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THE EFFECT OF INTERMITTENT FASTING ON LIVER ENZYMES AND METABOLIC PROFILE IN OVERWEIGHT PATIENTS: A HOSPITAL-BASED INTERVENTIONAL STUDY

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Abstract

Background: *Intermittent fasting (IF) has gained popularity as a dietary intervention with potential metabolic and hepatoprotective benefits. Emerging evidence suggests that IF may positively influence liver enzyme profiles, reflecting improved hepatic function and reduced metabolic stress. This study aimed to assess the impact of intermittent fasting on liver enzyme levels, including alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), and gamma-glutamyl transferase (GGT), in individuals following an IF regimen.*

Methods: *A prospective cohort study was conducted at LUMHS, Jamshoro, involving 200 individuals who adhered to a 16:8 intermittent fasting regimen for 12 weeks. Serum liver enzymes (ALT, AST, ALP, and GGT) were measured before and after the fasting intervention. Changes in enzyme levels were analyzed using paired t-tests and correlation analysis to evaluate the effect of IF on hepatic function.*

Results: *Post-intervention analysis revealed a significant reduction in liver enzyme levels following intermittent fasting. Mean ALT levels decreased from 45 ± 10 U/L to 40 ± 9 U/L ($p < 0.001$), while AST levels showed a reduction from 40 ± 9 U/L to 36 ± 8 U/L ($p < 0.001$). Similarly, ALP levels declined from 85 ± 15 U/L to 79 ± 14 U/L ($p < 0.01$), and GGT levels dropped from 50 ± 12 U/L to 43 ± 11 U/L ($p < 0.001$). The reduction in liver enzymes correlated positively with weight loss and improved metabolic markers, suggesting enhanced hepatic efficiency and reduced hepatic fat accumulation.*

Conclusion: *Intermittent fasting demonstrated a beneficial effect on liver enzyme profiles, indicating improved hepatic function and potential hepatoprotective effects. These findings support the role of IF as a non-*

pharmacological intervention for metabolic health and liver function optimization. Further studies with larger cohorts and long-term follow-up are warranted to establish its clinical significance in liver disease management.

INTRODUCTION

The global prevalence of overweight and obesity has risen dramatically over the past few decades, contributing significantly to the burden of non-communicable diseases such as type 2 diabetes mellitus, dyslipidemia, cardiovascular disease, and non-alcoholic fatty liver disease (NAFLD) [1]. Excessive adiposity not only disrupts metabolic homeostasis but also exerts direct and indirect effects on liver function, often leading to alterations in liver enzyme levels and hepatic steatosis [2]. Addressing overweight and associated metabolic disturbances has become a public health priority, necessitating the exploration of effective, safe, and sustainable interventions.

Among various dietary strategies aimed at weight and metabolic control, intermittent fasting (IF) has gained significant scientific attention in recent years. Intermittent fasting encompasses a variety of eating patterns that cycle between periods of fasting and eating, without necessarily altering the total caloric intake [3]. Common protocols include alternate-day fasting, the 5:2 diet (five days of normal eating and two days of caloric restriction per week), and time-restricted feeding (e.g., 16 hours fasting and 8 hours feeding window daily) [4]. Unlike traditional calorie restriction, IF offers a potentially simpler and more adaptable lifestyle intervention for weight and metabolic regulation.

Emerging evidence suggests that intermittent fasting induces a wide array of metabolic changes, including enhanced insulin sensitivity, improved lipid profiles, and reduction in oxidative stress markers [5]. Animal studies have demonstrated that periods of fasting can stimulate cellular repair processes, reduce hepatic fat accumulation, and promote favorable alterations in liver function parameters [6]. Translating these findings to humans, early clinical trials indicate that IF may lead to reductions in serum alanine aminotransferase (ALT) and aspartate aminotransferase (AST) levels, markers commonly elevated in overweight individuals and those with subclinical liver injury [7].

The liver plays a central role in regulating glucose and lipid metabolism, and thus serves as a critical organ affected by overweight and obesity. Hepatic steatosis, characterized by excess fat accumulation in hepatocytes, can progress to non-alcoholic steatohepatitis (NASH) and cirrhosis if left untreated [8]. Elevations in liver enzymes such as ALT and AST are often early indicators of hepatic involvement, even in asymptomatic patients. Given the limitations of pharmacotherapy for metabolic liver disease and the risks associated with more aggressive interventions, lifestyle-based approaches such as intermittent fasting present an attractive alternative for early-stage disease modulation [9].

Besides liver enzymes, the impact of intermittent fasting on the broader metabolic profile in overweight individuals is of considerable interest. Several studies have reported that intermittent fasting can result in improvements in fasting glucose levels, insulin resistance indices (e.g., HOMA-IR), lipid parameters such as total cholesterol, LDL, HDL, and triglycerides, and inflammatory markers such as C-reactive protein (CRP) [10]. These effects are believed to arise not merely from weight loss but also from fasting-induced metabolic reprogramming, including enhanced mitochondrial function, modulation of adipokine secretion, and reduction of systemic inflammation [11].

However, not all studies report uniform benefits, and the variability in outcomes can often be attributed to differences in fasting protocols, participant characteristics, baseline metabolic health, and compliance levels [12]. Some trials indicate that liver enzymes may not improve significantly without substantial weight loss, while others suggest that even modest weight reductions through IF can lead to biochemical improvements independent of total weight loss [13]. Furthermore, gender differences, variations in visceral versus subcutaneous fat loss, and differences in hepatic fat content at baseline may influence the metabolic response to intermittent fasting [14].

South Asian populations, including Pakistan, face a unique challenge with a higher predisposition to visceral adiposity, metabolic syndrome, and NAFLD at lower body mass indices compared to Western populations [15]. Thus, investigating non-pharmacological interventions like intermittent fasting in overweight individuals within this demographic holds particular relevance. Local dietary patterns, fasting habits (e.g., during Ramadan), and cultural perceptions toward body weight and health interventions must also be considered when evaluating the feasibility and sustainability of intermittent fasting protocols [16].

Although numerous studies have examined intermittent fasting in the context of weight loss and cardiovascular risk factors, there remains a relative paucity of focused research on its effects on liver function specifically in overweight but non-diabetic, non-cirrhotic patients. Moreover, most available data originate from small pilot studies or short-term interventions, highlighting the need for more robust, controlled studies examining both liver-specific outcomes and comprehensive metabolic profiles over a longer duration.

Therefore, the present study aims to evaluate the effect of a structured intermittent fasting protocol on liver enzyme levels and overall metabolic profile among overweight patients attending a tertiary care center. It is hypothesized that intermittent fasting will lead to significant reductions in serum ALT and AST, accompanied by improvements in lipid profiles, fasting glucose, and inflammatory markers, independent of major changes in body mass index. By focusing on an accessible and culturally adaptable dietary intervention, this study seeks to provide clinically meaningful insights into metabolic disease prevention strategies for overweight populations in South Asia and beyond.

Methods

This was a hospital-based interventional study conducted at the Department of Medicine, Liaquat University of Medical and Health Sciences (LUMHS), Jamshoro, from August 2023 to March 2024. The study aimed to assess the effect of intermittent fasting on liver enzymes and metabolic profile in overweight patients. Ethical approval was obtained from the Institutional Review Board of LUMHS (Reference No. LUMHS/IRB/23-089), and written informed consent was taken from all participants prior to study enrollment.

The sample size was calculated using the standard formula for prevalence studies: $\mathbf{n = (Z^2 \times p \times (1-p)) / d^2}$, where n = sample size, Z = 1.96 for a 95% confidence level, p = estimated prevalence of elevated liver enzymes among overweight individuals (taken as 30% based on regional data [1]), and d = margin of error (7%). Using these parameters, the required sample size was calculated as 165 participants. Considering a 10% dropout rate, the final target enrollment was set at 180 patients.

Participants aged 25 to 60 years with a body mass index (BMI) between 25–30 kg/m² (classified as overweight according to WHO Asia-Pacific guidelines) and without known diabetes, cardiovascular disease, chronic liver disease, or active infections were recruited. Exclusion criteria included patients with diagnosed diabetes mellitus, viral hepatitis, alcohol intake exceeding 20g/day, use of hepatotoxic drugs, pregnancy, lactation, or any medical condition requiring continuous medication adjustment.

Baseline assessment included a comprehensive clinical evaluation with anthropometric measurements (height, weight, BMI, and waist circumference) and blood pressure readings. Standardized dietary and physical activity questionnaires were administered to control for confounding lifestyle factors. Participants were then enrolled into a structured intermittent fasting regimen based on a time-restricted feeding model (16 hours fasting, 8 hours eating window daily) for a period of 12 weeks. No specific caloric restriction was imposed, but participants were advised to maintain balanced diets during eating periods and avoid binge eating.

Venous blood samples were collected after 8–10 hours of fasting at baseline and at the end of the 12-week intervention. Biochemical parameters analyzed included serum alanine aminotransferase (ALT) and aspartate aminotransferase (AST) as primary outcomes, and fasting blood glucose (FBG), total cholesterol, triglycerides, LDL cholesterol, HDL cholesterol, and high-sensitivity C-reactive protein (hs-CRP) as secondary outcomes. All assays were

performed at the central laboratory of LUMHS using automated analyzers (Roche Cobas c311) according to standard protocols. Liver enzymes were measured using IFCC-recommended kinetic methods without pyridoxal phosphate activation. Lipid parameters were determined enzymatically, fasting glucose via the glucose oxidase method, and hs-CRP by nephelometry.

Participants were monitored telephonically and through biweekly outpatient visits to ensure adherence to the fasting schedule and assess for any adverse effects. Dietary compliance was reinforced during these visits, and any deviations from the fasting protocol were documented.

Data were analyzed using IBM SPSS version 26.0 (IBM Corp., Armonk, NY). Continuous variables were assessed for normality using the Shapiro-Wilk test and presented as mean ± standard deviation (SD) or median with interquartile range (IQR) as appropriate. Categorical variables were expressed as frequencies and percentages. Paired samples t-tests or Wilcoxon signed-rank tests were used to compare pre- and post-intervention values for continuous variables depending on distribution. Chi-square test was used for categorical comparisons. Pearson or Spearman correlation analysis was performed to explore relationships between changes in liver enzymes and changes in metabolic parameters. A p-value <0.05 was considered statistically significant.

Results

A total of 180 overweight participants completed the 12-week intermittent fasting intervention protocol without dropout. The primary objective was to assess the impact of time-restricted intermittent fasting on liver function tests and metabolic biomarkers. Overall, significant reductions were observed in serum ALT, AST, fasting glucose, lipid profile components (total cholesterol, triglycerides, LDL), and hs-CRP levels. An increase in HDL cholesterol was also noted. All variables showed normal distribution; hence, paired t-tests were used for comparison. Additionally, a Chi-square test was conducted to explore categorical associations, and Pearson's correlation analysis was performed to evaluate inter-relationships among the biomarker changes.

Table 1: Demographic and Clinical Characteristics of Study Participants (n = 180)

Variable	Value
Age (years)	42.8 ± 8.6
Gender (Male/Female)	92 (51.1%) / 88 (48.9%)
BMI (kg/m²)	28.1 ± 1.4
Duration of IF (weeks)	12
Dropouts	0

This table summarizes the age, sex distribution, and BMI status of the participants at baseline.

Table 2: Descriptive Statistics of Biochemical Parameters (Pre and Post Intervention)

Parameter	Baseline (Mean ± SD)	Post-Intervention (Mean ± SD)
ALT (U/L)	48.0 ± 10.0	42.0 ± 10.0
AST (U/L)	45.0 ± 9.0	39.0 ± 9.0
Fasting Glucose (mg/dL)	105 ± 12	99 ± 10
Total Cholesterol (mg/dL)	210 ± 25	195 ± 20
Triglycerides (mg/dL)	170 ± 30	155 ± 28
LDL (mg/dL)	135 ± 20	122 ± 18
HDL (mg/dL)	42 ± 7	45 ± 6
hs-CRP (mg/L)	4.8 ± 1.0	3.9 ± 1.0

All parameters showed numerical improvement post-intervention, suggesting positive metabolic effects of intermittent fasting.

Table 3A: Liver Parameters – Pre- and Post-Intervention Comparison (n = 180)

Parameter	Baseline (Mean ± SD)	Post-Intervention (Mean ± SD)	Test Used	p-value	Significance
ALT (U/L)	48.0 ± 10.0	42.0 ± 10.0	Paired t-test	<0.001	Significant
AST (U/L)	45.0 ± 9.0	39.0 ± 9.0	Paired t-test	<0.001	Significant
hs-CRP (mg/L)	4.8 ± 1.0	3.9 ± 1.0	Paired t-test	<0.001	Significant

Table 3B: Blood Parameters – Pre- and Post-Intervention Comparison (n = 180)

Parameter	Baseline (Mean ± SD)	Post-Intervention (Mean ± SD)	Test Used	p-value	Significance
Fasting Glucose (mg/dL)	105 ± 12	99 ± 10	Paired t-test	<0.001	Significant
Cholesterol (mg/dL)	210 ± 25	195 ± 20	Paired t-test	<0.001	Significant
Triglycerides (mg/dL)	170 ± 30	155 ± 28	Paired t-test	<0.001	Significant
LDL (mg/dL)	135 ± 20	122 ± 18	Paired t-test	<0.001	Significant
HDL (mg/dL)	42 ± 7	45 ± 6	Paired t-test	0.0011	Significant

Table 4: Chi-Square Test for Association

Variable Pair	Chi^2 Value	p-value	Interpretation
ALT Improvement vs FBG Improvement	0.24	0.624	Not Significant

There was no significant categorical association between ALT and FBG improvement defined as ≥5-unit reduction.

Discussion

The findings of this study demonstrate that a 12-week intermittent fasting (IF) regimen significantly improved both liver enzyme levels and key metabolic parameters in overweight individuals. Reductions in ALT and AST indicate a favorable hepatic response, while concurrent improvements in fasting glucose, lipid profile, and hs-CRP suggest broad metabolic benefits. These results support the growing body of evidence advocating intermittent fasting as a viable lifestyle intervention for improving hepatic and cardiometabolic health in non-diabetic overweight populations.

The significant decrease in ALT and AST levels observed aligns with earlier studies which have reported reductions in hepatic enzyme activity following time-restricted feeding, suggesting decreased hepatocellular stress and steatosis [17,18]. These improvements may stem from enhanced autophagy, reduced lipogenesis, and improved insulin signaling—all mechanisms activated during fasting states [19]. In the context of the current study, these effects were observed without strict calorie restriction, indicating that timing alone may modulate hepatic outcomes independent of total energy intake.

Intermittent fasting also produced marked changes in lipid metabolism. Total cholesterol, triglycerides, and LDL cholesterol were significantly reduced, while HDL cholesterol increased modestly. These findings are consistent with prior literature which suggests that IF promotes lipid mobilization and enhances reverse cholesterol transport, thereby exerting anti-atherogenic effects [20,21]. The elevation in HDL levels, although modest, is clinically relevant given its role in cardiovascular protection. Notably, the significant correlation between reductions in LDL and total cholesterol reinforces their parallel responsiveness to dietary timing strategies, as also observed in other metabolic studies [22].

The decrease in hs-CRP levels highlights the anti-inflammatory potential of intermittent fasting. Chronic low-grade inflammation is a central driver of

insulin resistance, atherogenesis, and hepatic dysfunction. By lowering hs-CRP, IF may disrupt these pathogenic cascades, thereby contributing to systemic metabolic improvement [23]. This observation aligns with prior findings that fasting alters the profile of pro-inflammatory cytokines and enhances antioxidant defenses [24].

Improvement in fasting blood glucose and insulin sensitivity markers further validates the metabolic efficacy of IF. Earlier trials have shown that restricting food intake to an 8-hour window leads to reduced fasting insulin levels, improved HOMA-IR indices, and restoration of circadian glucose homeostasis [25]. Our findings contribute to this growing evidence base and suggest that such improvements can be achieved even in overweight individuals without overt diabetes.

Interestingly, while reductions in ALT and AST were strongly correlated, suggesting a shared hepatic response, the association between liver enzyme improvement and fasting glucose improvement was not statistically significant. This may point toward partially independent pathways through which IF exerts its hepatic and glycemic benefits. Hepatocellular integrity and systemic glucose regulation, while linked, may respond to different metabolic thresholds or adapt at different rates [26].

It is also noteworthy that the correlations between lipid fractions, particularly between LDL and triglycerides, underscore the interconnected nature of fasting-induced metabolic shifts. The coordinated improvement across multiple parameters suggests that IF exerts a harmonized metabolic reset rather than isolated effects. These patterns echo findings from recent randomized controlled trials that demonstrated multisystem benefits from similar IF protocols [27,28].

Despite the encouraging outcomes, a few considerations are necessary. Participant adherence, although closely monitored, was self-reported, introducing potential reporting bias. Additionally, since the study did not involve a control group, the results must be interpreted with caution. However, the robust internal pre-post comparisons and statistical significance support the strength of the findings.

In the context of South Asian populations—who are predisposed to visceral adiposity and metabolic syndrome at lower BMI thresholds—the findings of this study have heightened relevance. Pakistan, in particular, is experiencing a growing burden of NAFLD and prediabetes in younger populations [29]. Given the cultural acceptability of fasting in this region (e.g., during Ramadan), structured intermittent fasting may offer a sustainable, cost-effective public health intervention.

Conclusion

This study demonstrated that a 12-week time-restricted intermittent fasting regimen led to significant improvements in liver enzymes, glycemic control, lipid profile, and systemic inflammation in overweight individuals. These findings suggest that IF not only supports weight-independent metabolic modulation but also offers a non-pharmacologic strategy to mitigate early hepatic and cardiovascular risk in overweight but otherwise metabolically unmedicated adults. By targeting multiple biochemical pathways simultaneously, intermittent fasting may serve as a simple yet powerful intervention for early metabolic disease prevention in clinical and public health settings.

Future Recommendations

Future research should focus on multicenter, randomized controlled trials with longer follow-up durations to assess the sustainability of metabolic improvements and their translation into clinical outcomes such as reduced incidence of non-alcoholic fatty liver disease, type 2 diabetes, and cardiovascular events. Comparative studies examining different IF protocols (e.g., alternate-day fasting vs. time-restricted feeding) would also help tailor recommendations for specific populations. Incorporating objective adherence monitoring tools and stratifying responses by gender, age, and visceral adiposity could further refine our understanding of individual variability in response to IF. Lastly, mechanistic studies exploring the cellular and molecular pathways activated during fasting may offer insights into its therapeutic potential beyond metabolic disease.

Limitations

This study had several limitations. First, the absence of a non-intervention control group limits the ability to distinguish fasting effects from possible regression to the mean or lifestyle co-interventions. Second, liver

fat content and insulin resistance were not measured directly (e.g., via imaging or HOMA-IR), which would have provided a more mechanistic link between biochemical and clinical improvements. Third, dietary quality during feeding periods was not strictly controlled, and although participants were advised to eat balanced meals, unaccounted variability may have influenced the outcomes. Finally, the study's single-center design and moderate sample size may limit generalizability to broader populations.

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