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RESEARCH ARTRICLE

Association of plasma glucose, serum lipid profile, and liver enzymes with non-alcoholic fatty liver disease - A case–control study

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ABSTRACT

Background: The prevalence and complications of nonalcoholic fatty liver disease (NAFLD) have increased dramatically over the past decade. The pandemic of lifestyle disorders and their role in the development of NAFLD encourage studies in this aspect. **Aims and Objectives:** To evaluate the association of laboratory parameters such as plasma glucose (fasting, postprandial, and glycated hemoglobin), serum lipid profile, and liver enzymes with NAFLD. **Materials and Methods:** Our case–control study enrolled 150 participants [81 cases (sonological evidence of fatty infiltration of liver) and 79 controls] in a period of 2 years. All laboratory investigations were done in the central laboratory of the institution using appropriately standardized techniques. Institutional ethics committee approved this study and written informed consent was obtained from all study participants. Data were analyzed using free software R®, nominal variables were compared using independent sample *t*-test and Mann–Whitney U-test; categorical variables were compared using Chi-square test. $P < 0.05$ was considered statistically significant. **Results:** Higher number of male participants (56.3%) enrolled in the study. Significant difference in total cholesterol (TC) ($P < 0.001$), low-density lipoprotein (LDL) ($P < 0.001$), triglycerides (TG) ($P < 0.001$), alanine aminotransferase (ALT) ($P = 0.002$), TC/high-density lipoprotein (HDL) ($P = 0.001$), and TG/HDL ($P < 0.001$) was observed between cases and controls. Significant association with NAFLD was observed for TG ($P = 0.001$), TC ($P < 0.001$), dyslipidemia ($P = 0.02$), aaspartate aminotransferase (AST) ($P = 0.03$), and ALT ($P < 0.001$). **Conclusion:** High prevalence of prediabetes and diabetes were observed among participants undergoing voluntary health checkup. Significant difference in TC, LDL, TG, TC/HDL, TG/HDL, and ALT was observed between cases and controls. Significant association with NAFLD was observed for TG, TC, dyslipidemia, AST and ALT.

KEY WORDS: NAFLD; Lipid Profile; Liver Enzymes; Plasma Glucose

INTRODUCTION

The occurrence of hepatic steatosis in the absence of alcohol consumption is called non-alcoholic fatty liver disease (NAFLD). This vaguely defined blanket term ranges from nonalcoholic fatty liver which is fatty infiltration of more than 5% of liver parenchyma to non-alcoholic steatohepatitis which is characterized by necroinflammation of hepatocytes preceding fatty infiltration. Although the prevalence, burden, and complications of this disease have increased dramatically over the past decade, the definition still has not evolved over time. The progression of NAFLD has not been completely unraveled, though reports of progression to hepatocellular carcinoma (HCC) and end-stage liver disease have been poisted.[1] The global prevalence of NAFLD is estimated to be 4-46%[1] with an estimated average of 25%[2] and

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has demonstrated paralleled increase over the past decade similar to other lifestyle disorders. The use of radiographic investigations over liver enzymes for the diagnosis of NAFLD has contributed to the increase in reported prevalence of NAFLD to some extend.[3] India has ascended to the position of one of the countries with the highest prevalence (>30%)[3] of NAFLD though the exact prevalence has not been reported.

Insulin resistance (IR)[4] and its components such as dyslipidemia[5] and other lifestyle disorders are considered as risk factors for development of NAFLD. The association between liver enzymes and NAFLD has not yet been clearly elucidated.[6] Owing to the abundance of lifestyle disorders among Keralites, an association of NAFLD with HCC,[7] increased cardiovascular risk,[8] and reduced survival[1] among NAFLD patients necessitates research in this entity. This study is conducted with the view of assessing the association between NAFLD and laboratory parameters such as plasma glucose, liver enzymes, and serum lipid parameters in our setting.

MATERIALS AND METHODS

The present cross-sectional case–control study enrolled 160 participants, 81 cases [with B-mode ultrasonographic (US) evidence of fatty infiltration of liver (Grades 1-3)] and 79 controls [with no US evidence of fatty infiltration of liver] aged ≥20 years, undergoing voluntary health checkup at Department of General Medicine, Sree Gokulam Medical College and Research Foundation, Venjaramoodu, Trivandrum, between 2014 and 2016. Unwilling participants, participants with history, clinical, laboratory or histological evidence of alcoholism, history of steatosis inducing drug use (e.g., methotrexate), HCC, anemia, and hemoglobinopathies were excluded from the study. Participants were categorized as prediabetics and diabetics according to ADA diagnostic criteria.[9] Low-density lipoprotein (LDL) ≥70 mg/dL and ≥190 mg/dL were considered elevated in diabetic and non-diabetic participants respectively, and high-density lipoprotein (HDL) < 40 mg/dL and < 50 mg/dL were considered reduced in male and female participants respectively. Triglyceride (TG) ≥ 130 mg/dL and total cholesterol (TC) ≥ 200 mg/dL were considered elevated, and participants with elevated LDL or reduced HDL or both were considered dyslipidemic. TC/HDL ratio ≥4 and TG/HDL ratio ≥2 were considered elevated, and participants were also categorized based on these ratios.[10] Aspartate aminotransferase (AST) and alanine aminotransferase (ALT) were considered as elevated if these parameters were higher than twice the upper limit of normal. All laboratory investigations were done in the central laboratory with appropriately standardized techniques. Sample size was calculated to detect a minimum odds of 0.2 assuming 20% exposure among controls (diabetes),[11] α of 0.05 and β of 0.2, case-to-control ratio of 1, and 10% attrition rate. The study was approved by the Institutional Ethics Committee, and written informed consent was obtained from all study participants. Data were collected in separate case record forms and were analyzed using free software R®™. Values are rounded off to the nearest decimal and are expressed as mean [standard error of mean (SEM)] (normal distribution) or median [interquartile range (IQR)] (non-normal distribution). Normality of distribution was assessed using Shapiro–Wilk test. Nominal variables were compared using independent sample *t*-test and Mann–Whitney U-test. Categorical variables were compared using Chi-square test. Odds ratio (OR) with 95% confidence interval (CI) was used to describe association in 2x2 table. $P < 0.05$ was considered statistically significant.

RESULTS

Among the study participants, 90 (56.3%) were males and 70 (43.8%) were females. The mean age, FPG, PPG, HbA1C, TC, LDL, HDL, TG, TC/HDL, TG/HDL, AST, and ALT of the study participants were 46.9 (0.9) years, 123.5 (3.2) mg/dL, 184.1 (5.6) mg/dL, 6.6 (0.1) %, 223 (3.7) mg/dL, 130.8 (2.8) mg/dL, 45.9 (1.2) mg/dL, 137.7 (5.9) mg/dL, 5.3 (0.2), 3.5 (0.2), 35.1 (2.7) IU/L, and 61.8 (3.5) IU/L, respectively. The baseline parameters of study participants are demonstrated in Table 1.

Gender-stratified comparison of parameters demonstrated significant difference in age ($P = 0.007$), FPG ($P = 0.02$), AST ($P = 0.003$), ALT ($P < 0.001$), HDL ($P < 0.001$), TG ($P = 0.01$), TC/HDL ($P = 0.03$), and TG/HDL ($P < 0.001$). No difference was observed in HbA1C ($P = 0.4$), PPG ($P = 0.6$), TC ($P = 0.06$), and LDL ($P = 0.1$) (Table 2).

No difference in gender distribution was observed between cases and controls ($P = 0.2$). Between cases and controls,

Table 1: Baseline parameters of the study participants

Parameter	n	Mean (SEM)	Median (IQR)
Age (years)	160	46.9 (0.9)	
FPG (mg/dL)	160		109 (94–143.5)
PPG (mg/dL)	160		161 (130–221.5)
HbA1C (%)	160		6 (5.4–7.6)
TC (mg/dL)	160	223 (3.7)	
LDL (mg/dL)	160		120 (106–152.75)
HDL (mg/dL)	160		43 (36–55)
TG (mg/dL)	160		119.5 (90.3–163.8)
TC/HDL	160		5 (3.8–6.4)
TG/HDL	160		2.8 (1.7–4.3)
AST (IU/L)	160		27 (22.3–35.8)
ALT (IU/L)	160		50 (37–72)

SEM: Standard error of mean, IQR: Interquartile range, FPG: Fasting plasma glucose, PPG: Postprandial glucose, HbA1C: Glycated hemoglobin, TC: Total cholesterol, LDL: Low-density lipoprotein, HDL: High-density lipoprotein, TG: Triglycerides, AST: Aspartate aminotransferase, ALT: Alanine aminotransferase

Table 2: Gender-stratified comparison of parameters

Parameter	Gender	*n*	Mean (SEM), Median (IQR)	*P*
Age (years)	Male	90	44.8 (1.2), 46 (34–53.3)	0.007*
	Female	70	49.7 (1.3), 51 (42.8–55)	
FPG (mg/dL)	Male	90	127.5 (4.1), 116 (98–153.5)	0.02∫
	Female	70	118.2 (5), 101.5 (89–128.5)	
PPG (mg/dL)	Male	90	184.1 (7), 177 (130–226)	0.6
	Female	70	184.2 (9.2), 155 (130–207.5)	
HbA1C (%)	Male	90	6.7 (0.2), 5.9 (5.4–7.9)	0.4
	Female	70	6.5 (0.2), 6 (5.4–7)	
TC (mg/dL)	Male	90	217 (5.1), 209.5 (184.8–245)	0.06
	Female	70	230.7 (5.3), 228.5 (203.3–260.8)	
LDL (mg/dL)	Male	90	126.9 (3.6), 120 (99–148.3)	0.1
	Female	70	135.8 (4.4), 122 (111–155.5)	
HDL (mg/dL)	Male	90	41.7 (1.2), 39 (33–50)	<0.001∫
	Female	70	51.3 (2), 49 (39–65.3)	
TG (mg/dL)	Male	90	155.2 (9.4), 134 (88.3–192.5)	0.01∫
	Female	70	115.3 (4.9), 110 (91.8–131.3)	
TC/HDL	Male	90	5.6 (0.2), 5.1 (4.1-6.8)	0.03∫
	Female	70	5 (0.2), 4.7 (3.7-5.7)	
TG/HDL	Male	90	4.1 (0.3), 3.5 (1.9-5.7)	<0.001∫
	Female	70	2.6 (0.2), 2.3 (1.5-3.2)	
AST (IU/L)	Male	90	39.4 (4.6), 29 (24–40.3)	0.003∫
	Female	70	29.5 (1.9), 25 (20.8–32.3)	
ALT (IU/L)	Male	90	71.7 (5.3), 59 (44–77.3)	<0.001∫
	Female	70	49.2 (3.4), 39 (33.8–54.3)	

SEM: Standard error of mean, IQR: Interquartile range, FPG: Fasting plasma glucose, PPG: Postprandial glucose, HbA1C: Glycated hemoglobin, TC: Total cholesterol, LDL: Low-density lipoprotein, HDL: High-density lipoprotein, TG: Triglycerides, AST: Aspartate aminotransferase, ALT: Alanine aminotransferase, *indicates significant difference between groups using independent sample *t*-test, ∫ indicates significant difference between groups using Mann–Whitney U-test

Table 3: Comparison of parameters between cases and controls

Parameter	NAFLD	*n*	Mean (SEM), Median (IQR)	*P*
Age (years)	Yes	81	46.5 (1.3), 48 (38.5–52.5)	0.6
	No	79	47.4 (1.3), 49 (36–55)	
FPG (mg/dL)	Yes	81	122.3 (4.5), 110 (94–141)	0.9
	No	79	124.7 (4.6), 109 (92–142)	
PPG (mg/dL)	Yes	81	183.9 (7.6), 160 (138–221)	0.6
	No	79	184.3 (8.3), 169 (126–222)	
HbA1C (%)	Yes	81	6.6 (0.2), 5.9 (5.4–7.4)	0.9
	No	79	6.6 (0.2), 6 (5.4–7.7)	
TC (mg/dL)	Yes	81	241.1 (5), 239 (212.5–271.5)	< 0.001*
	No	79	204.4 (4.6), 199 (180–225)	
LDL (mg/dL)	Yes	81	143.8 (3.8), 139 (116.5–167)	< 0.001∫
	No	79	117.5 (3.6), 114 (94–125)	
HDL (mg/dL)	Yes	81	45.7 (1.8), 42 (33–54)	0.4
	No	79	46.1 (1.4), 44 (37–55)	
TG (mg/dL)	Yes	81	161.2 (9), 144 (107.5–196.5)	< 0.001∫
	No	79	113.7 (6.6), 99 (76–134)	
TC/HDL	Yes	81	5.9 (2.2), 5.5 (4.2–7.1)	0.001∫
	No	79	4.8 (1.6), 4.7 (3.6–5.2)	
TG/HDL	Yes	81	4.1 (2.7), 3.5 (1.9–5.7)	<0.001∫
	No	79	2.8 (1.9), 2.3 (1.5–3.3)	
AST (IU/L)	Yes	81	41.4 (5.2), 29 (22–43)	0.1
	No	79	28.6 (1.1), 27 (23–30)	
ALT (IU/L)	Yes	81	74.4 (6.3), 57 (39.5–92)	0.002∫
	No	79	48.9 (1.9), 44 (35–59)	

SEM: Standard error of mean, IQR: Interquartile range, FPG: Fasting plasma glucose, PPG: Postprandial glucose, HbA1C: Glycated hemoglobin, TC: Total cholesterol, LDL: Low-density lipoprotein, HDL: High-density lipoprotein, TG: Triglycerides, AST: Aspartate aminotransferase, ALT: Alanine aminotransferase, NAFLD: Non-alcoholic fatty liver disease. *indicates significant difference between groups using independent sample t-test, ∫ indicates significant difference between groups using Mann– Whitney U-test,

significant difference was observed in TC ($P < 0.001$), LDL ($P < 0.001$), TG ($P < 0.001$), ALT ($P = 0.002$), TC/HDL ($P = 0.001$), and TG/HDL ($P < 0.001$). Significant difference was not observed in age ($P = 0.6$), FPG ($P = 0.9$), PPG ($P = 0.6$), HbA1C ($P = 0.9$), AST ($P = 0.1$), and HDL ($P = 0.3$) between cases and controls (Table 3). No association with NAFLD was observed for diabetes mellitus ($P = 0.2$), serum LDL ($P = 0.6$), and serum HDL ($P = 0.1$) (Tables 4-6 respectively).

Significant association with NAFLD was observed for serum TG ($P = 0.001$), serum TC ($P < 0.001$), and dyslipidemia ($P = 0.02$) (Tables 7-9 respectively).

No association with NAFLD was observed for TC/HDL ($P = 0.2$) and TG/HDL ($P = 0.07$) (Tables 10 and 11 respectively). We found a significant association between serum AST and NAFLD ($P = 0.03$), ALT and NAFLD ($P < 0.001$), more than twice the upper limit of normal of AST and NAFLD ($P = 0.004$), and more than twice the upper limit of normal of ALT and NAFLD ($P = 0.045$) (Tables 12-15 respectively).

DISCUSSION

The study participants were of considerably lower age (47 years) for individuals undergoing voluntary health checkup which could be due to exemplary health care policies of the state government contributing to the increased awareness regarding non-communicable diseases.[12] This could also be an indicator of the high burden of lifestyle disorders among Keralites forcing them to undergo health checkup in early life. A higher proportion of the study participants were males which

Table 4: Association between diabetes mellitus and NAFLD

Blood glucose level categorization[9]	NAFLD		Total
	Yes	No	
Normal	16	23	39
Prediabetes	35	24	59
Diabetes	30	32	62
Total	81	79	160

NAFLD: Non-alcoholic fatty liver disease. No association was observed between diabetes mellitus and NAFLD (*P*=0.2)

Table 5: Association between serum LDL and NAFLD

Serum LDL	NAFLD		Total
	Yes	No	
Elevated (≥70 mg/dL in diabetics, ≥130 mg/dL in nondiabetics)	38	34	72
Normal (<70 mg/dL in diabetics, <130 mg/dL in nondiabetics)	43	45	88
Total	81	79	160

LDL: Low-density lipoprotein, NAFLD: Non-alcoholic fatty liver disease. No association was observed between serum LDL and NAFLD (*P*=0.6; OR: 1.2, 95% CI: 0.6-2.2)

Table 6: Association between serum HDL and NAFLD

Serum HDL	NAFLD		Total
	Yes	No	
Reduced (<40 mg/dL in males or<50 mg/dL in females)	40	49	89
Normal (≥40 mg/dL in males or≥50 mg/dL in females)	41	30	71
Total	81	79	160

HDL: High-density lipoprotein, NAFLD: Non-alcoholic fatty liver disease. No association was observed between serum HDL and NAFLD (P=0.1; OR: 1.7, 95% CI: 0.9–3.1)

Table 7: Association between serum triglycerides and NAFLD

Serum triglycerides	NAFLD		Total
	Yes	No	
Elevated (≥130 mg/dL)	44	23	67
Normal (<130 mg/dL)	37	56	93
Total	81	79	160

NAFLD: Non-alcoholic fatty liver disease. There was significant association between serum triglycerides and NAFLD (*P*=0.001; OR: 2.9, 95% CI: 1.5–5.6 indicating 2.9 odds of encountering NAFLD in participants with elevated serum triglycerides)

Table 8: Association between serum TC and NAFLD

Serum TC	NAFLD		Total
	Yes	No	
Elevated (≥200 mg/dL)	68	39	107
Normal (<200 mg/dL)	13	40	53
Total	81	79	160

NAFLD: Non-alcoholic fatty liver disease, TC: total cholesterol. There was significant association between serum TC and NAFLD (*P*<0.001; OR: 5.4, 95% CI: 2.6-11.2 indicating 5.4 odds of encountering NAFLD in participants with elevated TC)

Table 9: Association between dyslipidemia and NAFLD

Dyslipidemia (elevated serum LDL and/reduced serum HDL)	NAFLD		Total
	Yes	No	
Yes	51	63	114
No	30	16	46
Total	81	79	160

LDL: Low-density lipoprotein, HDL: High-density lipoprotein, NAFLD: Non-alcoholic fatty liver disease. Significant association was observed between dyslipidemia and NAFLD (*P*=0.02; OR: 0.4, 95% CI: 0.2–0.9, indicating 0.4 Odds of encountering NAFLD in participants with dyslipidemia suggesting a protective effect of dyslipidemia)

Table 10: Association between TC HDL ratio and NAFLD

TC/HDL	NAFLD		Total
	Yes	No	
<4	19	25	44
≥4	62	64	116
Total	81	79	160

TC: Total cholesterol, HDL: High-density lipoprotein, NAFLD: Non-alcoholic fatty liver disease. No association between TC/HDL ratio and NAFLD was observed (*P*=0.2; OR: 1.5, 95% CI: 0.8–3)

Table 11. Association between TG HDL ratio and NAFLD

TG/HDL	NAFLD		Total
	Yes	No	
<2	21	31	52
≥2	60	48	108
Total	81	79	160

TG: Triglycerides, HDL: High-density lipoprotein, NAFLD: Non-alcoholic fatty liver disease. No association between TG/HDL ratio and NAFLD was observed (*P*=0.07; OR: 1.8, 95% CI: 0.9–3.6)

could be an indicator of lower number of female automobile users in Kerala making hospital access difficult for them or due to the lower proportion of independent females among Keralites[13] or due to an increased willingness among male participants to undergo voluntary health checkup. Plasma glucose parameters of the study participants were in normal range except HbA1C which was in the prediabetic range indicating the fraction of the overwhelming global pandemic diabetes, among Keralites. All serum cholesterol parameters were in the normal range except TC (~220 mg/dL) which could

Table 12: Association between serum AST and NAFLD

Serum AST	NAFLD		Total
	Yes	No	
Elevated (>upper limit of normal)	25	13	38
Normal (≤upper limit of normal)	56	66	122
Total	81	79	160

AST: Aspartate aminotransferase, NAFLD: Non-alcoholic fatty liver disease. Significant association was observed between serum AST and NAFLD (P=0.03; OR: 2.3, 95% CI: 1.1–4.8 indicating 2.3 odds of encountering NAFLD in participants with elevated serum AST)

Table 13: Association between serum ALT and NAFLD

Serum ALT	NAFLD		Total
	Yes	No	
Elevated (> upper limit of normal)	25	4	29
Normal (≤ upper limit of normal)	56	75	131
Total	81	79	160

ALT: Alanine aminotransferase, NAFLD: Non-alcoholic fatty liver disease. Significant association was observed between serum ALT and NAFLD (P<0.001; OR: 8.4, 95% CI: 2.8–25.4 indicating 8.4 odds of encountering NAFLD in participants with elevated serum ALT).

Table 14: Association between more than 2 times elevation of AST and NAFLD

AST elevation (>2 times upper limit of normal)	NAFLD		Total
	Yes	No	
Yes	8	0	8
No	73	79	152
Total	81	79	160

AST: Aspartate aminotransferase, NAFLD: Non-alcoholic fatty liver disease, significant association was observed between more than two times elevation of AST and NAFLD (P=0.004, OR could not be calculated as there was a group with no participants).

Table 15: Association between two fold elevation of ALT and NAFLD

ALT elevation (>2 times upper limit of normal)	NAFLD		Total
	Yes	No	
Yes	4	0	4
No	77	79	156
Total	81	79	160

ALT: Alanine aminotransferase, NAFLD: Non-alcoholic fatty liver disease. Significant association was observed between two fold elevation of ALT and NAFLD (P=0.045, OR could not be calculated as there was a group with no participants)

be attributable to the use of coconut oil which can produce elevation in TC.[14] Liver enzymes of the study participants were in the normal range.

Female participants were significantly older (P = 0.007) which could be due to the above-discussed reasons. Since the gender-based difference in prevalence of prediabetes, diabetes mellitus, and plasma glucose has not been reported,[15] the significantly higher FPG in male participants could be incidental. Significantly higher serum triglycerides among male participants could be due to the triglyceride catabolizing effect of estrogen in females.[16] The effects of estrogen such as elevation of Apo A1 levels, reduced expression of hepatic lipase, and HDL scavenger receptor class B type I[17] could be the reason for significantly higher HDL among female participants, and this also explains the significantly higher TC/HDL and TG/HDL among male participants. Liver enzymes were significantly higher among male participants, which could be due to a small but non-significantly higher number diabetic males. Diabetes and IR contribute to hepatic lipid accumulation and subsequent inflammation leading to elevation in liver enzymes.[18] Significantly higher TC (P < 0.001), LDL (P < 0.001), TG (P < 0.001), ALT (P = 0.002), TC/HDL (P = 0.001), and TG/HDL (P < 0.001) were observed among cases. Elevated TC[19-22] and elevated LDL[23] have been depicted as independent risk factors for the development of NAFLD. Elevated cholesterol levels cause accumulation of cholesterol intracellularly in the liver and hepatic blood vessels causing steatosis and inflammation.[24] IR leads to an inflammatory state in adipose tissues leading to lipolysis and release of TG into the systemic circulation which is taken up by the liver. When coupled with reduced export owing to defective VLDL formation in IR cause steatosis in hepatocytes.[25] Since IR is strongly associated with NAFLD, the elevated TG among cases can be explained on the basis of this. Significantly higher ALT was observed among cases, which is a typical finding in NALFD[26] ascribed to the inflammatory state in hepatocytes. Our study demonstrated significantly higher TC/HDL and TG/HDL in cases concurring to published literature suggesting an increased risk of NAFLD with higher TG/HDL and TC/HDL ratio.[10] This could be due to higher TC and TG or lower HDL in cases. There was no significant difference in other parameters though hyperglycemia and reduced HDL are commonly associated with NAFLD.[26] Contrary to the reports of strong association of NAFLD with prediabetes and diabetes mellitus,[27] we did not find any association (P = 0.2) which could be due to the high prevalence of prediabetes and diabetes mellitus among Keralites[15] making them extremely common among both cases and controls. No association between elevated LDL and NAFLD was observed, although a strong association has been reported.[23] This could be explained on the basis of the recent guidelines which makes the delineation of elevated LDL levels vague[28] and could also be due to the use of coconut oil among majority of Keralites which could cause elevation of serum LDL in both cases and controls.[14] Surprisingly, we did not find a significant association between reduced HDL and NAFLD which could be an indicator of the increasing sedentarism among Keralites reducing HDL levels among both cases and controls. A significant association between

triglycerides and NAFLD similar to previous reports[25,29] was observed. IR causes an inflammatory state in adipose tissue[30] and increases de novo synthesis of fatty acids by the liver[25] causing increased serum triglycerides, which are taken up by the liver. IR cause reduced export from liver and subsequently NAFLD. Our study also demonstrated a significant association between serum TC and NAFLD similar to the previous reports.[24] Increased serum cholesterol leads to deposition of lipids and cholesterol in hepatocytes and hepatic blood vessels triggering an early inflammatory response and causing rapid progression of the disease.[24] We considered elevated LDL and/or reduced HDL as dyslipidemia, and we found significant association between dyslipidemia and NAFLD which could indicate the effect of elevated LDL[31] or reduced HDL[32] or both on NAFLD. Since NAFLD involves inflammatory response in the liver, elevation of liver enzymes (AST and ALT) in NAFLD is an expected finding.[33] We did not find a significant association between TC/HDL, TG/HDL, and NAFLD.

Being a case–control study, the use of surrogate markers for diagnosing co-morbidities and large sample size are the strengths of the study. Lack of prospective follow-up to determine the progression of disease is a major limitation of our study. Further exploration is required to determine the exact association of these parameters with NAFLD in larger sample.

CONCLUSION

High prevalence of prediabetes and diabetes was seen in participants undergoing voluntary health checkup. A significant difference in TC, LDL, TG, TC/HDL, TG/HDL, and ALT was observed between cases and controls. Significant association with NAFLD was observed for TG, TC, dyslipidemia, AST and ALT.

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