pISSN 2320-6071 | eISSN 2320-6012

Original Research Article

DOI: https://dx.doi.org/10.18203/2320-6012.ijrms20253602

Impact of dietary patterns on non-alcoholic fatty liver disease: a case-control study at a tertiary care hospital in western India

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Received: 14 August 2025 Revised: 18 September 2025 Accepted: 07 October 2025

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ABSTRACT

Background: Non-alcoholic fatty liver disease (NAFLD) is a growing concern worldwide, particularly in India, due to lifestyle changes and dietary habits. Diet plays a significant role in the onset of NAFLD, yet the specific dietary patterns contributing to this condition vary across populations. This study aims to assess the association between dietary patterns and NAFLD across different populations.

Methods: We conducted a case-control study at a tertiary care hospital in Vadodara, Gujarat, involving 200 participants (100 cases with NAFLD and 100 matched controls without NAFLD). Dietary intake was assessed using a modified 24-hour dietary recall method. To identify common dietary patterns, principal component analysis (PCA) was performed using the open-source statistical software R. Multivariable logistic regression was performed to estimate the odds ratios (ORs) for NAFLD risk, adjusting for confounders such as age, sex, BMI, physical activity, and smoking status.

Results: The study, involving 200 participants (100 with NAFLD and 100 controls), revealed that high intake of processed foods and low fiber intake were significantly associated with an increased risk of NAFLD. Specifically, high consumption of processed foods was linked to a 3.4-fold increased risk of NAFLD (Odds Ratio [OR] 3.4; 95% Confidence Interval [CI]: 2.1-5.6). Similarly, low fiber intake was associated with a 2.7-fold increased risk (OR 2.7; 95% CI: 1.7-4.5). These results highlight the strong impact of dietary patterns on the risk of NAFLD.

Conclusions: This study highlights that a Western dietary pattern is strongly associated with an increased risk of NAFLD, while adherence to a traditional diet appears protective. Dietary modifications could serve as a potential strategy for NAFLD prevention in at-risk populations.

Keywords: Case-control study, Dietary patterns, India, Non-alcoholic fatty liver disease, Traditional diet, Western diet

INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) is one of the most prevalent chronic liver disorders worldwide and is closely linked to obesity, insulin resistance and other metabolic risk factors. Global estimates indicate that roughly one quarter of adults are affected, and the disease burden is rising alongside the worldwide obesity epidemic. In India the burden is particularly high: a

recent systematic review and meta-analysis of studies across India estimated a pooled adult prevalence of ≈38.6% (95% CI 32-45.5), with even higher rates in highrisk groups and many urban cohorts.^{2,3}

Dietary composition and dietary patterns are major modifiable determinants of NAFLD risk. Multiple mechanistic and clinical studies show that excessive energy intake, high free sugars (notably fructose), refined carbohydrates and saturated fat promote hepatic de novo

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lipogenesis, intrahepatic triglyceride accumulation and insulin resistance key pathways in NAFLD pathogenesis. ⁴⁻⁶ Intervention and feeding studies demonstrate that saturated fats more strongly increase intrahepatic triglyceride content compared with polyunsaturated or monounsaturated fats, while diets low in free sugars and refined carbohydrates reduce liver fat. ^{6,7}

Epidemiological evidence supports the harms of Westernized and ultra-processed diets and the benefits of fiber-rich, plant-forward dietary patterns. Meta-analyses and cohort studies link Western dietary patterns and high ultra-processed food (UPF) consumption with increased NAFLD risk, while higher intake of dietary fiber and adherence to Mediterranean or other plant-forward healthy dietary patterns are generally associated with lower NAFLD prevalence or improvements in surrogate markers of liver fat and metabolic health. ⁸⁻¹³

Specific evidence in Indian and Asian populations highlights both the high prevalence and regional variation of NAFLD and dietary drivers. Reviews and region-specific studies in South Asia emphasize rapid nutritional transition, increasing refined carbohydrate and processed food intake, and a high prevalence of NAFLD even in non-obese individuals underscoring the need for population-specific dietary research and interventions. ^{3,9,14-16}

Given these converging lines of evidence, the present casecontrol study from Vadodara, Gujarat, aimed to examine habitual dietary patterns (including processed food and fiber intake) and their associations with NAFLD in an Indian tertiary-care population, using principal component analysis to define common patterns and multivariable models to adjust for major confounders.

METHODS

Study design

This study was conducted as a case-control study to evaluate the association between dietary patterns and the risk of developing non-alcoholic fatty liver disease (NAFLD). The design allows for the comparison of dietary habits between individuals with NAFLD (cases) and those without the disease (controls). The objective was to identify specific dietary patterns that may be associated with an increased risk of NAFLD.

Study area

The study was carried out at SSG Hospital, a tertiary care hospital in Vadodara, Gujarat. The hospital serves a diverse population, providing a suitable setting for capturing a wide range of dietary patterns and socioeconomic backgrounds. Participants were recruited from the outpatient departments of gastroenterology and hepatology.

Sample size

The total sample size for the study was 200 participants, with 100 cases and 100 controls. This sample size was calculated based on prior prevalence data of NAFLD and dietary habits in similar populations, ensuring adequate power to detect statistically significant associations between dietary patterns and NAFLD risk.

Sampling procedure

Participants were selected using systematic random sampling. Cases were individuals diagnosed with NAFLD based on clinical and radiological assessments, such as ultrasound or elastography. Controls were recruited from the same hospital setting, matched to cases by age and gender but with no clinical or radiological evidence of NAFLD or other liver disorders.

Study period

The study was conducted over a six-month period, from August 2023 to February 2024. This timeframe allowed for the recruitment of a sufficient number of participants and ensured seasonal variations in dietary patterns were accounted for.

Inclusion and exclusion criteria

Inclusion criteria for cases included adults aged 18 years and above with a confirmed diagnosis of NAFLD. Controls were adults aged 18 years and above without any known liver diseases.

Exclusion criteria

Exclusion criteria for both groups included a history of alcohol consumption (≥30g/day for men, ≥20g/day for women), the presence of viral hepatitis, autoimmune liver diseases, or other chronic conditions such as diabetes or cardiovascular disease that could confound the relationship between diet and NAFLD. Participants with incomplete dietary data or those unwilling to provide informed consent were also excluded.

Ethical approval

The study protocol was approved by the Institutional Ethics Committee of SSG Hospital, Vadodara (IEC/2023/178). Written informed consent was obtained from all participants.

Questionnaire

Dietary habits were assessed using a structured questionnaire adapted from validated tools for Indian populations. ^{11,18} It was pilot tested in 20 individuals for clarity and reliability. Nutrient intake was calculated using Indian Council of Medical Research (ICMR) food composition tables.

Statistical analysis

Data were analyzed using R software (version 4.3.1). Principal component analysis (PCA) was used to identify dietary patterns. Logistic regression estimated adjusted odds ratios (ORs) with 95% confidence intervals (CIs). A p-value <0.05 was considered statistically significant.

RESULTS

Demographic and clinical characteristics

The study enrolled a total of 200 participants, comprising 100 individuals diagnosed with non-alcoholic fatty liver disease (NAFLD) and 100 matched controls. The mean age of participants with NAFLD was 45.7 years (range: 31.5-59.8 years), and for the controls, it was 44.5 years (range: 32.2-58.4 years). The gender distribution was 58% male and 42% female in both groups (Table 1).

Table 1: Demographic and clinical characteristics of participants.

Characteristic	NAFLD group (n=100)	Control group (n=100)
Age (mean±SD)	45.7±8.6	44.5±8.9
Gender (Male)	58	58
Gender (Female)	42	42
BMI (mean±SD)	30.3±4.2	24.8±3.5
Waist circumference (cm)	102.6±8.3	89.7±7.9

Dietary patterns

Dietary assessments revealed significant differences in the consumption of specific food groups between the NAFLD group and the controls. The NAFLD group had higher intake of processed foods, sugary beverages, and red meats compared to the control group. Conversely, the control group had greater consumption of fruits, vegetables, and whole grains (Table 2).

Table 2: Dietary intake in NAFLD vs. Control group.

Food group	NAFLD Group (mean±SD)	Control Group (mean±SD)
Processed foods (g/day)	110.3±25.4	85.7±23.6
Sugary beverages (ml/day)	250.8±54.3	180.5±48.7
Red meats (g/day)	150.6±35.2	120.4±30.1
Fruits (servings/day)	2.5±1.1	4.3±1.3
Vegetables (servings/day)	3.1±1.2	5.2±1.5
Whole grains (servings/day)	2.0±0.9	3.5±1.1

Nutritional intake analysis

Statistical analysis indicated a significant difference in the intake of total calories and specific macronutrients

between the two groups. The NAFLD group consumed an average of 2,350.7 kcal/day, while the control group had an average intake of 1,945.6 kcal/day. The NAFLD group had a higher average intake of saturated fats (55.3 g/day vs. 42.8 g/day) and lower intake of fiber (18.6 g/day vs. 25.4 g/day) compared to the control group (Table 4).

Table 3: Nutritional intake in NAFLD vs. Control group.

Nutrient	NAFLD Group (mean±SD)	Control Group (mean±SD)
Total calories (kcal/day)	2,350.7±350.2	1,945.6±320.5
Saturated fats (g/day)	55.3±12.5	42.8±10.4
Fiber (g/day)	18.6±5.8	25.4±6.2

Prevalence of specific dietary patterns

Analysis showed that individuals in the NAFLD group were more likely to follow dietary patterns characterized by high consumption of processed foods and low intake of fiber-rich foods. The odds ratio for developing NAFLD associated with high intake of processed foods was 3.4 (95% CI: 2.1-5.6), and for low fiber intake, it was 2.7 (95% CI: 1.7-4.5) (Table 4).

These results suggest that specific dietary patterns are strongly associated with an increased risk of NAFLD. The observed trends align with previous research indicating that high consumption of processed foods and low fiber intake are risk factors for liver disease.

Table 4: Odds ratios for dietary patterns and NAFLD risk.

Dietary pattern	Odds Ratio (95% CI)
High processed foods	3.4 (2.1-5.6)
Low fiber intake	2.7 (1.7-4.5)

DISCUSSION

In this case-control study of 200 participants from a tertiary hospital in Vadodara, Gujarat, we identified strong dietary associations with non-alcoholic fatty liver disease (NAFLD). Specifically, NAFLD cases consumed significantly more processed foods, saturated fat, and total calories, while controls reported higher fiber intake and greater consumption of fruits, vegetables, and whole grains. These findings suggest that dietary quality, beyond total energy intake, plays a crucial role in NAFLD development in this population.

Our findings regarding processed and ultra-processed food (UPF) intake are consistent with recent evidence. We observed that participants with higher processed food consumption were more likely to have NAFLD. This parallels results from Zhang et al, who in a meta-analysis showed a significant positive association between UPF

consumption and NAFLD risk.¹⁰ Similarly, Grinshpan et al concluded that diets high in UPFs increase susceptibility to NAFLD and related metabolic disorders.¹¹ Together, these data reinforce that Westernized dietary patterns rich in refined and processed foods may be major drivers of NAFLD both in Western and South Asian contexts.

Fiber intake was inversely related to NAFLD in our study, with controls consuming significantly higher fiber-rich foods compared to cases. This result aligns closely with Zhu et al, who demonstrated that higher dietary fiber intake was linked to lower NAFLD prevalence, partly mediated by improved metabolic health.⁸ In another NHANES-based analysis, Chen et al confirmed an inverse association between fiber intake and hepatic steatosis assessed by transient elastography.⁹ Our data extend these findings to an Indian population, highlighting the protective role of fiber across diverse populations.

We also found that cases had higher saturated fat and calorie intake than controls. These results mirror mechanistic feeding studies in which saturated fat intake significantly increased intrahepatic triglyceride accumulation compared to polyunsaturated fat.⁶ Hydes et al and Yki-Järvinen et al similarly emphasized that diets high in saturated fat and refined carbohydrates promote de novo lipogenesis and impair insulin sensitivity, thereby accelerating NAFLD progression.^{5,7} Thus, our casecontrol observations in an Indian setting are in agreement with global experimental and observational data.

By contrast, protective dietary patterns such as the Mediterranean diet have consistently shown benefits for liver health. Our observation that controls consumed more fruits, vegetables, and whole grains supports prior work. Anania et al and Moosavian et al reported that adherence to the Mediterranean diet improved liver fat and metabolic parameters in NAFLD patients. Similarly, Miryan et al showed that higher adherence to plant-based dietary indices was associated with better liver outcomes. These comparisons indicate that diets rich in fiber and plant-based foods may offer protection against NAFLD even outside the Mediterranean region.

At a regional level, our findings fit with existing literature from India and South Asia. A meta-analysis by Elhence et al estimated an overall NAFLD prevalence of $\approx\!38.6\%$ in Indian adults, underscoring the public health burden. Importantly, Duseja et al and Kuchay et al highlighted that NAFLD is common in Indian populations even among non-obese individuals, likely due to dietary transitions, genetic predispositions, and visceral adiposity. Our study contributes additional evidence by showing that poor dietary quality, characterized by high processed food intake and low fiber intake, is a major correlate of NAFLD in an urban Indian sample.

Clinical and public health implications

Our findings reinforce that dietary counseling focused on reducing processed/ultra-processed foods, lowering saturated fat and refined carbohydrate intake, and increasing fiber-rich whole foods (fruits, vegetables, legumes and whole grains) should be central to NAFLD prevention strategies in India. Population-level interventions to reduce UPF availability and improve access to fiber-rich whole foods may be particularly impactful.

This study has limitations. First, the case-control design precludes causal inference and raises the possibility of reverse causation. Second, diet was assessed by self-report (24-hour recall/FFQ format), which can introduce recall and measurement bias. Third, the sample was drawn from a single tertiary-care hospital, which may limit generalizability to the broader community. Fourth, although models adjusted for age, sex, BMI, physical activity and smoking, residual confounding (e.g., socioeconomic status, genetic predisposition, detailed measures of physical activity and caloric expenditure) cannot be excluded. Finally, our study did not include objective biomarkers of dietary intake or follow participants longitudinally to assess incidence or progression. Future prospective cohort studies with repeated dietary measures and objective biomarkers would strengthen causal inference and clarify mechanisms.^{7,15}

CONCLUSION

In line with current global and regional literature, our results indicate that Westernized/processed dietary patterns and low fiber intake are associated with increased odds of NAFLD in an Indian tertiary-care population. These findings support dietary modification as a practical target for NAFLD prevention and provide region-specific evidence to inform clinical counseling and public health policy.

Funding: No funding sources Conflict of interest: None declared

Ethical approval: The study was approved by the Institutional Ethics Committee of SSG Hospital, Vadodara (IEC/2023/178)

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Cite this article as: Godria P, Patel C, Patel H, Thaker P, Bhuva R, Patel D, et al. Impact of dietary patterns on non-alcoholic fatty liver disease: a case-control study at a tertiary care hospital in western India. Int J Res Med Sci 2025;13:4790-4.