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Original Article

Nutrition and physical activity in Asian Indians with non-alcoholic fatty liver: A case control study

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ABSTRACT

Aim: We tested the hypothesis that Asian Indians with non-alcoholic fatty liver disease (NAFLD) would have imbalanced diets and lower intensity of physical activity than those without NAFLD.
Methods: We studied dietary intake, intensity of physical activity and anthropometric and metabolic profiles in subjects with NAFLD and in healthy controls. Complete clinical, biochemical, dietary and physical activity profiles were studied for 169 cases and 173 controls in a prospective manner. Bivariate and multivariate analyses were carried out to identify the predictors of NAFLD [odds ratio (OR) and 95% confidence intervals (95%CI)].
Results: The mean dietary intakes of total energy, carbohydrate, protein, total fat, saturated fat and total cholesterol were significantly higher, while intake of monounsaturated fatty acids and polyunsaturated fatty acids was significantly lower in cases as compared to controls ($p < 0.01$ for all). Further, mean physical activity in a day (expressed as MET.Minutes) and total energy expenditure were significantly lower in cases than in controls (33.3 ± 3.6 *vs.* 36.2 ± 0.5, $p = 0.001$ and 2707.6 ± 505.6 *vs.* 2904.3 ± 690.3, $p = 0.02$, respectively). On multivariate analysis, percentage dietary total fat intake (OR: 13.4; 95% CI: 4.6 −39.3, $p = 0.001$), homeostatis model assessment for insulin resistance (OR: 6.9; 95% CI: 3.2−14.8, $p = 0.001$) abdominal obesity (OR: 2.7; 95% CI: 1.5−5.0, $p = 0.001$) and high serum triglycerides (OR: 2.1; 95%CI: 1.2−3.8, $p = 0.007$) were associated with an increased risk for development of NAFLD.
Conclusion: Decrease in intake of total dietary fats and improvement of insulin resistance, abdominal obesity and blood triglycerides should be important measures for management of NAFLD in Asian Indians in north India.

1. Introduction

Non-alcoholic fatty liver disease (NAFLD) is closely associated with the metabolic syndrome and insulin resistance. Importantly, insulin resistance is independently correlated to NAFLD regardless of adiposity [1,2]. Several studies in India indicate that about $^1/_3^{rd}$ of the urban population in India has the metabolic syndrome [3,4]. NAFLD is present in ~6−32% of general population in India [5−7].

Nutrition is believed to play an important role in the pathogenesis of NAFLD. Animal models and human studies suggest that dietary factors like saturated fats, dietary cholesterol and fructose can affect fatty infiltration and lipid peroxidation in the liver [8−11]. Early development of steatohepatitis has been shown in obese, hypercholesterolemic, diabetic *foz/foz* mice fed on high-fat diet for 12 months [12]. Effects of different diets on NAFLD has been researched mostly in other populations [13,14]. A study on Asian Indians in South India has shown that percent fat intake along with waist circumference (WC) and body mass index (BMI) are independent risk factors in NAFLD [15], but type of fat, detailed body composition analysis, measures of insulin resistance and physical activity profile was not evaluated.

Although physical activity has been investigated less in context of NAFLD, some data show that persons with NAFLD engaged in less reported leisure time physical activity, including total, aerobic, and resistance [16]. Further, an inverse association between fitness categories and the prevalence of NAFLD regardless of BMI has been shown [17]. In this context it is important to note that Asian Indians

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are less physically active than Europeans, which may lead to insulin resistance and the metabolic syndrome in former [18].

It is important to conduct research on NAFLD given increasing prevalence of obesity and the metabolic syndrome and large number of patients of type 2 diabetes mellitus (T2DM) in India. Further, the inter-relations of NAFLD, dietary composition and physical activity have not been adequately studied, particularly in Asian Indians.

We hypothesized that subjects with NAFLD would have imbalanced diets and lower intensity of physical activity. To test this hypothesis, we studied dietary intake, intensity of physical activity and anthropometric and metabolic profiles in subjects with NAFLD and in healthy controls, so as to identify potential predictors.

2. Subjects & methods

This study was conducted in outpatient department of Fortis Hospital, New Delhi (North India) after approval from the institutional ethics committee. The sample size has been calculated using data from previous study [6] which has shown 32% prevalence of NAFLD in Asian Indians in north India. We assumed to detect minimum odds ratio of 2 with α error of 5% and 80% power. With this assumption, the effective minimum sample size was 138 in each group. Exclusion, inclusion criteria and detailed history along with family history has been collected as defined previously [19]. A written informed consent was obtained.

Standardized food frequency questionnaire and 24-h dietary recall [19] were used to assess the dietary intake of nutrients such as energy, protein, carbohydrate, total fat, monounsaturated fats (MUFAs), polyunsaturated fatty acids (PUFAs), saturated fatty acids (SFAs), n-3 PUFAs, n-6 PUFAs, total cholesterol and vitamin E levels as previously [20]. The daily level of physical activity and total energy expenditure was assessed using a standard questionnaire developed in India [21,22].

-WC $\geq$90 cm for males and $\geq$80 cm for females was considered an indicator of abdominal obesity. The metabolic syndrome was defined based on presence of three out of following five components; WC, males $\geq$90 cm, females $\geq$80 cm, fasting blood glucose (FBG) $\geq$100 mg/dl, serum triglycerides (TG) $\geq$ 150 mg/dl, blood pressure $\geq$130/85 mmHg and high-density lipoprotein- cholestrol (HDL-c); males <40 mg/dl, and females <50 mg/dl [23].

2.1. Procedures

Measurements [Weight, height, BMI,WC, hip circumference (HC) and blood pressure] and bio-chemical analysis [FBG, total cholesterol (TC), TG, HDL-c, aspartate aminotransferase (U/l) and alanine aminotransferase (U/l), and fasting serum insulin] were done as described previously [6]. The lower limit of detection of insulin assay was 0.01 μU/ml, and reference range was 2.1–22 μU/ml.The intra-assay and inter-assay percentage coefficient variables were 1.95% and 2.23%, respectively. Fasting insulin and homeostasis model assessment for insulin resistance (HOMA-IR) were used to measure insulin resistance. The value of HOMA-IR was calculated by the following equation: fasting insulin (μU/ml) X fasting glucose (mmol/l)/22.5.

Dual Energy X-ray Absorptiometry (DEXA) [QDR-2000; Hologic, Waltham, MA, USA] was used to measure body composition [% body fat (% BF), body fat mass (BFM), fat free mass (FFM)] and data were analyzed using the software version 7.20. The procedure for liver ultrasound was followed as described previously [19].

2.2. Statistical analysis

The data were tested for normal distribution by using Q-Q plot. The variables that were not distributed normally were further confirmed by testing for skewness. Such variables were subjected to log transformation and the transformed data were normally distributed. Results were presented as mean ± standard deviation. The differences between cases and controls were tested for non-normal continuous data using Wilcoxon rank sum test. The Chi-square and/or Fisher's exact test was used to compare proportions. All variables that were significant on bivariate analysis ($p < 0.05$) were used in the multivariate analyses to identify the independent predictors of NAFLD and to estimate odds ratio (OR) and 95% confidence intervals (95% CI). Charateristics of cases and controls were compared using paired sample *t*-test. $p < 0.05$ was considered as statistically significant.

3. Results

Complete clinical, biochemical, dietary and physical activity data were available for 169 cases (male: 109) and 173 controls (male: 130). Both groups were age- and BMI-matched. The presence of the metabolic syndrome was significantly higher among cases as compared to control (32.9% *vs.* 11.3%; $p = 0.001$).

Significant higher values of systolic and diastolic blood pressure, WC, HC, FM, BFM and %BF were recorded in cases as compared to controls (Table 1). Cases had significantly higher value of FBG, fasting insulin, HOMA-IR, ALT, TC and TG as compared to controls.

The mean dietary intake of total energy, total carbohydrate, protein, total fat, SFAs and TC was significantly higher, while intake of MUFAs and PUFAs was significantly lower in cases as compared to controls.

Total energy expenditure was significantly lower in cases than in controls (2707.6 ± 505.6, 2904.3 ± 690.3, $p = 0.02$, respectively). Mean physical activity in a day expressed as MET.minutes were significantly lower in cases than in controls (33.3 ± 3.6, 36.2 ± 0.5, $p = 0.001$respectively), as shown in Table 2.

3.1. Independent predictors of NAFLD

Bivariate analysis identified multiple factors associated with development of NAFLD. Percentage total fat intake (OR: 15.7; 95% CI: 6.09–40.4, $p < 0.001$), high intake of dietary cholesterol (OR: 4.3; 95%CI: 1.5–11.8, $p < 0.001$), presence of metabolic syndrome (OR: 3.8; 95% CI: 2.1–6.8, $p < 0.001$), high serum TG (OR: 2.8; 95% CI: 1.7–4.3, $p < 0.001$), abdominal obesity (OR: 2.8; 95% CI: 1.7–4.3, $p < 0.001$), HOMA-IR (OR: 1.8; 95% CI: 1.5–2.1, $p < 0.001$), fasting

Table 1
Clinical and Biochemical Profiles.

Variables	Cases (n, 169)	Controls (n, 173)	p value*
Age (years)	38.3 ± 6.9	37.2 ± 7.0	ns
Systolic blood pressure (mmHg)	125 ± 11.8	119.4 ± 11	0.01
Diastolic blood pressure (mmHg)	80.2 ± 8.7	77.4 ± 8.6	0.00
Body mass index (kg/m^2)	27.9 ± 2.7	27.36 ± 3.19	NS
Waist circumference (cm)	94.6 ± 9.6	89.8 ± 9.15	0.00
Hip circumference (cm)	97.1 ± 7.2	94.2 ± 8.1	0.00
Body fat mass (kg)	27.6 ± 7.3	25.4 ± 8.0	0.00
Fat free mass (kg)	47.6 ± 9.7	45.5 ± 8.7	0.05
Body fat (%)	33.9 ± 6.4	32 ± 8.4	0.05
Fasting blood glucose (mg/dl)	89.8 ± 12.0	87.0 ± 10.8	0.03
Fasting insulin (ln) (μU/ml)	2.1 ± 0.84	1.9 ± 0.58	0.04
HOMA-IR	0.6 ± 0.8	0.4 ± 0.6	0.02
Alanine aminotransferase (U/l)	39.0 ± 21.5	34.4 ± 13.3	0.01
Aspartate aminotransferase (U/l)	36.03 ± 19.4	33.1 ± 11.4	0.09
Alkaline phosphatase (IU/L)	136.0 ± 57.5	134.9 ± 63.1	0.87
Total cholesterol (mg/dl)	189.8 ± 31.2	177.7 ± 30.0	0.00
Triglyceride (mg/100 ml)	172.9 ± 77.9	146.5 ± 66.0	0.00
HDL-c (mg/100 ml)	39.0 ± 6.2	38.9 ± 11.3	0.91
LDL-c (mg/dl)	110.2 ± 24.7	103.2 ± 26.4	0.01

Table 2
Daily Macro- and Micro-nutrient Intake and Physical Activity.

Variables	Cases (n, 169)	Controls (n, 173)	p value
Energy (ln) (Kcal)	7.6 ± 0.46	7.5 ± 0.25	0.01
Carbohydrate (ln) (g)	5.15 ± 0.37	5.07 ± 0.30	0.03
Protein (ln) (g)	3.8 ± 0.38	3.6 ± 0.31	0.00
Total Fat (ln) (g)	4.7 ± 0.56	4.6 ± 0.9	0.04
Monounsaturated fatty acids (g)	26.3 ± 10.4	34.7 ± 11.6	0.01
Polyunsaturated fatty acids (ln) (g)	2.6 ± 0.1	2.7 ± 0.1	0.01
Saturated fats (ln) (g)	3.37 ± 0.64	3.2 ± 0.48	0.02
n-3 polyunsaturated fatty acids (g)	0.53 ± 1.3	0.6 ± 1.1	0.60
n-6 polyunsaturated fatty acids (g)	3.5 ± 0.8	3.6 ± 0.37	0.15
Total cholesterol (ln) (g)	4.6 ± 0.86	4.2 ± 1.15	0.01
Total fiber (g)	25.9 ± 10.8	25.7 ± 8.2	0.66
Vitamin E (IU)	2.6 ± 1.2	2.8 ± 0.68	0.8
Physical activity (MET per min)	33.3 ± 3.6	36.2 ± .5	0.001
Total energy expenditure (Kcal)	2707.6 ± 505.6	2904.3 ± 690.3	0.02

*ln = log natural.

insulin (OR: 1.1; 95% CI: 1.0–1.1, p < 0.001) and -total fat (OR: 1.0; 95%CI: 1.1–1.7, p < 0.01).

On multivariate analysis, only four variables namely percentage dietary fat intake (OR: 13.4; 95%CI: 4.6–39.3, p = 0.001), HOMA-IR (OR: 6.9; 95%CI: 3.2–14.8, p = 0.001) abdominal obesity (OR: 2.7; 95%CI: 1.5–5.0, p = 0.001) and high TG (OR: 2.1; 95%CI: 1.2–3.8, p = 0.007) were associated with an increased risk for development of NAFLD (Table 3).

4. Discussion

In this study on apparently healthy Asian Indians, we have found that percentage dietary fat intake, along with three components of the metabolic syndrome (insulin resistance, abdominal obesity and high serum triglycerides) were independent risk factors for the development of NAFLD.

In India, over the past 30 years (1973–2004) intake of energy derived from carbohydrates is decreased by 7% and energy derived from fats increased by 6% [24]. Importantly, in our previous study, we noted that percentage caloric intake from saturated fat was 9.4%, which is an independent risk factor for subclinical inflammation as depicted by high levels of highly sensitive c-reactive protein (hs-CRP) levels [25]. Further, we have also reported higher high-sensitivity C-reactive protein- levels in persons with NAFLD as compared to those without NAFLD independent of obesity and abdominal obesity [26]. Data published by the National Sample Survey Organization (Government of India, National Sample Survey Organization, 2009–2010) show that there have been substantial increase in the consumption of edible oils over a period of previous 16 years [27]. All these dietary changes and physical inactivity has contributed to obesity and the metabolic syndrome in India.

Asian Indians are prone to develope NAFLD at lower BMI levels as compared to the wester populations [28]. Importantly, lean, non-alcoholic, non-diabetic, non-smoking Asian Indians when compared to matched Caucasians, Hispanics, Black and Eastern Asians had 2- to 3-folds increase in insulin resistance - and 2-fold increase in hepatic triglyceride content [28]. These results could be explained by high levels of non-esterified fatty acids (NEFAs) generated due to excess abdominal adiposity, (subcutaneous and intra-abdominal) in Asian Indians [29,30]. Importantly, high post-meal plasma levels of non-esterified fatty acids NEFAs-have been reported in Asian Indians as compared with BMI-matched white Caucasians [31].

Table 3
Stepwise logistic regression model for predictors of NAFLD.

Variables	p-value	Odds ratio	95% CI
Percentage dietary fat	0.001	13.4	4.6–39.3
HOMA-IR	0.001	6.9	3.2–14.8
Abdominal obesity	0.001	2.7	1.5–5.0
High serum triglycerides	0.007	2.1	1.2–3.8

In our study, although higher intakes of energy, carbohydrate, cholesterol, SFAs, n-3 PUFAs and lower intake of MUFAs and n-6 PUFAs were observed in subjects with NAFLD as compared with controls, no significant relationships between NAFLD and these dietary factors were observed. Consistent with our findings, Musso et *al* [9] studied small number of subjects (n = 25) with NAFLD and age gender matched 25 healthy controls and found that the intake of saturated fats (13.7% ±3.1% *vs.* 10.0% ±2.1% total kcal, respectively, Pp= 0.0001) and cholesterol (506 ± 108 *vs.* 405 ± 111 mg/d, respectively, p = 0.002) were significantly higher among subjects with NAFLD as compared to healthy controls. In this context, it is important to note that high fat diets rapidly increase plasma TG and NEFAs levels, increase hepatic triglycerides content, and cause insulin resistance [32,33]. Similarly, in another short-term intervention study (3-days) with hypercaloric diet (characterized by high fat and high-energy content) has shown significant increase in plasma NEFAs (0.92 ± 0.33 *vs.*0.54 ± 0.29, p = 0.002) and hepatic triglycerides (4.26 ± 2.78 *vs.* 2.01 ± 1.79; p < 0.001) as compared with baseline levels [34].

A cross-over 6-week dietary study on Mediterranean diets (high in MUFAs) has shown reduction in liver steatosis by 34% as measured by magnetic resonance^{1}H spectroscopy in subjects with NAFLD even without weight loss [35]. In Asian Indians with NAFLD, 6-month randomized intervention trial using olive and canola oils (rich in MUFAs and having a balanced n-6/n-3 PUFAs ratio) as a cooking medium resulted in a significant reduction in severity of fatty liver [grade I, from 73.3% to 23.3% and from 60.5% to 20%, respectively (p < 0.01); grade II, from 20% to 10% and from 33.4% to 3.3%, respectively (p < 0.01); and grade III, from 6.7% to none and from 6.1% to none, respectively] and liver span (pre- and post difference, 1.14 ± 2 cm; p < 0.05 and 0.66 ± 0.33 cm; p < 0.05, respectively) [19]. Overall, it appears that dietary fats are important determinants of NAFLD in Asian Indians, and high MUFA diets are beneficial.

Analysis of a large population-based cohort NHANES 2003–2006 survey (n, 6093), showed that subjects with of NAFLD had significantly (p < 0.01) reduced physical activity levels [36]. Our study also confirmed that the level of physical activity is low among subjects with NAFLD, however it did not emerge as a significant predictor. Physical activity has beneficial effect on insulin resistance [37], free fatty acid metabolism [37] and decreases hepatic triglyceride accumulation [38]. Intervention with regular aerobic

exercise has shown reduction in liver fat despite no or minimal weight loss [38].

We acknowledge limitations of our study. Use of food composition table for estimation of macro- and micro-nutrients does not account for the moisture content or cooking loss of the nutrients. We also excluded unacceptable energy levels of the participants (<500 kcal or >5000 kcal). The physical activity measures may also overestimate activity, particularly moderate and vigorous activities [39].

Individual contribution

PN, SBP: formulation study question: AM, SBP, designing study: SBP: data collection: PN, SBP: analysing the data.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.dsx.2019.01.054.

Conflict of interest and financial disclosure

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