JHWCR JOURNAL OF HEALTH, WELLNESS AND COMMUNITY RESEARCH 3007-0570

https://doi.org/10.61919/skrctj06

Original Article

Impact of Timed Carbohydrate Restriction on Liver Fat Regression in Non-Diabetic Adults With Obesity

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"Cite this Article" Received: 03 February 2026; **Accepted**: 10 March 2026; **Published**: 14 March 2026

Author Contributions: Concept: AK; Design: AS, LAN, AK; Data Collection: AS, LAN, ASe, ASa, SRA, ASh; Analysis: AK; Drafting: AS, LAN, ASe, ASa, SRA, ASh, AK.. **Ethical Approval**: Vertex Institute of Science and Technology, Mardan, Pakistan. **Informed Consent**: Written informed consent was obtained from all participants; **Conflict of Interest**: The authors declare no conflict of interest. **Funding**: No external funding; **Data Availability**: Available from the corresponding author on reasonable request; **Acknowledgments**: NA

ABSTRACT

Background: Hepatic steatosis is a common obesity-associated metabolic condition that may occur in non-diabetic adults and contribute to progressive hepatic and cardiometabolic risk. Dietary modification is central to management, but the effect of restricting carbohydrate intake to an earlier daily time window remains insufficiently defined. Objective: To evaluate whether timed carbohydrate restriction improves hepatic steatosis severity and metabolic indicators in non-diabetic adults with obesity. Methods: A parallel-group randomized controlled trial was conducted in Southern Punjab, Pakistan, from August 2025 to January 2026. Eighty non-diabetic adults aged 25–60 years with obesity and ultrasonography-confirmed hepatic steatosis were randomized to timed carbohydrate restriction or standard dietary advice. The intervention group consumed carbohydrate-containing foods only between 8:00 a.m. and 4:00 p.m. for 12 weeks, while the control group received conventional weight-management advice without carbohydrate-timing restriction. The primary outcome was change in ultrasound-graded hepatic steatosis severity. Secondary outcomes included body mass index, waist circumference, fasting insulin, HOMA-IR, and alanine aminotransferase. Results: Seventy-three participants completed follow-up assessment. Hepatic steatosis score decreased from 2.73 ± 0.61 to 1.38 ± 0.55 in the intervention group and from 2.69 ± 0.59 to 2.01 ± 0.63 in the control group. The between-group difference in mean change was -0.67, and the time-by-group interaction was significant ($F=24.58$, $p<0.001$). At 12 weeks, the intervention group also had lower BMI, waist circumference, fasting insulin, HOMA-IR, and alanine aminotransferase. Conclusion: Timed carbohydrate restriction was associated with greater 12-week improvement in hepatic steatosis and metabolic indicators among non-diabetic adults with obesity. Keywords: Carbohydrates; Chrononutrition; Diet Therapy; Hepatic Steatosis; Insulin Resistance; Obesity; Randomized Controlled Trial

INTRODUCTION

Obesity is a major public health problem that contributes substantially to the global burden of metabolic disease and increases the risk of hepatic lipid accumulation, insulin resistance, cardiovascular morbidity, and progressive liver-related complications. Among adults with excess adiposity, hepatic steatosis represents an important early manifestation of metabolic dysfunction because triglyceride deposition within hepatocytes may occur even in the absence of overt diabetes. Non-diabetic adults with obesity therefore constitute a clinically important risk group in whom liver fat accumulation may remain underrecognized until metabolic or hepatic abnormalities become more advanced. Lifestyle

modification remains the first-line strategy for improving obesity-associated hepatic steatosis, yet conventional approaches based primarily on caloric restriction and weight reduction are frequently limited by poor long-term adherence and variable metabolic response (1).

Dietary composition has a direct influence on hepatic fat metabolism because carbohydrate intake affects postprandial glucose exposure, insulin secretion, hepatic de novo lipogenesis, and substrate oxidation. Diets that reduce excessive carbohydrate exposure, particularly from refined or rapidly absorbable sources, have been associated with improvements in insulin sensitivity, body composition, and hepatic lipid accumulation. However, strict carbohydrate-restricted dietary regimens may be difficult to maintain in routine clinical practice, especially in populations where carbohydrate-rich foods form a central component of daily dietary patterns. This has encouraged interest in more feasible nutritional strategies that modify not only the quantity and quality of carbohydrate intake but also the timing of carbohydrate consumption (2).

Chrononutrition provides a biologically plausible framework for evaluating carbohydrate timing because glucose tolerance, insulin responsiveness, lipid oxidation, gastrointestinal hormone secretion, and hepatic metabolic processes vary across the circadian cycle. Earlier daytime food intake may occur during periods of relatively greater insulin sensitivity and more efficient substrate handling, whereas late-day carbohydrate exposure may contribute to impaired glucose regulation and greater lipid storage. Time-restricted eating and meal-timing interventions have shown favorable effects on metabolic health in several populations, but their direct application to hepatic steatosis regression remains insufficiently defined. In particular, it is unclear whether restricting carbohydrate-containing foods to an earlier daily window can produce measurable improvement in liver fat severity among adults with obesity who do not have diabetes (3).

Existing literature has largely focused on weight loss, total caloric restriction, ketogenic or low-carbohydrate diets, glycemic control, or broad time-restricted eating protocols rather than the specific effect of timed carbohydrate restriction on hepatic steatosis. Many prior investigations have also included participants with diabetes, metabolic syndrome, or heterogeneous cardiometabolic profiles, making it difficult to isolate the clinical relevance of carbohydrate timing in non-diabetic adults with obesity. This distinction is important because non-diabetic individuals with obesity may still have insulin resistance, central adiposity, elevated liver enzymes, and ultrasound-confirmed hepatic steatosis despite not meeting diagnostic criteria for diabetes. A practical intervention targeting the timing of carbohydrate intake may therefore offer an accessible strategy for improving liver-related and metabolic outcomes in this population (4).

The present parallel-group randomized controlled trial was designed to evaluate whether restricting carbohydrate-containing foods to an earlier daily window from 8:00 a.m. to 4:00 p.m. improves hepatic steatosis severity compared with standard dietary advice in non-diabetic adults with obesity and ultrasonography-confirmed hepatic steatosis. The study specifically compared changes in hepatic steatosis score, body mass index, waist circumference, fasting insulin, insulin resistance, fasting glucose, and alanine aminotransferase over a 12-week intervention period. It was hypothesized that adults receiving timed carbohydrate restriction would demonstrate greater regression of ultrasound-graded hepatic steatosis and greater improvement in metabolic indicators than adults receiving conventional dietary advice alone (5).

MATERIALS AND METHODS

A parallel-group randomized controlled trial was conducted in Southern Punjab, Pakistan, from August 2025 to January 2026 to evaluate the effect of timed carbohydrate restriction on hepatic steatosis regression among non-diabetic adults with obesity. The total study duration was six months and included participant screening, recruitment, baseline assessment, randomization, intervention delivery, adherence monitoring, and post-intervention outcome assessment. The active intervention period lasted 12 weeks.

A randomized controlled design was selected to compare the effect of an earlier daily carbohydrate intake window with standard dietary advice while minimizing selection bias and improving causal interpretability of the intervention effect.

Adults aged 25–60 years were eligible if they had obesity, defined as body mass index of at least 30 kg/m², and hepatic steatosis confirmed by abdominal ultrasonography at screening. Participants were required to be non-diabetic, clinically stable, weight-stable during the preceding three months, and willing to follow dietary instructions and attend follow-up assessments. Individuals were excluded if they had diagnosed diabetes mellitus, significant alcohol intake, viral hepatitis, known chronic liver disease of another etiology, pregnancy, lactation, previous bariatric surgery, current use of weight-loss medication, severe cardiovascular disease, or any medical, occupational, or behavioral condition likely to interfere with dietary adherence or follow-up completion. Eligible participants were recruited through clinical screening and community referral pathways, and written informed consent was obtained before enrollment.

The sample size was estimated using effect assumptions derived from previous dietary intervention studies evaluating liver fat or hepatic steatosis-related outcomes in adults with obesity. A minimum sample of 72 participants was considered sufficient to detect a clinically meaningful between-group difference with 80% statistical power at a 5% significance level. To compensate for expected attrition, 80 participants were enrolled and randomly allocated in a 1:1 ratio to the timed carbohydrate restriction group or the control group, with 40 participants assigned to each arm. Randomization was performed using a computer-generated random sequence prepared by an independent investigator not involved in recruitment, intervention delivery, or outcome assessment. Allocation concealment was maintained using sequentially numbered, sealed, opaque envelopes that were opened only after baseline assessment and participant enrollment.

Participants assigned to the timed carbohydrate restriction group received individualized dietary counseling instructing them to consume all carbohydrate-containing foods exclusively between 8:00 a.m. and 4:00 p.m. for 12 weeks. Carbohydrate-containing foods included bread, rice, wheat products, cereals, potatoes, sweetened foods, fruit juices, desserts, sugar-containing beverages, and other starch- or sugar-dominant items commonly consumed in the local diet. Outside the 8-hour carbohydrate window, participants were advised to avoid carbohydrate-containing foods and were permitted to consume water, unsweetened beverages, protein-based foods, and non-starchy vegetables. The intervention was designed as a timing-focused dietary strategy rather than a severe carbohydrate-elimination protocol, with counseling emphasizing meal planning, culturally familiar food substitutions, avoidance of late-day carbohydrate intake, and consistency across weekdays and weekends.

Participants in the control group received standard dietary advice for weight management based on balanced caloric restriction, portion control, reduced intake of fried and sugar-rich foods, and encouragement of regular meal patterns without restriction on the timing of carbohydrate intake. Both groups received dietary counseling at baseline and every two weeks during the intervention period to maintain comparable professional contact and to reduce differential attention bias. Participants in both groups were encouraged to maintain their usual physical activity pattern during the trial so that observed changes could be more directly interpreted in relation to the dietary intervention. Adherence was monitored using weekly dietary logs and telephone follow-up. Dietary logs recorded the timing of carbohydrate-containing meals and snacks, major food categories consumed, deviations from assigned instructions, and missed follow-up contacts.

The primary outcome was change in hepatic steatosis severity from baseline to 12 weeks. Hepatic steatosis was assessed using standardized abdominal ultrasonography performed at baseline and after completion of the intervention. Steatosis severity was graded using an ordinal ultrasound-based scoring approach based on hepatic echogenicity, visualization of intrahepatic vessels, posterior beam attenuation, and liver–kidney contrast. Higher scores indicated greater steatosis severity. Ultrasound assessment was

performed by trained assessors who remained blinded to group allocation, and the same standardized grading approach was applied at both assessment points to reduce measurement bias. Secondary outcomes included body weight, body mass index, waist circumference, fasting glucose, fasting insulin, homeostatic model assessment of insulin resistance, and serum alanine aminotransferase concentration. Anthropometric and biochemical measurements were obtained at baseline and at 12 weeks using the same measurement procedures across groups.

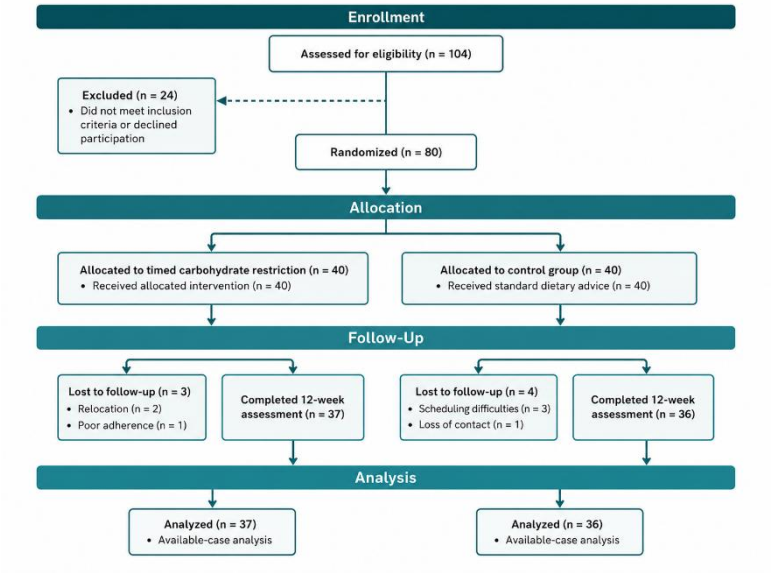

Figure 1 CONSORT Flowchart

Several steps were used to reduce bias and improve data integrity. Allocation concealment was maintained until after enrollment, outcome assessors and data analysts were blinded to group assignment, and both groups received repeated counseling contacts during follow-up. Standardized data collection forms were used for demographic, clinical, anthropometric, biochemical, dietary adherence, and outcome variables. Data were checked for completeness and internal consistency before analysis. Missing follow-up data were handled according to the prespecified analytic approach; because post-intervention outcome comparisons were based on participants with completed 12-week assessments, the analysis was treated as an available-case analysis rather than a full intention-to-treat analysis. Baseline characteristics were reported for the full randomized cohort, while post-intervention outcomes were reported for participants with completed follow-up assessments.

Data were analyzed using SPSS version 26. Continuous variables were summarized as mean ± standard deviation after assessment of distributional assumptions using the Shapiro–Wilk test, while categorical variables were summarized as frequencies and percentages. Baseline comparability between groups was assessed using independent-samples t-tests for continuous variables and chi-square or Fisher's exact tests for categorical variables, as appropriate. Within-group pre–post changes were evaluated using paired-samples t-tests. Between-group comparisons at 12 weeks were performed using independent-samples t-tests, and repeated measures analysis of variance was used to evaluate time, group, and time-by-group interaction effects for hepatic steatosis and other repeated continuous outcomes. Pearson correlation analysis was used to examine associations between change in hepatic steatosis severity and changes in metabolic or anthropometric parameters. Statistical significance was set at $p<0.05$.

RESULTS

During the six-month study period from August 2025 to January 2026, 104 individuals were screened for eligibility. Of these, 24 were excluded because they either did not meet the eligibility criteria or declined participation. Eighty eligible participants were randomized in a 1:1 ratio, with 40 allocated to the timed carbohydrate restriction group and 40 allocated to the control group. During the 12-week

intervention period, three participants in the timed carbohydrate restriction group were lost to follow-up, including two due to relocation and one due to poor adherence. Four participants in the control group withdrew, including three due to scheduling difficulties and one due to loss of contact. Final 12-week outcome assessment was completed by 73 participants, including 37 in the timed carbohydrate restriction group and 36 in the control group. Baseline characteristics were summarized for the full randomized cohort, whereas post-intervention outcome analyses were based on participants with completed follow-up assessments.

Table 1. Participant Flow During the Trial

Trial Stage	Total, n	Timed Carbohydrate Restriction, n	Control, n
Screened for eligibility	104	—	—
Excluded before randomization	24	—	—
Randomized	80	40	40
Lost to follow-up	7	3	4
Completed 12-week assessment	73	37	36

Table 1 footnote: Dash indicates not applicable at the screening or pre-randomization stage. Of the 104 individuals screened, 80 were randomized and 73 completed the 12-week follow-up assessment. Follow-up completion was 92.5% in the timed carbohydrate restriction group and 90.0% in the control group.

Table 2. Baseline Demographic, Anthropometric, Metabolic, and Hepatic Characteristics of Randomized Participants

Variable	Total Sample (N=80), Mean ± SD or n (%)	Timed Carbohydrate Restriction (n=40), Mean ± SD or n (%)	Control (n=40), Mean ± SD or n (%)	p-value
Age, years	43.8 ± 8.1	44.1 ± 8.4	43.5 ± 7.9	0.742
Male sex, n (%)	42 (52.5)	21 (52.5)	21 (52.5)	1.000
BMI, kg/m²	33.4 ± 2.8	33.6 ± 2.9	33.2 ± 2.7	0.538
Waist circumference, cm	108.1 ± 8.5	108.7 ± 8.2	107.5 ± 8.9	0.546
Fasting glucose, mg/dL	95.3 ± 7.4	95.7 ± 7.1	94.9 ± 7.8	0.641
HOMA-IR	3.84 ± 0.88	3.89 ± 0.91	3.79 ± 0.85	0.617
ALT, U/L	49.6 ± 11.2	50.1 ± 10.9	49.0 ± 11.6	0.664
Hepatic steatosis score	—	2.73 ± 0.61	2.69 ± 0.59	—

Table 2 footnote: BMI, body mass index; HOMA-IR, homeostatic model assessment of insulin resistance; ALT, alanine aminotransferase; SD, standard deviation. Baseline hepatic steatosis values were available for participants included in the final outcome analysis rather than the full randomized cohort. The randomized groups were comparable at baseline for age, sex distribution, body mass index, waist circumference, fasting glucose, HOMA-IR, and alanine aminotransferase. Baseline hepatic steatosis scores among participants with completed outcome assessment were also closely similar between the timed carbohydrate restriction group and the control group.

Table 3. Post-Intervention Comparison of Hepatic Steatosis Severity at 12 Weeks

Outcome	Timed Carbohydrate Restriction (n=37), Mean ± SD	Control (n=36), Mean ± SD	Mean Difference	95% CI	p-value
Hepatic steatosis score	1.38 ± 0.55	2.01 ± 0.63	-0.63	-0.90 to -0.36	<0.001

Table 3 footnote: CI, confidence interval; SD, standard deviation. Mean difference was calculated as timed carbohydrate restriction minus control. At 12 weeks, the timed carbohydrate restriction group had a lower hepatic steatosis score than the control group. The between-group mean difference was -0.63, with a 95% confidence interval from -0.90 to -0.36.

Table 4. Within-Group Change in Hepatic Steatosis Severity From Baseline to 12 Weeks

Group	n	Baseline, Mean ± SD	Week 12, Mean ± SD	Mean Change ± SD	p-value
Timed carbohydrate restriction	37	2.73 ± 0.61	1.38 ± 0.55	-1.35 ± 0.52	<0.001
Control	36	2.69 ± 0.59	2.01 ± 0.63	-0.68 ± 0.49	<0.001

Table 4 footnote: SD, standard deviation. Mean change was calculated as week 12 minus baseline. Hepatic steatosis scores decreased in both groups over the 12-week intervention period. The reduction was numerically larger in the timed carbohydrate restriction group, where the mean hepatic steatosis score

decreased from 2.73 ± 0.61 to 1.38 ± 0.55, compared with a decrease from 2.69 ± 0.59 to 2.01 ± 0.63 in the control group.

Table 5. Between-Group Difference in Change in Hepatic Steatosis Severity

Outcome	Timed Carbohydrate Restriction (n=37), Mean Change ± SD	Control (n=36), Mean Change ± SD	Difference in Mean Change	95% CI	Cohen's d
Hepatic steatosis score	-1.35 ± 0.52	-0.68 ± 0.49	-0.67	-0.90 to -0.44	-1.32

Table 5 footnote: CI, confidence interval; SD, standard deviation. Difference in mean change was calculated as timed carbohydrate restriction minus control. Cohen's d was calculated using pooled standard deviation of the change scores.

The reduction in hepatic steatosis score was greater in the timed carbohydrate restriction group than in the control group. The between-group difference in mean change was -0.67, with a 95% confidence interval from -0.90 to -0.44. The standardized effect size was -1.32, indicating a large magnitude of difference in hepatic steatosis reduction between groups.

Table 6 footnote: Repeated measures analysis assessed change in hepatic steatosis severity across baseline and 12 weeks between groups. Repeated measures analysis showed evidence of change over time, between-group difference, and a time-by-group interaction for hepatic steatosis severity. The time-by-group interaction supported a greater reduction in hepatic steatosis score over 12 weeks in the timed carbohydrate restriction group compared with the control group.

Table 6. Repeated Measures Analysis of Hepatic Steatosis Severity

Effect	F	p-value
Time	68.42	<0.001
Group	11.37	0.001
Time × Group	24.58	<0.001

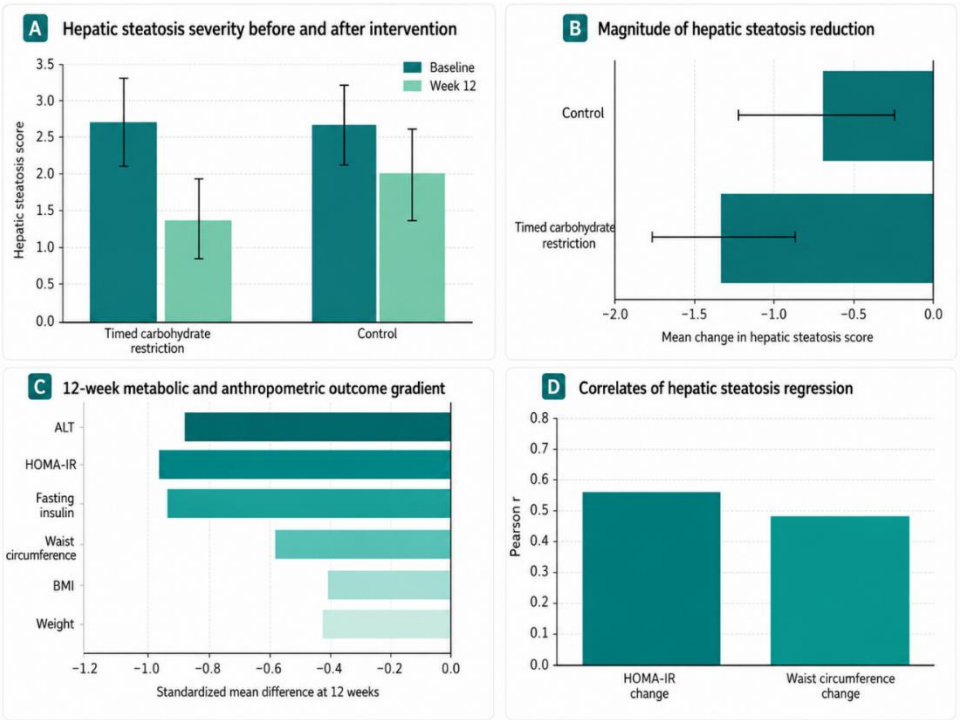

Figure 2 Timed carbohydrate restriction and hepatic steatosis regression after 12 weeks.

The panelled figure demonstrates greater hepatic steatosis regression in the timed carbohydrate restriction group compared with the control group. Hepatic steatosis score decreased from 2.73 ± 0.61 to 1.38 ± 0.55 in the intervention group and from 2.69 ± 0.59 to 2.01 ± 0.63 in the control group, with a

between-group difference in mean change of -0.67 and a large standardized effect size of -1.32. At 12 weeks, standardized between-group differences also favored timed carbohydrate restriction for weight, BMI, waist circumference, fasting insulin, HOMA-IR, and ALT, with the strongest gradients observed for HOMA-IR, fasting insulin, and ALT. Reductions in hepatic steatosis were positively correlated with reductions in HOMA-IR ($r=0.56$, $p<0.001$) and waist circumference ($r=0.48$, $p<0.001$), indicating that improvement in insulin resistance and central adiposity accompanied liver fat regression.

Table 7. Secondary Outcomes at 12 Weeks Among Participants With Completed Follow-Up

Variable	Timed Carbohydrate Restriction (n=37), Mean ± SD	Control (n=36), Mean ± SD	p-value
Weight, kg	88.6 ± 9.4	92.7 ± 10.2	0.021
BMI, kg/m²	31.8 ± 2.6	32.9 ± 2.8	0.039
Waist circumference, cm	101.4 ± 7.3	105.8 ± 8.1	0.014
Fasting insulin, μIU/mL	12.4 ± 2.8	15.1 ± 3.4	<0.001
HOMA-IR	2.68 ± 0.61	3.29 ± 0.74	<0.001
ALT, U/L	35.7 ± 8.6	43.5 ± 10.1	<0.001

Table 7 footnote: BMI, body mass index; HOMA-IR, homeostatic model assessment of insulin resistance; ALT, alanine aminotransferase; SD, standard deviation. At 12 weeks, the timed carbohydrate restriction group showed lower mean body weight, body mass index, waist circumference, fasting insulin, HOMA-IR, and alanine aminotransferase compared with the control group. These findings indicate that the intervention group had more favorable anthropometric, insulin-resistance, and hepatic enzyme profiles at the end of follow-up.

Table 8. Correlation Between Reduction in Hepatic Steatosis Severity and Change in Metabolic or Anthropometric Parameters

Variable	r	p-value
Change in HOMA-IR	0.56	<0.001
Change in waist circumference	0.48	<0.001

Table 8 footnote: HOMA-IR, homeostatic model assessment of insulin resistance; r, Pearson correlation coefficient. Reductions in hepatic steatosis severity were positively correlated with reductions in HOMA-IR and waist circumference. The correlation with HOMA-IR was stronger than the correlation with waist circumference, suggesting that improvement in insulin resistance had a closer association with hepatic steatosis regression than central anthropometric change in the analyzed sample.

DISCUSSION

The present randomized controlled trial demonstrated that timed carbohydrate restriction was associated with greater 12-week regression of ultrasound-graded hepatic steatosis among non-diabetic adults with obesity compared with standard dietary advice. Participants assigned to consume carbohydrate-containing foods only between 8:00 a.m. and 4:00 p.m. showed a larger reduction in hepatic steatosis score than participants receiving conventional dietary guidance, with the intervention group decreasing from 2.73 ± 0.61 to 1.38 ± 0.55 and the control group decreasing from 2.69 ± 0.59 to 2.01 ± 0.63. The between-group difference in mean change was -0.67, and the repeated measures analysis showed a significant time-by-group interaction, indicating that the pattern of improvement over 12 weeks differed between groups. These findings suggest that aligning carbohydrate intake with an earlier daytime window may be a clinically useful adjunct to standard lifestyle management for obesity-associated hepatic steatosis, although the observed benefit should be interpreted as the effect of the assigned dietary strategy rather than as proof of an isolated timing effect independent of total dietary intake or weight change (6).

The improvement in hepatic steatosis observed in the timed carbohydrate restriction group is biologically plausible within the framework of chrononutrition and circadian metabolic regulation. Glucose tolerance, insulin sensitivity, hepatic lipid metabolism, and substrate oxidation vary across the day, and earlier nutrient intake may better coincide with periods of more efficient metabolic handling. By restricting carbohydrate exposure to the earlier part of the day, the intervention may have reduced

late-day postprandial insulin demand and hepatic lipogenic drive, thereby supporting a more favorable metabolic environment for liver fat reduction. Previous chrononutrition and time-restricted feeding literature has suggested that meal timing can influence weight regulation, glucose metabolism, insulin action, and lipid handling, which is consistent with the direction of effects observed in this trial (7,8).

A clinically important finding was the greater improvement in insulin-related outcomes in the timed carbohydrate restriction group. At 12 weeks, fasting insulin and HOMA-IR were lower in the intervention group than in the control group, and reductions in hepatic steatosis severity were moderately correlated with reductions in HOMA-IR. This association supports the close physiological relationship between insulin resistance and hepatic fat accumulation. Reduced insulin resistance may decrease hepatic de novo lipogenesis and facilitate mobilization of intrahepatic triglyceride stores, thereby contributing to the observed improvement in steatosis severity. However, because the study did not include mediation analysis or baseline-adjusted models incorporating weight change, caloric intake, and physical activity, the relationship between improved insulin resistance and liver fat regression should be interpreted as an associated metabolic pattern rather than a confirmed causal pathway (9,10).

The intervention group also demonstrated more favorable anthropometric outcomes at 12 weeks, including lower body weight, body mass index, and waist circumference. The positive correlation between reductions in hepatic steatosis and waist circumference suggests that central adiposity improvement accompanied liver fat regression. This finding is clinically relevant because visceral and central adiposity are strongly linked with hepatic lipid deposition and cardiometabolic risk. Nevertheless, the available data do not allow separation of the relative contributions of carbohydrate timing, caloric reduction, dietary quality, adherence behavior, and weight loss. Therefore, while timed carbohydrate restriction appears to be a practical and effective dietary strategy in this trial, the results should not be interpreted as demonstrating that carbohydrate timing alone was responsible for all observed hepatic and metabolic improvements (11,12).

The findings add to the growing clinical interest in dietary timing interventions for metabolic disease prevention and management. Unlike strict ketogenic or highly restrictive low-carbohydrate diets, the intervention evaluated in this trial focused on limiting the timing of carbohydrate intake rather than completely eliminating carbohydrate-containing foods. This approach may be more acceptable in populations where carbohydrate-rich foods are culturally and routinely consumed. From a clinical implementation perspective, advising patients to consume carbohydrate-containing meals earlier in the day may be easier to communicate and monitor than complex macronutrient prescriptions. The repeated counseling contacts and dietary logs used in this study also reflect a feasible model for integrating timed carbohydrate restriction into routine lifestyle counseling, particularly for adults with obesity and ultrasound-confirmed hepatic steatosis (13,14).

Several methodological strengths support the interpretability of the findings. The randomized controlled design reduced selection bias and improved causal inference compared with observational dietary studies. Allocation concealment helped protect the randomization process, and blinding of outcome assessors and data analysts reduced the risk of measurement and analytical bias. Both groups received repeated dietary counseling contacts, which minimized differential attention between intervention arms. The inclusion of non-diabetic adults with obesity addressed a clinically important group that may have hepatic steatosis and insulin resistance before progression to overt diabetes. The assessment of hepatic, anthropometric, and metabolic outcomes also allowed the intervention effect to be evaluated across clinically relevant domains rather than through liver fat grading alone (15,16).

The study also has limitations that should be acknowledged. First, the follow-up period was limited to 12 weeks, so the durability of hepatic steatosis regression and long-term adherence to timed carbohydrate restriction could not be determined. Second, dietary adherence was assessed through self-reported dietary logs and telephone follow-up, which may be vulnerable to recall bias and social desirability bias. Third, hepatic steatosis was assessed using ultrasonography, which is practical and clinically accessible

but less sensitive and less quantitative than magnetic resonance imaging proton-density fat fraction or controlled attenuation parameter. Fourth, the analysis was based on participants who completed follow-up assessments, so the post-intervention results represent an available-case analysis rather than a full intention-to-treat analysis. Fifth, the study did not report detailed energy intake, macronutrient intake, sleep timing, or objectively measured physical activity, limiting the ability to isolate the independent contribution of carbohydrate timing from other behavioral changes (17,18).

The single-region setting may also limit generalizability. Southern Punjab was selected because of relatively consistent carbohydrate-based eating patterns, but results may differ in populations with different dietary traditions, occupational schedules, socioeconomic contexts, or baseline metabolic risk profiles. Moreover, the trial excluded individuals with diabetes, significant alcohol intake, viral hepatitis, and other chronic liver diseases; therefore, the findings should not be generalized directly to patients with diabetes, advanced liver disease, or mixed etiologies of steatosis. Future multicenter trials with larger samples, longer follow-up, objective dietary monitoring, advanced liver fat quantification, and baseline-adjusted or mediation-based statistical models would help clarify whether the observed benefits are sustained and whether carbohydrate timing exerts effects beyond weight loss and overall dietary improvement (19,20).

CONCLUSION

Overall, the study supports timed carbohydrate restriction as a promising, behaviorally feasible dietary strategy for improving ultrasound-graded hepatic steatosis and metabolic risk indicators in non-diabetic adults with obesity. The findings are clinically meaningful because they suggest that the timing of carbohydrate intake may be incorporated into lifestyle advice without requiring severe macronutrient elimination. However, the results should be interpreted with appropriate caution because the available data do not fully distinguish timing-specific effects from broader dietary adherence, caloric restriction, or weight-related metabolic improvements.

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