. The allocation sequence was generated

before participant enrollment. After enrollment, each participant selected one envelope, and the enclosed assignment determined group allocation.

Participants allocated to the intermittent fasting group followed a 16:8 time-restricted eating protocol for 12 weeks, consisting of a daily 16-hour fasting period and an 8-hour eating window. No predefined caloric restriction was imposed, and participants were instructed to maintain their usual dietary composition throughout the study period. Water and other non-caloric beverages were allowed during fasting.

The control group received standard lifestyle advice without a structured fasting program.

Medication regimens remained unchanged throughout follow-up. Adherence was monitored through weekly telephone contact and participant fasting logs.

3.6. Statistical Analysis

Statistical analyses were performed using IBM SPSS Statistics version 31. Continuous variables were assessed for normality using the Shapiro-Wilk test and are presented as mean ± SD or median (IQR), as appropriate.

Because several variables were non-normally distributed, non-parametric analyses were used throughout. Baseline comparisons were performed using the Mann-Whitney U test. Within-group changes were analyzed using the Wilcoxon signed-rank test, whereas between-group comparisons were based on change-from-baseline (Δ) values using the Mann-Whitney U test. Categorical variables were compared using the chi-square test or Fisher exact test.

All tests were two-sided, and $P < 0.05$ was considered statistically significant.

3.7. Ethical Considerations

The study was approved by the Ethics Committee of AJA University of Medical Sciences, Tehran, Iran (IR.AJAUMS.REC.1404.021). Written informed consent was obtained from all participants before enrollment. The trial was not prospectively registered.

4. Results

A total of 102 participants were randomized, with 51 assigned to the intermittent fasting group and 51 to the control group. All participants completed the 12-week follow-up and were included in the final analysis (Figure 1). No outcome data were missing.

Baseline demographic, clinical, laboratory, and ultrasonographic characteristics were comparable between groups (Table 1), except for platelet count, which was significantly higher in the intermittent fasting group ($P = 0.008$).

4.1. Anthropometric Outcomes

Compared with controls, participants in the intermittent fasting group showed significantly greater reductions in body weight, BMI, and waist circumference after 12 weeks (all $P < 0.001$) (Table 2). No significant within-group changes were observed in the control group. The magnitude of weight reduction in the intermittent fasting group was greater than that reported in several previous studies. This finding may reflect high adherence to the intervention, differences in baseline anthropometric characteristics, and the relatively intensive nature of the fasting protocol. However, because dietary intake was not formally quantified during the intervention, the relative contributions of intermittent fasting and spontaneous caloric restriction cannot be determined. Therefore, these findings should be interpreted with caution and confirmed in larger multicenter studies.

4.2. Inflammatory Markers

Intermittent fasting produced significant reductions in ESR and CRP, whereas no significant changes occurred in the control group. Between-group differences in change from baseline were significant for both markers (both $P < 0.001$) (Table 2).

4.3. Non-Invasive Fibrosis Indices

Significant improvements were observed in all evaluated fibrosis-related indices (FIB-4, APRI, NAFLD Fibrosis Score, and BARD score) in the intermittent fasting group, whereas no significant changes occurred in the control group. Between-group comparisons favored intermittent fasting for all indices (all $P < 0.001$) (Table 2).

4.4. Ultrasonographic Findings

Improvement in hepatic steatosis was observed more frequently in the intermittent fasting group than in the control group, whereas worsening was less common. The distribution of ultrasonographic outcomes differed significantly between groups ($P < 0.001$).

No serious adverse events were reported, and all participants completed the intervention without treatment discontinuation.

5. Discussion

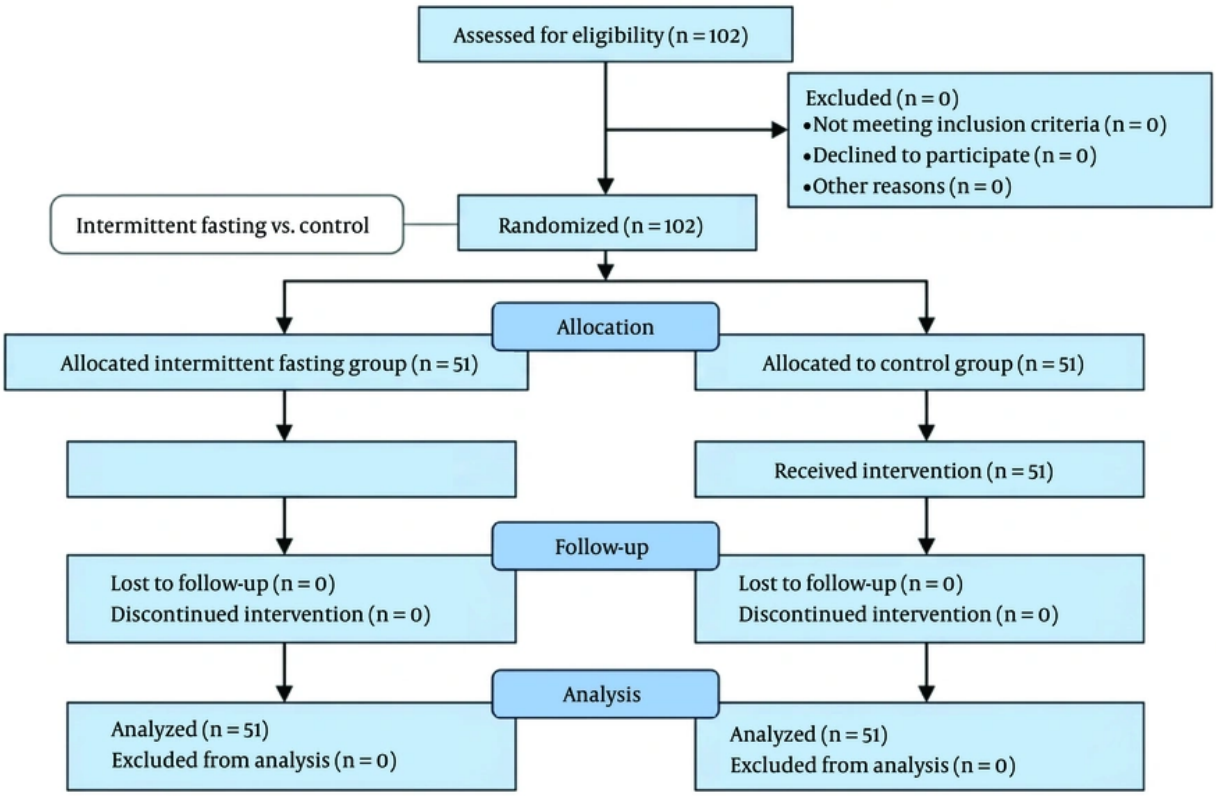

Figure 1. CONSORT 2010 flow diagram of participant enrollment, randomization, allocation, follow-up, and analysis.

The present randomized controlled trial demonstrated that a 12-week intermittent fasting intervention was associated with favorable changes in several clinically relevant outcomes among adults with NAFLD. Participants assigned to intermittent fasting experienced significant reductions in body weight, BMI, waist circumference, inflammatory markers, non-invasive fibrosis indices, and ultrasonographic steatosis severity compared with those receiving standard lifestyle advice.

One of the most notable findings was the substantial reduction in anthropometric parameters in the intervention group. These results are consistent with previous studies reporting beneficial effects of intermittent fasting on body weight, adiposity, and metabolic health. Reductions in visceral adiposity may be particularly relevant in NAFLD, as excess visceral fat contributes to insulin resistance, hepatic lipid accumulation, and chronic low-grade inflammation. The lack of comparable improvements in the control group suggests that a structured fasting regimen may be more effective than routine lifestyle recommendations alone.

Inflammatory activity also improved significantly during the intervention period. Marked reductions in ESR and CRP were observed among participants undergoing intermittent fasting. These findings are biologically plausible and may reflect improved insulin sensitivity, reduced oxidative stress, and favorable modulation of inflammatory signaling pathways, as described in previous experimental and clinical studies.

Another important observation was the significant improvement in all evaluated non-invasive fibrosis-related indices, including FIB-4, APRI, NAFLD Fibrosis Score, and BARD score. Because these scores are widely used for risk stratification and estimation of fibrosis probability in clinical practice, consistent improvement across multiple validated indices suggests a favorable effect of intermittent fasting on factors associated with disease severity and progression. However, these

Table 1. Baseline Demographic, Clinical, Laboratory, and Ultrasonographic Characteristics of Study Participants [a]

Variables	Control Group (n = 51)	Intermittent Fasting Group (n = 51)	P-Value
Age (y)	36 ± 8.5	35.5 ± 9.5	0.78
Sex			0.92
Male	28 (54.9)	27 (52.9)	
Female	23 (45.1)	24 (47.1)	
Weight (kg)	86.4 ± 5.4	88.4 ± 4.3	0.41
BMI (kg/m^2)	31.9 ± 3.2	31.6 ± 2.4	0.59
Waist circumference (cm)	104.8 ± 4.2	104.7 ± 3.7	0.89
AST (U/L)	51 ± 12	52 ± 13	0.68
ALT (U/L)	47 ± 10	49 ± 12	0.36
Platelet count (×10^9/L)	230 ± 70	261 ± 40	0.008
ESR (mm/h)	15 (8 - 23)	17 (9 - 25)	0.25
CRP (mg/L)	12 (5 - 13.4)	13.7 (9 - 18)	0.16
FIB-4 score	1.29 ± 0.43	1.31 ± 0.38	0.80
APRI score	0.93 ± 0.21	0.93 ± 0.37	0.93
NAFLD Fibrosis Score	−1.38 ± 0.56	−1.42 ± 0.46	0.69
BARD score	2.3 ± 0.8	2.3 ± 0.6	1
Steatosis Grade			0.46
Grade 1	34 (66.7)	38 (74.51)	
Grade 2	17 (33.3)	13 (25.49)	

[a] Values are expressed mean ± standard deviation, median (interquartile range [IQR]), or No. (%). Baseline comparisons were performed using the Mann-Whitney U test for continuous variables and the chi-square test or Fisher exact test for categorical variables, as appropriate. All baseline comparisons were performed before initiation of the intervention.

Table 2. Twelve-Week Changes in Clinical, Inflammatory, and Fibrosis-Related Variables [a]

Variables	Control (Pre)	Control (Post)	P-Value	Fasting (Pre)	Fasting (Post)	P-Value	Between-Group P
Weight (kg)	86.4 ± 5.4	86.09 ± 6.3	0.36	88.4 ± 4.3	75.4 ± 5.7	< 0.001	< 0.001
BMI (kg/m^2)	31.9 ± 3.2	31.7 ± 2.8	0.28	31.6 ± 2.4	27.8 ± 3.1	< 0.001	< 0.001
Waist (cm)	104.8 ± 4.2	104.7 ± 3.7	0.47	105.4 ± 3.6	94.8 ± 4.1	< 0.001	< 0.001
ESR (mm/h)	15 (8 - 23)	16 (10 - 27)	0.15	17 (9 - 25)	4 (2 - 12)	< 0.001	< 0.001
CRP (mg/L)	12 (5 - 13.4)	13.4 (6 - 18)	0.18	13.7 (9 - 18)	3 (1 - 12.3)	< 0.001	< 0.001
FIB-4	1.29 ± 0.43	1.28 ± 0.33	0.78	1.31 ± 0.38	0.76 ± 0.42	< 0.001	< 0.001
BARD score	2.3 ± 0.8	2.39 ± 0.7	0.90	2.3 ± 0.6	1.6 ± 0.8	< 0.001	< 0.001
APRI	0.93 ± 0.21	0.91 ± 0.19	0.58	0.93 ± 0.37	0.38 ± 0.14	< 0.001	< 0.001
NAFLD Fibrosis Score	−1.38 ± 0.56	−1.53 ± 0.53	0.07	−1.42 ± 0.46	−2.47 ± 0.72	< 0.001	< 0.001

[a] Values are expressed as mean ± SD or median (IQR). P values denote within-group comparisons. The between-group P value was calculated by comparing change-from-baseline (Δ) values between the two groups using the Mann-Whitney U test.

findings should be interpreted cautiously. All evaluated scores are indirect surrogate markers derived from clinical and laboratory variables and cannot establish true histological fibrosis regression. The observed improvements may partly reflect reductions in inflammation, body weight, and metabolic dysfunction rather than direct reversal of hepatic fibrosis. Therefore, these results should be interpreted as improvements in fibrosis-related surrogate markers rather than definitive evidence of fibrosis regression.

The ultrasonographic findings further support the beneficial metabolic effects observed in the intervention group. A substantially greater proportion of participants undergoing intermittent fasting demonstrated improvement in hepatic steatosis grade compared with controls. These observations are consistent with previous studies suggesting that dietary

interventions resulting in sustained negative energy balance may reduce hepatic fat accumulation and improve imaging-based markers of steatosis.

Our findings are generally consistent with recent randomized controlled trials and systematic reviews evaluating intermittent fasting in patients with NAFLD. In the randomized trial by Holmer et al. (11), intermittent calorie restriction significantly reduced hepatic steatosis and improved metabolic parameters, although effects on fibrosis-related markers were limited. Likewise, a recent systematic review and meta-analysis by Saleh et al. (14) demonstrated that intermittent fasting was associated with significant reductions in body weight, BMI, waist circumference, liver enzyme levels, and hepatic fat content in patients with NAFLD, while evidence regarding improvement in hepatic fibrosis remains limited because most available studies relied on surrogate fibrosis markers rather than histological assessment. These findings support the beneficial metabolic effects observed in our study while reinforcing the need for longer-term randomized trials incorporating liver biopsy or elastography to determine whether intermittent fasting can truly modify hepatic fibrosis.

Beyond statistical significance, the observed reductions in body weight, BMI, waist circumference, inflammatory markers, and fibrosis-related surrogate scores appear clinically meaningful. In particular, the substantial reduction in body weight and improvement in ultrasonographic steatosis grading suggest that the intervention may have practical relevance for routine management of patients with NAFLD.

5.1. Limitations

Several limitations should be acknowledged. First, liver biopsy was not performed, and histological assessment was not available; therefore, changes in non-invasive fibrosis scores cannot be interpreted as direct evidence of fibrosis regression. Second, dietary intake and caloric consumption were not formally quantified during the intervention period. Consequently, the relative contribution of fasting itself versus spontaneous caloric restriction cannot be determined. Third, physical activity was not systematically assessed and may have acted as a confounding factor. Fourth, adherence to the fasting protocol was primarily based on self-reported records and weekly follow-up contacts rather than objective monitoring methods. Fifth, the study was not prospectively registered in a clinical trial registry. Sixth, ultrasonographic assessments were performed by different radiologists at the same imaging center. Inter-observer variability was not formally assessed and may have influenced steatosis grading. Finally, the single-center design and relatively short follow-up period may limit the generalizability and long-term interpretation of the findings.

5.2. Strengths

Despite these limitations, the study has several important strengths. The randomized controlled design, balanced study groups, complete follow-up of all enrolled participants, and comprehensive assessment of anthropometric, inflammatory, biochemical, imaging, and fibrosis-related outcomes provide a broad evaluation of the potential effects of intermittent fasting in NAFLD. Furthermore, the concurrent use of multiple validated fibrosis-related indices enhances the robustness of the findings.

No serious adverse events were reported during the study period. Intermittent fasting was generally well tolerated, and no participant discontinued the intervention because of adverse effects.

5.3. Conclusions

In conclusion, intermittent fasting appears to be a feasible and potentially beneficial dietary intervention for adults with NAFLD. The intervention was associated with significant improvements in anthropometric measures, systemic inflammatory markers, ultrasonographic steatosis, and non-invasive fibrosis-related indices. Nevertheless, because fibrosis assessment was based exclusively on surrogate markers, definitive conclusions regarding histological fibrosis regression cannot be made. Larger multicenter randomized trials with longer follow-up periods, detailed dietary monitoring, assessment of physical activity, and direct histological or elastographic endpoints are warranted to clarify the long-term clinical significance of these findings.

Footnotes

AI Use Disclosure: The authors declare that no generative AI tools were used in the creation of this article.

Authors' Contribution: M. A. P., S. S., and V. M. conceived and designed the evaluation and drafted the manuscript; K. B. participated in designing the evaluation, performed part of the statistical analysis, and helped draft the manuscript; M. A. P. re-evaluated the clinical data, performed the statistical analysis, and revised the manuscript; N. F. collected, interpreted, and

reanalyzed the clinical and statistical data and revised the manuscript. All authors read and approved the final manuscript.

Conflict of Interests Statement: The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data Availability: The dataset presented in the study is available on request from the corresponding author during submission or after publication.

Ethical Approval: The study protocol was approved by the Ethics Committee of AJA University of Medical Sciences, Tehran, Iran (IR.AJAUMS.REC.1404.02). Written informed consent was obtained from all participants prior to enrollment

Funding/Support: The authors received no external funding for this study.

Informed Consent: Written informed consent was obtained from all participants before enrollment.

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