Original Article

Tracking lipid profile and atherogenic indices in the prediabetics of Andaman Nicobar Islands: A retrospective hospital-based study

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Abstract

Context: Cardio vascular disease (CVD) is the leading cause of mortality and morbidity in diabetes mellitus (DM) contributing to 65% of all deaths with diabetic complications. The most important cause of CVD is atherosclerosis, and dyslipidemia acts as a marker of developing atherosclerosis. The derangement of lipid profile and atherogenic indices start in the prediabetic state, much before the development of DM. Detection of the deranged lipid profile and atherogenic indices in the prediabetic state can help devise the aggressive treatment strategy right from this stage, so as to arrest the development of CVD as a complication of diabetes. **Aims:** To compare the lipid profile and atherogenic indices of prediabetics with controls and diabetics. **Settings and Design:** The electronic medical records of 239 subjects were reviewed retrospectively. **Materials and Methods:** About 187 cases consisting of 137 diabetics and 50 prediabetics were evaluated for serum fasting blood sugar, post prandial blood sugar, HbA1c, total cholesterol (TC), triglycerides (TGs), high-density lipoprotein cholesterol (HDLc), and low-density lipoprotein cholesterol (LDLc). Atherogenic indices [TC/HDLc, LDLc/HDLc, (TC-HDLc)/HDLc, TG/HDLc] were also evaluated in the two groups. Rest 52 age- and sex-matched subjects were taken as controls. **Statistical Analysis Used:** The comparisons were evaluated using SPSS statistical package version 20. **Results:** TC, TG, LDLc, and the atherogenic indices were significantly increased in prediabetics as compared with controls. HDLc was significantly decreased in prediabetics. **Conclusions:** The altered lipid profile and atherogenic indices in prediabetics signify the increased susceptibility of prediabetics to CVD in the long run. Hence, we recommend screening of prediabetics for dyslipidemia to arrest the development of cardiovascular complications.

Keywords: Atherogenic indices, cardiovascular diseases, diabetics, lipid profile, prediabetics

Introduction

Diabetes mellitus (DM) is considered to be a global pandemic nowadays. The prevalence of diabetes worldwide stands at 8.3% as per the data recorded in 2013 and this magic figure is expected to reach 10.1% by 2035 of which 80% will be in the developing countries.[1] India stands out way ahead as the diabetic capital of the world with 69.1 million diabetics.[2] The prevalence of diabetes in many Indian cities stands in between 9% and 12% with a national average of 7.7% in 2016.[3] It is a slowly developing but progressive condition; and it can take many years to progress from prediabetic to diabetic state without interventions.[4]

The American Diabetes Association (ADA) defines prediabetes as impaired fasting glucose (100–125 mg/dL), impaired glucose tolerance (2-h glucose level of 140–199 mg/dL during an oral glucose tolerance test), or glycated hemoglobin (HbA1c) level of 5.7%–6.4% or both.[5] Many studies have reported that

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lifestyle modifications in the prediabetic stage can not only delay the development of DM by ≥10 years, but it can also prevent the development of the disease. This increases the quality of life and life expectancy and reduces the economic burden on the society.[6,7] Prevalence of prediabetes in India stands in between 10% and 14% as against the global prevalence of 8%.[8,9] Around 70% of the prediabetics eventually develop diabetes if not managed early.[10,11] The annual turnover rate being 5%–10%.

Cardio vascular disease (CVD) is the leading cause of mortality and morbidity in DM and according to some estimates it accounts for approximately 75%–80% of overall deaths with diabetic complications.[12] It has also been found to be significantly associated with prediabetes.[13,14] Among the causal factors of CVD, atherosclerosis is the foremost. Dyslipidemia, frequently occurring in diabetic patients, plays a critical role in the acceleration of macrovascular atherosclerosis and thus contribute to the excess risk of CVD.

Atherogenic ratios

Mere absence of a deranged lipid profile does not exclude the possibility of CVD. In 2008, Glasziou *et al.* in their study concluded that low-density lipoprotein cholesterol (LDLc)-based therapy leads to either undertreatment or overtreatment of patients with CVD. Therefore, it was assumed that markers other than LDLc such as non-high-density lipoprotein cholesterol (HDLc) and atherogenic indices have higher significance in cardiovascular risk management and identification of individuals at risk.[15]

The fatality of this silent killer/DM lies in the fact that it escapes detection or prewarning in its inceptive stage, and by the time it is detected, it has already started its damaging effects. With such a high prevalence of prediabetes in our country, the role of primary care physicians become very significant. The first level of contact with the physician can lead to the chance diagnosis of prediabetes as the stage is almost bereaved of symptoms. Due to the significant dyslipidemia and the resulting cardiovascular morbidity and mortality in cases of DM, diagnosis of this impending disease at the level of prediabetes and the state of lipid profile and other atherogenic indicators in the prediabetic stage will help devise the aggressive treatment strategy and management of such patients so as to nip the disease in its budding stage.

The aim of this study was to compare the lipid profile along with atherogenic indices (Castelli's risk index I, Atherogenic index, Atherogenic coefficient (AC), Surrogate marker of insulin resistance) of prediabetics with controls and diabetics. There are very few studies in India doing the same and is the first of its kind in the union territory of Andaman and Nicobar Islands.

Materials and Methods

The retrospective study reviewed the electronic medical records of 187 patients included over a six month period from February 2017 to July 2017 and whose blood samples were sent to the Department of Biochemistry for analysis. Patients with history of renal disorders, hypothyroidism, smokers, alcoholics, those on lipid lowering therapy, and hypoglycemic drugs were excluded from the study. About 187 patients were divided into two study groups: prediabetics and diabetics. The prediabetic group included the following criteria: fasting glucose (100–125 mg/dL), impaired glucose tolerance (2-h glucose level of 140–199 mg/dL after a standardized meal), and glycosylated hemoglobin (HbA1c) level of 5.7%–6.4%.

Diabetic group was selected as per ADA guidelines.[5] Diabetics with history of the disease for ≥1 year and not on any form of treatment or dietary management for the last 1 year are included in the study so as to get a fair picture of the effect of hyperglycemia on the lipid profile indices. Fifty-two age- and sex-matched controls were taken for the study.

Fasting venous blood samples were collected for the photometric estimation of lipid profile in CST 240 clinical chemistry analyser, Snibe diagnostics, China, using homogenous assay kits.[16-19] Fasting blood sugar (FBS), 2-h postprandial blood glucose (PPBG) after a standardized meal were estimated on venous blood samples, which were analyzed by spectrophotometry using Biolabo kits.[20] Samples for HbA1c were analyzed by immunoturbidimetric assay using Biolabo kits.[21] The Atherogenic ratios were calculated as follows:[22-25]

Castelli's Risk Index-I (CRI-I) = TC (Total cholesterol)/HDLc

Atherogenic Index (AI) = LDLc/HDLc

Atherogenic Coefficient (AC) = (TC–HDLc)/HDLc

Surrogate marker of Insulin resistance = TG (triglycerides)/HDLc

Westgard's rules were followed for monitoring the internal quality control on the analyzer. During the course of the study, there was no change in the equipment, reagents, calibration standards, and controls.

Statistical analysis

LDLc, very low density lipoprotein cholesterol, and CVD risk was calculated using appropriate formulae and continuous variables were expressed as mean ± standard deviation (SD). The normality of data was tested using Kolmogorov–Smirnov test and Shapiro Wilk test. The comparisons were evaluated by Kruskal Wallis test (nonparametric analysis of variance) using SPSS statistical package version 20. Mann–Whitney *U*-test was applied following statistically significant value to identify pairwise differences. The association between the groups and lipid profile was analyzed using Pearson's correlation analysis. A P value < 0.05 was considered statistically significant.

Results

In total, 187 cases were included in the study of which 137 (73%) were diabetics and 50 (26.7%) were prediabetics. Fifty-two

controls were also evaluated. Of the total cases, 86 (45.9%) were males and 101 (54.0%) were females as shown in Table 1. The mean age of diabetics was found to be 52.51 ± 10.73 years and that of prediabetics 52.90 ± 11.71 years. Interpreting it with the help of table IV, we find that there is no statistically significant difference in the age distribution of prediabetics, diabetics, and controls (*P* value >0.05) in the study population.

Table 2 shows the mean ± SD of FBS, 2-h PPBG and HbA1c in the study groups. A highly significant difference (*P*-value <0.01) is observed in the blood sugar indices (FBG, PPBG, HbA1c) when compared between the study groups. Table 3 summarizes the difference in the means of these parameters and their statistical significance.

Table 4 compares the lipid profile parameters along with the atherogenic ratios in the different study groups. Table 5 depicts the difference in the means of these parameters and their statistical significance. TC, TG, LDLc, and the different atherogenic ratios were significantly raised in the prediabetic group in comparison to the control group (*P*-value <0.001) except TG/HDLc.

Discussion

The prediabetics were diagnosed only on the presence of both the criteria as given by ADA for the same so that the lipid profile indices are a true reflection of prediabetic state. Second, we took cut off for normal fasting blood sugar <110 mg/dL, because in India, it is commonly used by the biochemists and clinicians. Diabetics who were not on treatment for the last ≥1 year are included in the study so as to get a fair picture of the effect of hyperglycaemia on the lipid profile indices.

The mean age of prediabetics and diabetics in our study is almost in coherence with the study by Bhatnagar MK *et al.*[26] Among the cases whose records were reviewed, 54.01% were females. This might be because of the high turnout of females in the OPD as compared with males as most of the females were housewives and could attend the OPD in the office hours.

Total cholesterol

The mean total cholesterol (TC) as found in our study in the prediabetic group was 223.64 ± 17.20 mg/dL which is significantly higher (*P*-value <0.01) than the control group (155.25 ± 15.67 mg/dL). Diabetics have serum TC of 287.09 ± 34.72 mg/dL, which, in turn, is significantly higher than both prediabetics and controls. Higher amount of glucose results in increased synthesis of pyruvate and then citrate in the Krebs cycle pathway. Citrate can move out of mitochondria and can form acetyl CoA in the cytosol. This acetyl CoA in case of glucose surplus state can be channeled to cholesterol synthesis. Ford *et al.* found that TC in prediabetics of 211 mg/dL and 196 mg/dL in a National Health and Nutrition Examination survey (NHANES), USA, in two successive periods (from 1988 to 1991 and 2005 to 2008, respectively) on age adjustment. The diabetics in this study were found to have a better lipid profile than prediabetes. This might be because most of the diabetics were on medications.[27] Kansal *et al.* in their study found mean TC of 184.75 ± 46.02 mg/dL in the prediabetic study group.[28] The difference in the observed value might be attributed to the stringent criteria of selection of prediabetic cases in our study.

Triglycerides

The mean serum TG in prediabetics as found in our study was 151.74 ± 33.14 mg/dL as compared with 107.23 ± 18.89 mg/dL in controls, which is significantly lower (*P*-value <0.01). The value for the same in case of diabetics stands at 219.77 ± 59.82 mg/dL, which is significantly higher than the prediabetics and controls (*P* value <0.01). The same value in NHANES study was found to be 137 mg/dL.[27] Similarly, Miyazaki *et al.* in their study observed raised TG levels in prediabetic subjects.[29] This might be due to the reduced activity of hepatic lipase and lipoprotein lipase, the clearing agent of Tg from the circulation, due to the decrease in activity of insulin. Also there's increased hepatic synthesis of VLDL due to the excess substrates available.[30]

LDLc

In our study, the mean total LDLc in prediabetics was significantly higher than the controls (152.19 ± 16.91 vs

Table 1: Demographic data (age and sex of the study groups)

		Number	Percentage	Average age in years (genderwise)	Age in years (mean±SD)
Control	Male	20	38.46	53.15	52.23±13.54
	Female	32	61.54	51.66	
Pre-Diabetic	Male	23	46.00	57.35	52.90±11.71
	Female	27	54.00	49.11	
Diabetic	Male	63	45.99	52.16	52.51±10.73
	Female	74	54.01	52.81	

Table 2: Glycemic indices (FBS, PPBS, and HbA1c) in the study groups

	Controls (C) Mean±SD	Prediabetic (P) Mean±SD	Diabetic (D) Mean±SD	Chi-square	*P*	Pairwise test of significance
FBS	92.52±8.16	120.06±1.67	171.25±64.34	112.7	<0.001	<0.001*
PPBS	122.58±9.11	186.54±3.77	262.42±75.74	148.7	<0.001	<0.001*
HbA1c	4.86±0.51	5.81±0.14	7.42±0.81	184.5	<0.001	<0.001*

PBS: Fasting blood sugar. *The mean difference is significant at 0.001 level among all the three groups

87.11 ± 15.77 mg/dL). Diabetics have a higher LDLc than the prediabetics and controls (*P* value <0.05). Shin *et al.* also found LDLc to be significantly higher in prediabetic subjects (150.5 ± 38 mg/dL) than nondiabetic controls (134 ± 34.6 mg/dL). They also proved that there was a correlation between raised blood glucose level and LDLc.[31] The same was found to be 138 and 118 mg/dL in the two successive periods in NHANES study.[27] The increase in glycemia causes glycation of apoprotein B lysine residues, thus making it unable to be recognized by LDL receptor and hence decreased catabolism of LDLc.[32]

HDLc

The prediabetic population in our study was found to have significantly lower serum HDLc than the controls (41.10 ± 3.07 vs 46.69 ± 4.80 mg/dL). In the same line, the mean HDLc in diabetics is still significantly lower than the other two study groups. Shin *et al.* also found significantly lower HDLc in prediabetic subjects (49.9 ± 11.6 mg/dL) as compared with nondiabetic controls (54.7 ± 13.3 mg/dL).[31] Increased VLDL as mentioned above is acted upon by cholesterol ester transfer protein to form small HDLc, which is easily cleared from the circulation. This leads to decrease in the serum level of HDLc and apoA1. Hence, increase in production of VLDL-TG ends in atherogenic reduction of HDL, intravascular remodeling, and reduced reverse cholesterol transport from peripheral tissues, hepatocytes and macrophages to liver, further aggravating atherosclerosis.[33]

Table 3: Multiple comparisons of glycemic indices (FBS, PPBS, and HbA1c) in the study groups

Dependent variable			Mean difference	Standard error	Significance
FBS	Diabetic	Prediabetic	51.188*	8.096	0.000
		Controls	78.729*	7.981	0.000
	Prediabetic	Controls	27.541**	9.705	0.014
PPBS	Diabetic	Prediabetic	75.883*	9.530	0.000
		Controls	139.846*	9.394	0.000
	Prediabetic	Controls	63.963*	11.424	0.000
A1C	Diabetic	Prediabetic	1.60644*	0.10892	0.000
		Controls	2.56467*	0.10737	0.000
	Prediabetic	Controls	0.95823*	0.13057	0.000

PBS: Fasting blood sugar. * The mean difference is significant at the 0.001 level. **The mean difference is significant at the 0.05 level

TG/HDLc

It is considered a surrogate marker of insulin resistance and hence an indicator of the cardio metabolic profile.[34] McLaughlin *et al.* in their study concluded that TG/HDL ratio >3.5 predicts insulin resistance.[33] We found a mean value of 3.71 ± 0.88 in prediabetics and 9.15 ± 3.89 in diabetics. Nayak *et al.* in their study on 83 prediabetic cases found the corresponding mean value to be 3.9.[35] Ozder *et al.* in their study found a value of 7.98 ± 3.8 mg/dL in the diabetic group.[34]

Atherogenic coefficient (TC-HDLc)/HDLc

Atherogenic coefficient is a measure of all lipoproteins that are considered to be atherogenic (VLDL, LDL, IDL, Lpa) with respect to good cholesterol or HDLc. It reflects the atherogenic potential of the entire spectrum of lipoprotein fractions. Non-HDLc has been considered as the second target of therapy after LDLc as per National Cholesterol Education Program Adult Treatment Panel III guidelines especially in individuals with hypertriglyceridemia.[36] It enjoys the same status as Apo-B in assessing atherogenic cholesterol and lipoprotein burden. Prediabetic group in our study recorded a mean value of >4.0, which is significantly higher than controls. Mahat *et al.* in their study on the prediabetic cases in the city of Gwalior, India, found the value to be 4.87 ± 0.87 vs 3.23 ± 0.50 in controls.[37] Ranjit *et al.* in their study, however, found a mean value of 4.62 ± 0.19 in coronary artery disease (CAD)-positive diabetic subjects.[38]

Castelli's risk index-I (TC/HDLc)

CRI-I has been considered as a secondary goal of therapy by the Canadian working group for the treatment of dyslipidemia, particularly in individuals with TG >300 mg/dL. It is a more sensitive and specific index of cardiovascular risk than the routine lipid profile parameters and hence a marker of atherogenic dyslipidemia.[39] The value was found to be >5 in prediabetics, which is significantly higher than the controls. The same was found to be 5.87 ± 0.57 in the prediabetic cases by Mahat *et al.*[37] Ranjit *et al.* found the value to be >5.6 in the CAD-positive diabetic dyslipidemic subjects.[38]

Atherogenic index (LDLc/HDLc)

In the PROCAM study, it was observed that subjects with LDL-c/HDL-c (CRI-II) >5 showed six times higher rate of

Table 4: Lipid profile and atherogenic indices in the study groups

	Controls (C) Mean±SD	Prediabetic (P) Mean±SD	Diabetic (D) Mean±SD	Chi-square	*P*	Pairwise test of significance
TC	155.25±15.67	223.64±17.20	287.09±34.72	186.9	<0.001	<0.001*
TGL	107.23±18.89	151.74±33.14	219.77±59.82	154.1	<0.001	<0.001*
HDL	46.69±4.80	41.10±3.07	25.58±4.31	181.2	<0.001	<0.001*
LDL	87.11±15.77	152.19±16.91	217.86±35.47	184.6	<0.001	<0.001*
VLDL	21.45±3.78	30.35±6.63	43.95±11.96	154.1	<0.001	<0.001*
TC/HDL	3.36±0.49	5.47±0.60	11.84±3.29	188.4	<0.001	<0.001*
LDL/HDL	1.89±0.45	3.73±0.54	9.01±2.84	188.4	<0.001	<0.001*
(TC-HDL)/HDL	2.36±0.49	4.47±0.60	10.84±3.29	188.4	<0.001	<0.001*
TG/HDL	2.32±0.50	3.71±0.88	9.15±3.89	183.8	<0.001	<0.001*

TC: Total cholesterol; HDL: High-density lipoprotein cholesterol; LDL: Low-density lipoprotein cholesterol; TG: Triglyceride. *The mean difference is significant at 0.001 level amongst all the three groups

Table 5: Multiple comparisons of lipid profile and atherogenic indices in the study groups

Dependent variable			Mean difference	Standard Error	Significance
TC	Diabetic	Prediabetic	63.448*	4.700	0.000
		Controls	131.838*	4.633	0.000
	Prediabetic	Controls	68.390*	5.634	0.000
TG	Diabetic	Prediabetic	68.034*	8.039	0.000
		Controls	112.543*	7.925	0.000
	Prediabetic	Controls	44.509*	9.637	0.000
HDL	Diabetic	Prediabetic	−15.8226*	0.6943	0.000
		Controls	−21.4149*	0.6844	0.000
	Prediabetic	Controls	−5.5923*	0.8323	0.000
LDL	Diabetic	Prediabetic	65.66347*	4.78278	0.000
		Controls	130.74394*	4.71491	0.000
	Prediabetic	Controls	65.08046*	5.73348	0.000
VLDL	Diabetic	Prediabetic	13.60674*	1.60774	0.000
		Controls	22.50859*	1.58493	0.000
	Prediabetic	Controls	8.90185*	1.92732	0.000
TC/HDL	Diabetic	Prediabetic	6.36647*	0.41700	0.000
		Controls	8.48027*	0.41109	0.000
	Prediabetic	Controls	2.11379*	0.49989	0.000
LDL/HDL	Diabetic	Prediabetic	5.27881*	0.36007	0.000
		Controls	7.11323*	0.35496	0.000
	Prediabetic	Controls	1.83442*	0.43165	0.000
(TC-HDL)/ HDL	Diabetic	Prediabetic	6.36647*	0.41700	0.000
		Controls	8.48027*	0.41109	0.000
	prediabetic	Controls	2.11379*	0.49989	0.000
TG/HDL	Diabetic	Prediabetic	5.44009*	0.49338	0.000
		Controls	6.83008*	0.48638	0.000
	Prediabetic	Controls	1.38999	0.59146	0.051

TC: Total cholesterol; HDL: High-density lipoprotein cholesterol; LDL: Low-density lipoprotein cholesterol; TG: Triglyceride. *The mean difference is significant at 0.001 level

coronary events.[40] AI >4.1 is considered as a risk factor for the development of CAD, whereas it indicates a protective effect when the value <4.1. In our study, the corresponding value for prediabetics was found to be 3.73 ± 0.54, which was significantly higher than the controls. It was found to be >9 in diabetics. However, in a study by Miyazeki *et al.*, no statistically significant difference in AI was observed between prediabetics and controls.[29]

Conclusion

TC, LDLc, TG, and VLDLc were significantly raised in prediabetics as compared with normal healthy subjects, whereas HDLc was decreased significantly in prediabetics. The raised atherogenic indices as discussed in the study point toward their increased susceptibility for cardiovascular complications. Hence, we recommend screening of prediabetics for dyslipidemia to arrest the development of early cardiovascular complications.

Limitations of the study

Patients with H/O hemoglobinopathies could not be ruled out due to the unavailability of the investigation in the hospital during the period.

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Conflicts of interest

There are no conflicts of interest.

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