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RESEARCH

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Association of angiotensin-converting enzyme I/D polymorphism and apolipoprotein B with cardiometabolic abnormalities among young adults: a pilot study from Delhi

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Abstract

Background Angiotensin-converting enzyme (ACE) gene polymorphism and elevated apolipoprotein B (apoB) are important risk factors for several cardiometabolic abnormalities. However, much less attention has been given to the relationship between these risk factors and cardiometabolic abnormalities among young adults. Considering this gap, the present study explored the association of ACE I/D polymorphism and apoB with cardiometabolic abnormalities among young adults of Delhi, India.

Methods This cross-sectional study was conducted among young adults (aged 18–30) of either sex residing in Delhi, India. A total of 330 individuals were invited to participate in the study, and data on the socio-demographic variables were collected using a pre-tested interview schedule. Somatometric and physiological measurements were obtained using standard protocols. However, blood sample collection and biochemical and genetic analyses could successfully be performed for 178 individuals. Fasting blood glucose (FBG), total cholesterol (TC), triglyceride (TG), high-density lipoprotein (HDL), and low-density lipoprotein (LDL) levels were estimated using Erba XL-640 biochemical analyzer. LDL and TG values were used to calculate apoB levels. Genotyping for ACE I/D polymorphism was performed by allele-specific PCR amplification followed by electrophoresis. Statistical analysis was done using SPSS v.20.

Results ACE I/D polymorphism was not found to be associated with hypertension, obesity, and abnormal FBG, TG, and HDL levels. However, DD and ID genotypes and D allele, with II as the reference genotype, significantly reduced the risk for high TC (OR, *p* value = 0.14, 0.01*; 0.29, 0.04*; 0.22, < 0.01*, respectively) and high LDL (OR, *p* value = 0.17, 0.03*; 0.20, 0.03*; 0.19, < 0.01*, respectively). Except for abnormal FBG, the prevalence of all the studied cardiometabolic abnormalities was significantly higher in the 4th quartile of apoB when compared to other quartiles. Linear regression model revealed a significant positive association of apoB levels with diastolic blood pressure, studied obesity parameters, TC, TG, and LDL levels.

Conclusion The D allele of ACE I/D polymorphism was not associated with most of the studied cardiometabolic abnormalities in the present study. Further, the association of high apoB with cardiometabolic abnormalities hints toward the importance of apoB in the early diagnosis of CVDs.

Keywords ACE I/D polymorphism, Hypertension, Obesity, Cardiometabolic risk factors, Apolipoprotein, Young adults

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Introduction

Cardiometabolic abnormalities are a cluster of health conditions that are strongly associated with an increased risk of developing cardiovascular diseases (CVDs) [1, 2]. It includes hypertension, excess weight and obesity, insulin resistance, hyperglycaemia, lipid abnormalities, etc. [1, 2]. The prevalence of cardiometabolic abnormalities has increased with the upsurge in hypertension, diabetes, and obesity [3–5]. India is estimated to have the second largest number of people with diabetes globally (74 million), and this is projected to increase to 125 million (nearly 70% increase) by 2045 [6]. In addition, obesity has also emerged as a major health concern in India, with prevalence ranging from 16.9 to 36.3% in different states [7]. Further, a sizable proportion of Indians are affected with hypertension and dyslipidemia [8, 9]. A more worrisome trend is the increasing prevalence of cardiometabolic abnormalities among young adults globally as well as in India [10–13].

Cardiometabolic abnormalities are multifactorial disorders that can be caused by a variety of environmental, genetic, and epigenetic factors [2, 14]. One of the most widely investigated genetic markers with respect to cardiometabolic abnormalities in general and hypertension, in particular, is insertion/deletion (I/D) polymorphism in the angiotensin-converting enzyme (ACE) gene [15, 16]. ACE is an essential component of the renin–angiotensin–aldosterone system (RAAS), an important pathway regulating blood pressure, fluid volume, and electrolyte balance [17]. The polymorphism in the ACE gene determines the serum ACE levels [17]. ACE converts inactive angiotensin I to active vasoconstrictor angiotensin II and, thereby, plays an important role in blood pressure regulation [18]. ACE also regulates blood pressure by degrading bradykinin, which is a vasodilator [17]. Individuals with mutant alleles of the ACE gene have been shown to be more vulnerable to cardiometabolic complications and, in turn, CVDs [15, 16].

Hypertension has been shown to be substantially associated with ACE gene polymorphism; however, there is an ongoing debate pertaining to the relationship of ACE gene polymorphism with other metabolic disorders such as diabetes, dyslipidemia, and obesity. Also, the relationship between ACE gene polymorphism and cardiometabolic abnormalities, including hypertension, has been chiefly explored among middle and old-aged individuals. Since an individual's age can modify the relationship between genotype and phenotype, understanding the role of ACE I/D polymorphism in cardiometabolic abnormalities among young adults is crucial.

Besides genetic polymorphisms, several blood biochemicals are also good predictors of cardiometabolic abnormalities. Elevated levels of apolipoprotein B (apoB), an essential structural protein component of all major atherogenic lipoproteins, have emerged as an important biomarker of cardiometabolic abnormalities and CVD risk [19]. The very-low-density lipoprotein (VLDL), intermediate-density lipoprotein (IDL), low-density lipoprotein (LDL), and lipoprotein(a) [Lp(a)] all contain one molecule of apoB per particle [20]. ApoB is a key component of all atherogenic particles. Thus, apoB is a good indicator of the number of atherogenic lipoprotein particles in circulation [20]. In fact, Apo B has been found to provide a better evaluation of on-treatment residual risk than LDL-C measurement [20].

Despite an expanding body of literature on apoB, much less attention has been given to exploring the importance of apoB as a biomarker for future CVD risk among young adults. Considering these gaps, the present study aims to understand the association of ACE I/D polymorphism and apoB with selected cardiometabolic abnormalities (hypertension, general and abdominal obesity, dyslipidemia in terms of high TC, TG, LDL, low HDL, and abnormal fasting blood glucose) among young adults of Delhi.

Materials and methods

Study design and participants

The present study is a cross-sectional study that was conducted among the college-going young adults of Delhi within the age group of 18–30. A total of 330 individuals of both sexes were conveniently invited to participate in the study. However, due to the lack of consent, blood sample collection and biochemical and genetic analyses could successfully be performed for 178 individuals (57.9% females). All the recruited participants were healthy (had no self-reported physical or mental illness). Individuals suffering from any terminal illness or on any long-term medication/micronutrient supplements were excluded from the study. Further, pregnant and lactating mothers were excluded from the study.

The study protocol was approved by the Departmental Ethics Committee, [*name of the institute has been omitted for peer review*] (Ref No. Anth/2022-23/526). A participant information sheet was handed out, and written consent was obtained from the participants prior to data collection.

Fieldwork and data collection

The fieldwork for the study was conducted from March to May 2022. Sociodemographic data (age, sex, education, and occupation) were collected using a pre-tested and modified interview schedule.

Somatometric, and physiological measurements and respective cut-offs

For the present study, somatometric measurements, namely weight (in kg), height (in cm), waist circumference (in cm), and hip circumference (in cm), were taken using standardized protocols. Weight was measured using calibrated weighing machine, height using an anthropometer rod, and waist and hip circumference using a measuring tape (Freemans). The body mass index (BMI) was calculated as weight (in kg) divided by the square of height (in meters), waist-hip ratio (WHR) as waist circumference (WC) (in cm) divided by hip circumference (HC) (in cm), and waist-height ratio (WHtR) as WC (in cm) divided by stature (in cm).

Overweight was defined as body mass index (BMI) $\geq$ 23.0 kg/m^2 but < 25.0 kg/m^2, while generalized obesity was defined as BMI $\geq$ 25.0 kg/m^2 and underweight as BMI < 18.0 kg/m^2 [21]. For various statistical analyses, underweight and normal weight categories have been merged as non-obese (n = 135) and overweight and obese categories as overweight/obese (n = 43). Abdominal obesity was defined as waist circumference (WC) $\geq$ 90 cm for men and $\geq$ 80 cm for women; waist-hip ratio (WHR) $\geq$ 0.90 for men and $\geq$ 0.80 for women, and waist-height ratio (WHtR) $\geq$ 0.5 [21, 22].

Blood pressure (BP) was taken in the sitting position on the left arm of the participants using a digital sphygmomanometer (OMRON). Three BP readings, each at a gap of five minutes, were taken, and the average of the three readings was considered to estimate the hypertension status. The categorization of participants into different categories of hypertension was done as per the American Heart Association guidelines (Stage II, SBP $\geq$ 140 mmHg or DBP $\geq$ 90 mmHg; Stage I, SBP 130–139 mmHg or DBP 80–89 mmHg; elevated, SBP 120–129 mmHg and DBP < 80 mmHg; normal, SBP < 120 mmHg and DBP < 80 mmHg) [23]. For various statistical analyses in this study, normal and elevated categories of BP have been merged as normotensives (n = 135), and stage-I and Stage-II categories as hypertensives (n = 43).

Blood collection

A sample of 5 ml fasting (~ 12 h) intravenous blood (2 ml in EDTA-coated vial for DNA extraction and 3 ml in a plain vial for biochemical analysis) was collected from each participant by a trained technician. The 2 ml blood collected in the EDTA-coated vial was subjected to the salting out process for DNA extraction [24]. Extracted DNA samples were stored at − 80 °C until further analysis. Serum was separated from the blood collected in the plain vial. Serum samples were subjected to biochemical analysis on the same day.

Biochemical markers and respective cut-offs

Biochemical markers included in the present study are fasting blood glucose (FBG), total cholesterol (TC), triglyceride (TG), high-density lipoprotein (HDL), low-density lipoprotein (LDL), very low-density lipoprotein (VLDL), and apolipoprotein B (apoB). Erba XL-640 biochemical analyzer and commercial Erba XL reagents were used to estimate FBG, TC, TG, HDL, and LDL. Further, LDL and TG values were used to estimate apoB levels using the formula apoB (in mg/dl) = − 33.12 + 0.675*LDL + 11.95*ln(TG) [25].

Dyslipidaemia was classified according to the National Cholesterol Education Program-Adult Treatment Panel III (NCEP-ATP III) guidelines [26]. Cut-offs are as follows: hypercholesterolemia–serum cholesterol levels $\geq$ 200 mg/dl ($\geq$ 5.2 mmol/l); hypertriglyceridemia–serum triglyceride levels $\geq$ 150 mg/dl ($\geq$ 1.7 mmol/l); low level of high-density lipoprotein (HDL) < 40 mg/dl (< 1.04 mmol/l) for men and < 50 mg/dl (< 1.3 mmol/l) for women; and high level of low-density lipoprotein (LDL) $\geq$ 130 mg/dl ($\geq$ 3.4 mmol/l) [26]. FBG level > 110 mg/dl was considered high blood glucose [27].

Genetic analysis and genotyping

Genotyping for ACE I/D polymorphism was performed using allele-specific PCR amplification, followed by agarose gel electrophoresis [28]. The flanking primer sequences, as reported by Rigat et al., were used; forward primer: CTG GAG ACC ACT CCC ATC CTT TCT; reverse primer: GAT GTG GCC ATC ACA TTC GTC AGAT [28]. The polymorphism was detected as a 490-bp fragment in the presence of the insertion (I) allele and as a 190-bp fragment in the presence of the deletion (D) allele.

Statistical analysis

Statistical analysis was done using IBM SPSS v.22 software program. Chi-square was applied to see the difference in the distribution of ACE I/D genotypes with respect to cardiometabolic abnormalities. Binary Logistic Regression was performed to calculate the odds ratio. Further, the participants were divided into quartiles based on apoB levels, and the prevalence of cardiometabolic abnormalities was determined in each quartile. Linear regression was computed to determine the strength of the association between the cardiometabolic variables and apoB levels. A p value < 0.05 was considered statistically significant, and a p value between 0.05–0.09 was considered suggestive.

Results

General characteristics of the participants

Of 178 participants, 103 (57.9%) were females, and the mean age of the sample was 21.75 years. All the recruited participants were college students studying at undergraduate ($n=83$), postgraduate ($n=70$), and M.Phil./Ph.D. levels ($n=25$). Among the participants, 43 (24.2%) were hypertensive, 43 (24.2%) were overweight/obese, 43 (24.1%) had abdominal obesity in terms of WC, 71 (39.9%) had high WHR, 58 (32.5%) had high WHtR, 14 (7.8%) had high TC, 42 (23.6%) had high TG, 31 (17.4%) had low HDL, 11 (6.1%) had high LDL and 19 (10.6%) had high FBG.

Distribution of genotypes of ACE I/D polymorphism with respect to cardiometabolic variables

In the present study, the ACE I/D genotype followed the Hardy–Weinberg equilibrium with frequencies of 20.7%, 44.4%, and 34.9% for ACE II, ID, and DD, respectively. Significant differences in the distribution of ACE I/D genotypes (II, ID, DD) were observed with respect to normal and abnormal levels of TC (p value = 0.01) and LDL (p value = 0.01), where a higher proportion of individuals with II genotype were found to have abnormal TC and LDL levels (Table 1). No significant difference in the distribution of ACE I/D genotypes was found for other studied cardiometabolic abnormalities.

Table 1 Distribution of genotypes and alleles of ACE I/D polymorphism with respect to cardiometabolic variables

Variables	ACE genotypes			*p* value	Allele frequency	
	II *n* (%)	ID *n* (%)	DD *n* (%)		I	D
BP						
Normotensive	29 (78.4)	59 (74.7)	47 (75.8)	0.91	0.43	0.57
Hypertensive	8 (21.6)	20 (25.3)	15 (24.2)		0.38	0.61
BMI						
Non-obese	27 (73.0)	61 (77.2)	47 (75.8)	0.88	0.40	0.60
Overweight/Obese	10 (27.0)	18 (22.8)	15 (24.2)		0.39	0.60
WC						
High	10 (27.0)	13 (16.5)	20 (32.3)	0.08	0.37	0.63
Normal	27 (73.0)	66 (83.5)	42 (67.7)		0.43	0.56
WHR						
High	14 (37.8)	29 (36.7)	28 (45.2)	0.57	0.38	0.61
Normal	23 (62.2)	50 (63.3)	34 (54.8)		0.43	0.56
WHtR						
High	12 (32.4)	20 (25.3)	26 (41.9)	0.11	0.36	0.63
Normal	25 (67.6)	59 (74.7)	36 (58.1)		0.43	0.56
TC						
Abnormal	7 (18.1)	5 (6.3)	2 (3.2)	0.01*	0.67	0.33
Normal	30 (81.1)	74 (93.7)	60 (96.8)		0.41	0.59
TG						
Abnormal	11 (29.7)	15 (19.0)	16 (25.8)	0.39	0.43	0.57
Normal	26 (70.3)	64 (81.0)	46 (74.2)		0.44	0.56
HDL						
Normal	9 (24.3)	12 (15.2)	10 (16.1)	0.45	0.48	0.52
Abnormal	28 (75.7)	67 (84.8)	52 (83.9)		0.42	0.58
LDL						
Abnormal	6 (16.2)	3 (3.8)	2 (3.2)	0.01*	0.68	0.32
Normal	31 (83.8)	76 (96.2)	60 (96.8)		0.41	0.59
FBG						
Abnormal	1 (2.7)	10 (12.7)	8 (12.9)	0.21	0.32	0.68
Normal	36 (97.3)	69 (87.3)	54 (87.1)		0.44	0.56

BP, blood pressure; BMI, body mass index; WC, waist circumference; WHR, waist-hip ratio; WHtR, waist height ratio; TC, total cholesterol; TG, triglyceride; HDL, high-density lipoprotein; LDL, low-density lipoprotein; FBG, fasting blood glucose. II, insertion homozygote; ID, heterozygote; DD, deletion homozygote

*Significance at p value ≤ 0.05

Odds ratio analysis

Odds ratio analysis revealed that the D allele both in the homozygote (DD) and heterozygote (ID) condition and in combination (ID + DD), with II as the reference genotype, significantly reduced the risk for abnormal TC and abnormal LDL (Fig. 1). Other cardiometabolic abnormalities (hypertension, general and abdominal obesity, impaired blood glucose levels, and abnormal TG and HDL levels) were not found to be associated with ACE I/D polymorphism (Fig. 1).

ApoB in cardiometabolic abnormalities

To understand the association of apoB cardiometabolic abnormalities, first, the distribution of cardiometabolic abnormalities was seen in various quartiles of apoB, then linear regression analysis with apoB as the independent variable and continuous cardiometabolic parameters as dependent variables was performed. The analysis showed that the prevalence of all the cardiometabolic abnormalities was significantly higher in the 4^{th} quartile of apoB when compared to the other quartile except for FBG (Table 2). Further, linear regression analysis revealed a significant positive association between apoB and all the studied cardiometabolic parameters, except for Systolic BP, HDL, and FBG, indicating an increase in cardiometabolic abnormalities with an increase in apoB (Table 2).

Discussion

In India and other South Asian nations, the prevalence of obesity and metabolic syndrome is rising quickly, leading to increased mortality and morbidity from CVDs [29]. One in three South Asians is thought to be affected by metabolic syndrome [30]. Additionally, it was shown that nearly 30% of Asian Indian children and adolescents had insulin resistance, and many of them showed signs of metabolic syndrome [31]. Since metabolic syndrome and obesity persist throughout adulthood [29, 30], it is crucial to identify these clinical conditions early in life in order to reduce the CVD risk. Cardiometabolic abnormalities have multifactorial aetiologies governed by both environmental and genetic factors. This study attempted to explore the association of ACE I/D polymorphism and apoB with cardiometabolic abnormalities among young adults of Delhi.

In the present study, ACE I/D polymorphism was not found to be significantly associated with any of the studied metabolic abnormalities (hypertension, general and abdominal obesity, impaired blood glucose levels, and abnormal TG and HDL levels). However, the D allele, with the II genotype as the reference, was found to reduce the risk of high TC and high LDL. These observations are in contrast to what has been widely reported [32, 33]. A vast majority of studies have reported the D allele of ACE I/D polymorphism to be associated with an increased risk of hypertension as well as other cardiometabolic abnormalities [32, 33]. The ACE I/D polymorphism is related to an increased level of plasma ACE activity wherein the DD genotype is known to have a higher ACE activity than the II genotype [17]. ACE is responsible for the conversion of inactive angiotensin I to active angiotensin II, which is a potent vasoconstrictor and also metabolizes bradykinin, a potent vasodilator, which contributes to the pathophysiology of atherosclerosis and other cardiovascular diseases [17, 18].

One of the reasons behind the lack of association between ACE I/D polymorphism and cardiometabolic abnormalities in the present study could be the young age of the present sample. While most studies exploring the relationship between ACE I/D polymorphism and cardiometabolic abnormalities have been conducted among middle and older-aged samples, the present study has been conducted among young adults. Although, the small sample size of the present study could also be a factor.

Coming to the relationship between apoB and cardiometabolic abnormalities, prevalence of studied cardiometabolic abnormalities, except impaired FBG, were found to be significantly higher in the fourth quartile of apoB, than in the other quartiles. Further, apoB was found to be positively associated with studied cardiometabolic

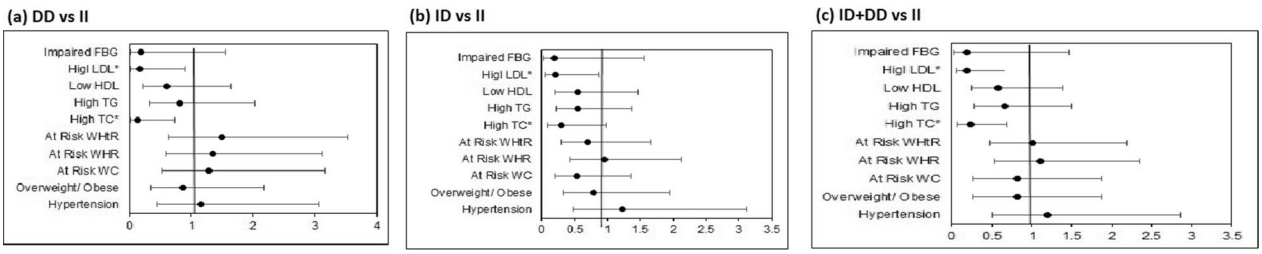

Fig. 1 Forest plot showing odds for cardiometabolic abnormalities in various genetic models **a** DD versus II, **b** ID versus II; ID + DD versus II. WC, waist circumference; WHR, waist-hip ratio; WHtR, waist height ratio; TC, total cholesterol; TG, triglyceride; HDL, high-density lipoprotein; LDL, low-density lipoprotein; FBG, fasting blood glucose. *Significance at *p* value ≤ 0.05

Table 2 Association of apoB with cardiometabolic abnormalities

Variable	Status	ApoB quartiles (in mg/dl)				*p* value	*β* (SE)	*p* value
		1st (< 72.0)	2nd (72.0–83.5)	3rd (83.5–98.8)	4th (≥ 98.8)			
BP	Normotensive	35 (79.55)	41(89.13)	33 (73.33)	25 (59.52)	0.01*	0.088 (0.056)[$]	0.12
	Hypertensive	9 (20.45)	5 (10.87)	12 (26.67)	17 (39.5)		0.106 (0.037)[&]	< 0.01*
BMI	Non-obese	38 (86.4)	40 (87.0)	32 (71.1)	24 (40.48)	< 0.01*	0.091 (0.016)	< 0.01*
	Overweight/Obese	6 (13.6)	6 (13.0)	13 (28.9)	18 (42.9)			
WC	High	5 (11.4)	8 (17.4)	13 (28.9)	17 (40.5)	< 0.01*	0.265 (0.049)	< 0.01*
	Normal	39 (88.6)	38 (82.6)	32 (71.1)	25 (59.5)			
WHR	High	10 (22.7)	17 (37.0)	23 (51.1)	21 (50.0)	0.02*	0.001 (0.000)	< 0.01*
	Normal	34 (77.3)	29 (63.0)	22 (48.9)	21 (50.0)			
WHtR	High	7 (15.9)	8 (17.4)	19 (42.2)	24 (57.1)	< 0.01*	0.002 (0.000)	< 0.01*
	Normal	37 (84.1)	38 (82.8)	26 (57.8)	18 (42.9)			
TC	Abnormal	0 (0.0)	0 (0.0)	1 (2.2)	13 (31.0)	< 0.01*	1.394 (0.060)	< 0.01*
	Normal	44 (100.0)	46 (100.0)	44 (97.8)	29 (69.0)			
TG	Abnormal	0 (0.0)	8 (17.4)	9 (20.0)	25 (59.5)	< 0.01*	1.717 (0.156)	< 0.01*
	Normal	44 (100.0)	38 (82.6)	36 (80.0)	17 (40.0)			
HDL	Abnormal	7 (15.9)	11 (23.9)	11 (24.4)	2 (4.8)	0.05*	0.023 (0.043)	0.60
	Normal	37 (84.1)	35 (76.1)	34 (75.6)	40 (95.2)			
LDL	Abnormal	0 (0.0)	0 (0.0)	0 (0.0)	11 (26.2)	< 0.01*	1.236 (0.020)	< 0.01*
	Normal	44(100.0)	46 (100.0)	45 (100.0)	31 (73.8)			
FBG	Abnormal	2 (4.5)	6 (13.0)	7 (15.6)	4 (9.5)	0.36	0.055 (0.045)	0.22
	Normal	42 (95.5)	40 (87.0)	38 (84.4)	38 (90.5)			

BP, blood pressure; BMI, body mass index; WC, waist circumference; WHR, waist hip ratio; WHtR, waist height ratio; TC, total cholesterol; TG, triglyceride; HDL, high-density lipoprotein; LDL, low-density lipoprotein; FBG, fasting blood glucose; apoB, apolipoprotein B; *β*, beta coefficient; SE, standard error

[$] Systolic blood pressure

[&] Diastolic blood pressure

*Significance at *p* value ≤ 0.05

variables (DBP, BMI, WC, WHR, WHtR, TC, TG, HDL, and LDL). These observations are in concordance with previous reports [19, 34, 35]. ApoB, a key structural protein component of all major atherogenic lipoproteins, plays an essential role in regulating lipid metabolism and is considered to be a physiologically relevant measure of the actual number of atherogenic lipid particles [36]. Recent studies have shown elevated apoB to be associated with an increased risk of cardiovascular diseases [36]. The present study reveals the importance of apoB in predicting cardiometabolic abnormalities among young adults. Currently, apoB is not a widely used biomarker for CVDs in India, especially among young adults, who are considered to be a low-risk population group. However, the present study hints toward the importance of apoB in the early diagnosis of cardiometabolic abnormalities. More studies should be taken to further explain the relationship between apoB and cardiometabolic abnormalities among young adults so that lipoprotein profiles can be considered for health risk assessment.

One of the major limitations of this study is the small sample size. The results of the present study should be validated on a larger sample size. Further, since the study was a cross-sectional study, the causal relationship between apoB and the studied cardiometabolic abnormalities could not be established.

Conclusion

The D allele of ACE I/D polymorphism was not found to be associated with most of the studied cardiometabolic abnormalities in the present study. If fact, the D allele may be protective against abnormal total cholesterol and abnormal low-density lipoprotein among young adults. Further, the study suggests a positive association between higher levels apoB and cardiometabolic abnormalities. This study hints toward the importance of apoB in the early diagnosis of cardiometabolic abnormalities. ApoB is not among the routine test for CVD risk in several low and middle-income countries, including India; however, the strong association of apoB with cardiometabolic abnormalities suggests that apoB should be included in routine tests for young adults.

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Author contributions
SS participated in data collection, laboratory analyses, data analyses, and writing—original draft. VC participated in data collection, laboratory analyses, data analyses, and writing—review & editing. SM participated in data collection, and laboratory analyses. BM participated in study design, methodology, and data interpretation. NKD participated in study design, methodology, data interpretation, and writing—review & editing. All authors approved the final version of the manuscript and agreed to be accountable for all aspects of the work.

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Availability of data and materials
The raw data will be made available by the corresponding author on request.

Declarations

Ethics approval and consent to participate
The study was approved by the Departmental Ethics Committee, Department of Anthropology, University of Delhi (Ref No. Anth/2022-23/526). Informed written consent was obtained from all participants prior to recruitment.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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