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Obese adolescents in central India: genetic insights into early risk of diabetes and cardiovascular disease

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Obesity in adolescents is a growing public health concern, contributing to early onset of cardiovascular disease (CVD) and diabetes. The present study evaluated anthropometric indices [body mass index (BMI), waist circumference (WC), hip circumference (HC), neck circumference (NC), waist-to-hip ratio (WHR), waist-to-height ratio (WHtR)] in relation to lipid and hormonal profiles [triglycerides (TG), total cholesterol (TC), low-density lipoprotein (LDL), very low-density lipoprotein (VLDL), high-density lipoprotein (HDL), HbA1c]. Genetic polymorphisms including CAPN10 rs3792267, KCNJ11 rs5219, PPAR-γ Pro12Ala, and variants in apolipoproteins (ApoA1, ApoE, ApoB, ApoCIII) were analysed to assess their contribution to obesity and metabolic risk. Significant associations were observed between anthropometric measures and lipid parameters, underlining the predictive value of simple indices for dyslipidaemia in adolescents. However, no correlation was found between HbA1c levels and obesity. Furthermore, genetic polymorphisms studied did not show significant association with obesity susceptibility or insulin secretion. Although apolipoprotein gene variants contributed to understanding lipid metabolism, no significant differences in genotype frequencies were detected between cases and controls. These findings highlight the importance of early identification of lipid abnormalities in obese adolescents, while suggesting a limited role of the selected genetic variants in this cohort.

Keywords: Obesity, adolescents, cardiovascular disease, diabetes, lipid profile, genetic polymorphism

Introduction

India has the largest adolescent population in the world, a developmental stage marked by rapid physical, psychological and social changes. Global prevalence of overweight and obesity in children increased from 4.2% in 1990 to 6.7% in 2010, with projections of 9.1% (60 million) by 2020[1]. In Africa, prevalence was expected to rise from 8.5% in 2010 to 12.7% in 2020, whereas Asia reported higher absolute numbers of affected children[2]. In India, prevalence of overweight in school children rose from 4.9% in 2003 to 6.6% in 2005, reflecting a rapid epidemiological transition[3,4]. Regional studies have shown marked heterogeneity, with rates influenced by socioeconomic status, urbanization and dietary patterns[5-9]. For instance, obesity prevalence was 5.6% in children from higher socioeconomic strata compared to 0.4% in lower strata[5]. Childhood obesity adversely affects cardiovascular (CV) health by worsening plasma lipids, blood pressure, glucose tolerance and systemic inflammation[10]. It increases risk of hypertension, coronary heart disease (CHD), heart failure and atrial fibrillation[11]. Although some studies describe an "obesity paradox," where obese patients with existing CV disease show better survival compared to lean counterparts[12-15], obesity remains a major driver of cardiometabolic risk in adolescents. Despite extensive data in adults, limited information is available regarding the genetic basis of obesity in Indian adolescents. Understanding the interplay between anthropometric indices, biochemical markers and genetic polymorphisms is important for identifying at-risk individuals. The present study aimed to investigate anthropometric, biochemical and genetic predictors of obesity-related CVD and diabetes risk in adolescents from Central India.

Materials and Methods

Study design and setting

The present case–control study was carried out at the Multidisciplinary Research Unit, Shyam Shah

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Medical College (SSMC), Rewa, Madhya Pradesh. Sampling was undertaken from the Medicine and Pediatrics outpatient departments of Sanjay Gandhi Memorial Hospital (SGMH), SSMC Rewa, and from schools in the districts of Rewa, Sidhi and Mauganj (Madhya Pradesh).

Subjects

Obese adolescents aged 10–19 yr were included as cases after obtaining written informed consent. Age- and sex-matched healthy, non-obese individuals served as controls. Subjects were excluded if they had a history of chronic disease, were under treatment or medication, had body mass index (BMI) ≤18 kg/m², were receiving lipid-lowering drugs, or had liver disease. These criteria were adopted to ensure safety of participants and reduce potential confounding.

Ethical considerations

The study protocol and informed consent forms were approved by the Institutional Ethics Committee, SSMC Rewa. Written informed consent was obtained from all participants (and parents/guardians, in case of minors) before enrolment.

Anthropometric measurements

Anthropometric indicators measured included weight, BMI, waist circumference (WC), hip circumference (HC), waist-to-hip ratio (WHR) and waist-to-height ratio (WHtR). Weight was measured with a digital weighing machine, with participants standing erect, barefoot, in light clothing, and without additional items. The weighing scale was set to zero before each measurement. Waist, hip and height were measured using a non-stretchable tape with the subject in standing position and head in Frankfort plane. Participants were classified using standard adult BMI cut-offs: normal weight (18.0–24.9 kg/m²), overweight (25.0–29.9 kg/m²), and obese (≥30.0 kg/m²).

Visceral Adiposity Index

The Visceral Adiposity Index (VAI), an established indicator of visceral fat dysfunction and cardiometabolic risk. VAI was calculated using the sex-specific formula proposed by Amato *et al.*[16] which utilizes waist circumference, BMI, triglycerides, and HDL-cholesterol.

Biochemical Analysis

Fasting venous blood samples were collected, and serum was separated by centrifugation at 3,000 rpm for 10 minutes. The lipid parameters, triglycerides (TG), total cholesterol (TC), and high-density lipoprotein cholesterol (HDL-C) were estimated using an automated biochemistry analyser (Mindray BS-430). TG and TC were measured by standard enzymatic colorimetric methods (GPO-PAP and CHOD-PAP), while HDL-C was estimated using a phosphotungstate–Mg^{2+} precipitation method. All assays were performed using commercially available reagent kits following the manufacturer's instructions. VLDL-C and LDL-C were calculated using the Friedewald formula [VLDL = TG/5; LDL = TC – (HDL + VLDL)].

DNA isolation and genotyping

Genomic DNA was extracted from whole blood using ReliaPrep™ Blood gDNA Miniprep System (Promega, USA). DNA concentration and purity were checked with Nanodrop spectrophotometer (OD 260/280 ratio 1.7–1.9). Genotyping of all SNPs was performed following PCR-based amplification and restriction enzyme digestion as described in the published literature. Specific primer sets, PCR conditions and restriction enzymes were selected according to previously published methods for each polymorphism (KCNJ11 rs5219[17], CAPN10 rs3792267[18], PPAR-γ Pro12Ala[19], ApoA1 G-75A and C+83T[20], ApoE rs429358 and rs7412[21], ApoB C7673T[22] and ApoCIII 3238C>G[23]). PCR amplification was carried out in a thermal cycler under optimized cycling conditions for each locus. The amplified products were subjected to restriction fragment length polymorphism (RFLP) analysis using the allele-specific restriction enzymes described in the original reference protocols. Digested fragments were separated on 2–3% agarose gel stained with ethidium bromide, and genotypes were assigned based on the characteristic restriction patterns under UV transillumination.

Statistical analysis

Data were compiled in Microsoft Excel 2007 and analysed using GraphPad Prism software (version 4.00, GraphPad, San Diego, USA). Descriptive statistics were presented as mean ± standard deviation (SD) or percentages. Group differences in means were tested using Student's *t*-test, while proportions were compared by Chi-square test (with Yates' correction where required). Spearman's correlation coefficient was applied to assess relationships between variables. Allele frequencies were compared by Chi-square test

with 1 degree of freedom and two-tailed *P* values. A $P < 0.05$ was considered statistically significant.

Results

A total of 340 subjects were enrolled in the study, of which 47.29% were males and 52.70% females. Based on body mass index (BMI), participants were categorized into overweight/obese (OW/O, n=170) and normal weight (NW, n=170) groups as per the inclusion criteria described in *Materials and Methods*.

Anthropometric measurements

The mean BMI in adolescents was significantly higher in OW/O group (28.83 ± 2.70) than in NW group (21.77 ± 3.94). Overweight males recorded a mean BMI of 29.73 ± 3.39 compared to 20.83 ± 3.92 in NW males ($P<0.005$), whereas overweight females had a mean BMI of 27.71 ± 4.67 compared to 20.69 ± 3.92 in NW females. Between genders, OW/O males showed higher BMI than OW/O females (29.73 ± 3.39 vs. 27.71 ± 4.67). Mean waist circumference (WC) was significantly higher in OW/O adolescents (92.29 ± 14.81) as compared to NW subjects (79.88 ± 9.70; $P<0.005$). Overweight males showed WC of 92.61 ± 15.68 versus 81.67 ± 10.56 in NW males, and overweight females had WC of 91.89 ± 13.79 compared to 78.63 ± 8.83 in NW females. Hip circumference (HC) was also higher in OW/O adolescents (98.37 ± 9.77) compared to NW counterparts (89.28 ± 10.70; $P<0.005$). Overweight males showed HC of 96.72 ± 7.40 compared to 89.28 ± 10.70 in NW males, while overweight females had HC of 100.40 ± 12.31 versus 87.84 ± 9.46 in NW females. Neck circumference (NC) was marginally higher in OW/O group (34.41 ± 6.13) compared to NW group (34.42 ± 7.43), though not statistically significant. Mean NC in overweight males was 35.09 ± 6.25 compared to 34.13 ± 4.70 in NW males; in females, OW/O group had NC of 33.58 ± 5.91 versus 32.97 ± 8.69 in NW group. Waist–hip ratio (WHR) did not show significant difference between groups. OW/O adolescents recorded WHR of 0.98 ± 0.57 versus 0.92 ± 0.11 in NW group. Overweight males and females had WHR of 0.95 ± 0.14 and 0.91 ± 0.05, respectively, compared to 0.94 ± 0.15 and 0.89 ± 0.08 in NW counterparts. Waist–height ratio (WHtR) was significantly higher in OW/O adolescents (0.59 ± 0.10) compared to NW group (0.50 ± 0.08; $P<0.05$). Overweight males recorded 0.58 ± 0.11 versus 0.50 ± 0.07 in NW males, while overweight females had 0.60 ± 0.09 compared to 0.51 ± 0.06 in NW females.

Visceral adiposity index (VAI)

VAI was calculated using sex-specific equations incorporating waist circumference, BMI, triglyceride levels, and HDL-cholesterol. Overweight/obese adolescents demonstrated markedly elevated VAI values (mean 2.14 ± 0.84) compared to normal-weight participants (0.98 ± 0.41), indicating greater visceral fat dysfunction. VAI showed a positive correlation with BMI (r=0.32), waist circumference (r=0.35), and triglycerides (r=0.41), and a negative correlation with HDL-C (r=–0.33). These trends indicate that adolescents with higher visceral adiposity also exhibit more pronounced lipid abnormalities, particularly hypertriglyceridemia and reduced HDL cholesterol. Collectively, these findings demonstrate that VAI is a sensitive marker of adipose tissue dysfunction in adolescents and aligns closely with traditional anthropometric and biochemical markers of cardiometabolic risk. Correlation of VAI with anthropometric and lipid parameters and Visceral Adiposity Index (VAI) and related parameters in study participants are given in (Table 1 & 2). Association between Visceral Adiposity Index (VAI) and key metabolic parameters are given in (Fig 1).

Biochemical parameters

Triglyceride (TG) levels were significantly elevated in OW/O adolescents (168.92 ± 11.02)

Table 1 — Visceral Adiposity Index (VAI) in Study Participants

Variable	Mean ± SD
VAI (overall)	1.83 ± 0.92
VAI (Overweight/Obese)	2.14 ± 0.84
VAI (Normal Weight)	0.98 ± 0.41

Table 2 — Correlation of VAI with Anthropometric and Lipid Parameters

Variable	BMI	WC	TG	HDL	VAI
VAI	0.049	0.142	0.347	–0.332	1.000
BMI	1.000	0.302	0.107	–0.187	0.049
WC	0.302	1.000	–0.010	–0.158	0.142
TG	0.107	–0.010	1.000	–0.010	0.347
HDL	–0.187	–0.158	–0.010	1.000	

[VAI strongly ↑ with Triglycerides (r = 0.347);VAI strongly ↓ with HDL-C (r = –0.332);VAI modest ↑ with Waist Circumference (r = 0.142)]

compared to NW group (127.15 ± 12.86; P<0.005). Overweight males had TG of 182.87 ± 7.44 versus 134.43 ± 15.92 in NW males; overweight females recorded 151.59 ± 12.83 compared to 121.78 ± 14.11 in NW females. Total cholesterol (TC) was also higher in OW/O adolescents (156.40 ± 16.09) than NW group (138.98 ± 33.33; P<0.05). Overweight males recorded 159.57 ± 36.29 versus 134.83 ± 26.82 in NW males, while overweight females had 152.47 ± 35.71 compared to 142.27 ± 37.09 in NW females. Low-density lipoprotein (LDL) levels were significantly raised in OW/O adolescents (91.21 ± 32.75) as compared to NW group (75.29 ± 26.11; P<0.05). Overweight males had LDL of 88.76 ± 38.63 versus 75.29 ± 26.11 in NW males, while overweight females recorded 94.24 ± 23.43 compared to 79.64 ± 28.21 in NW females.Very low-density lipoprotein (VLDL) was higher in OW/O adolescents (33.78 ± 13.20) compared to NW group (26.70 ± 11.01; P<0.05). Mean VLDL in overweight males was 33.57 ± 15.49 versus 30.43 ± 26.11 in NW males, while overweight females had 30.32 ± 8.57 compared to 24.07 ± 9.16 in NW females. High-density lipoprotein (HDL) was significantly lower in OW/O adolescents (38.19 ± 13.74) compared to NW group (45.31 ± 17.41; P<0.05). Overweight males showed HDL of 38.50 ± 13.49 versus 46.30 ± 16.31 in NW males, while overweight females had 37.81 ± 14.14 compared to 44.67 ± 18.12 in NW females. HbA1c levels did not differ significantly between OW/O and NW adolescents (P=0.324). However, overweight females showed a slight elevation (5.6 ± 0.3) compared to NW females (5.4 ± 0.4). Correlation analysis of anthropometric indices with lipid parameters is presented in (Table 3 & Table 4). Correlation of BMI with lipid fractions is depicted in (Fig 2), while that of waist circumference with lipid fractions is shown in (Fig 3).

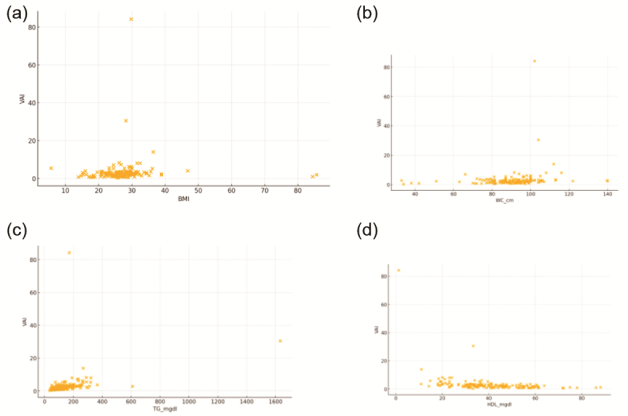

Fig. 1 — Scatter plots showing the association between Visceral Adiposity Index (VAI) and key metabolic parameters.

Table 3 — Anthropometric and biochemical parameters in overweight/obese and normal weight adolescents

Anthropometric Parameter	TG(mg/dL)	TC (mg/dL)	LDL(mg/dL)	HDL(mg/dL)	VLDL(mg/dL)
BMI	r=0.46,P<0.01	r=0.38,P<0.05	r=0.42,P<0.05	r=0.39,P<0.05	r=0.41,P<0.05
Waist Circumference(WC)	r=0.44,P<0.01	r=0.36,P<0.05	r=0.39,P<0.05	r=0.37,P<0.05	r=0.40,P<0.05
Hip Circumference(HC)	r=0.35,P<0.05	r=0.30,P<0.05	r=0.31,P<0.05	r=0.28,P<0.05	r=0.29,P<0.05
Neck Circumference(NC)	r=0.25,P<0.05	r=0.20,P<0.05	r=0.22,P<0.05	r=0.19,P<0.05	r=0.21,P<0.05
WHR	r=0.18,P<0.05	r=0.15,P<0.05	r=0.16,P<0.05	r=0.12,P<0.05	r=0.14,P<0.05
WHtR	r=0.48,P<0.01	r=0.40,P<0.05	r=0.43,P<0.05	r=0.41,P<0.05	r=0.44,P<0.05

Table 4 — correlation of anthropometric indices with lipid parameters in adolescent (Value expressed as correlation coefficient (r). Significant correlation marked at P<0.05 or P<0.01)

Parameter	Over weight/Obese(n=170)	Normal-Weight(n=170)	P value
BMI(kg/$_m^2$)	28.83± 2.70	21.77±3.94	<0.005
Wc(cm)	92.29± 14.81	79.88±9.70	<0.005
HC(cm)	98.37±9.77	89.28±10.70	<0.005
NC(cm)	34.41±6.13	34.42±7.43	NS
WHR	0.98±0.	0.92±0.11	NS
WHtR	0.59±0.10	0.50±0.08	<0.05
TG(mg/dL)	168.92±11.02	127.15±12.86	<0.005
TC(mg/dL)	156.40±16.09	138.98±33.33	<0.05
LDL(mg/dL)	91.21±32.75	75.29±26.11	<0.05
VLDL(mg/dL)	33.78±13.20	26.70±11.01	<0.05
HDL(mg/dL)	38.19±13.74	45.31±17.41	<0.05
HbA1c (%)	5.6±0.3	5.4±0.4	NS

(Value expressed as mean±SD. NS= Not significant)

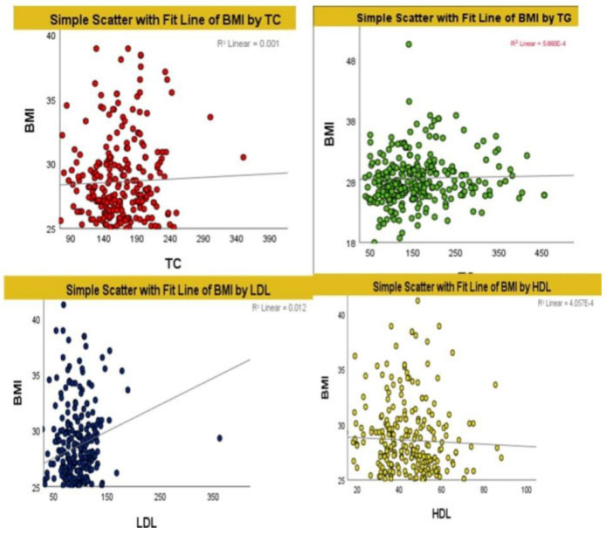

Fig. 2 — Scatter plots showing significant correlation between BMI and four lipid components (BMI in kg/m^2, Lipid component in mg/dl)

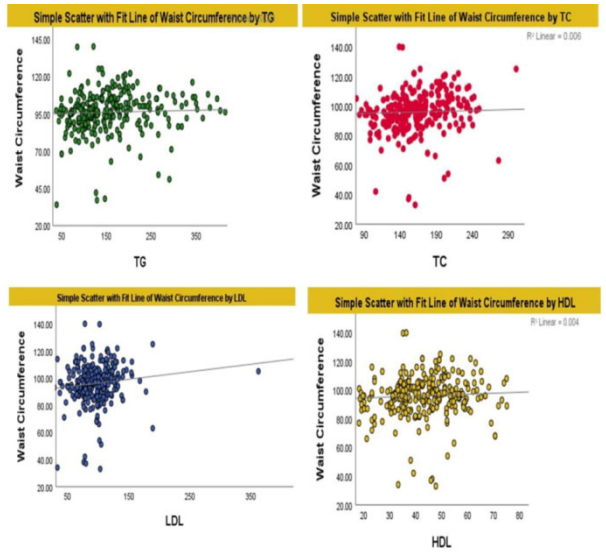

Fig. 3 — Scatter plots showing a significant correlation between WC and four lipid components (WC in cm, lipid component in md/dl)

Genetic study

The analysis of genotype and allele distributions across the studied polymorphisms revealed only marginal differences between obese adolescent diabetes cases and healthy controls. For KCNJ11 (rs5219)**,** the GG genotype was most prevalent among cases (54.7%), followed by GA (43%) and AA (2.35%), whereas controls showed a slightly higher proportion of GG (62.35%) and GA (37.64%) with no AA genotype detected; correspondingly, the G allele frequency was somewhat lower in cases (76.17%) compared to controls (81.17%), while the A allele was higher in cases (23.82% vs. 18.82%). In CAPN10 (rs3792267)**,** genotype frequencies in cases were 63% GG, 31% GA, and 1% AA, closely mirroring the controls, who showed 67% GG, 30% GA, and 3% AA, with high G allele frequencies in both groups (78.5% in cases and 81% in controls) and no statistically significant difference (p=0.352), indicating an absence of association with diabetes risk. For PPAR-γ (Pro12Ala)**,** cases predominantly exhibited the CC genotype (96%), with very low frequencies of CG (3%) and GG (1%), similar to controls where CC accounted for 95% and CG for 5%, with no GG observed; the G allele frequency showed negligible variation (97.1% in cases vs. 97.4% in controls). In the ApoA1 G-75A variant, nearly all cases were GG (99.5%), with only one individual showing the GA genotype (0.5%), while controls were entirely GG. For the ApoA1 C+83T mutation, all cases presented with the CC genotype (100%), whereas controls had 97% CC and 3% CT, resulting in the T allele being absent among cases but present at a very low frequency (1.5%) in controls. ApoE genotyping revealed a uniform E3/E3 phenotype across both groups (100%), with no detection of E2 or E4 alleles. In ApoB (C7673T)**,** cases were overwhelmingly CC (97.64%) with a small proportion of CT (2.35%) and no TT genotype, while controls were exclusively CC; the T allele was rare in cases (1%) and absent in controls. For ApoCIII (C-3238G)**,** the genotype distribution in cases (97% CC and 3% CG) was again comparable to controls (98% CC and 2% CG), with a slightly elevated G allele frequency in cases (2.5%) relative to controls (1.01%). Overall, the polymorphisms assessed showed no substantial or statistically meaningful differences between cases and controls, indicating no strong genetic association of these variants with diabetes risk in obese adolescents.

Discussion

The present study examined the association of anthropometric indices of obesity with lipid parameters and selected genetic polymorphisms implicated in metabolic and cardiovascular risk. Spearman correlation analysis revealed weak but significant positive correlations between BMI and total cholesterol (P=0.317, P<0.05), triglycerides (P=0.436, P<0.05), and LDL-C (P=0.397, P=0.125), along with a significant negative correlation with HDL-C (P=−0.236, P<0.001). Among the

anthropometric indices, waist circumference (WC) and waist–hip ratio (WHR) were more strongly correlated with lipid parameters than hip circumference (HC), which showed only weak associations. These findings support earlier evidence that abdominal adiposity is more predictive of cardiometabolic risk than generalized obesity. Visceral Adiposity Index (VAI) provided an additional perspective on visceral fat dysfunction in the study population. Notably, overweight/obese adolescents demonstrated markedly elevated VAI values, reflecting greater visceral adiposity despite similar chronological age. The positive correlations of VAI with BMI, waist circumference, and triglycerides, along with its inverse relationship with HDL-C, align with established evidence linking VAI to insulin resistance and cardiovascular risk. Genotyping analysis of KCNJ11 rs5219 showed predominance of the GG genotype in both cases and controls, with comparable allele frequencies (G allele: 76.17% in cases vs. 81.17% in controls). No significant association with obesity-related diabetes was observed.This result is in line with reports from the South Indian population[17] but differs from studies by Qin *et al.*[24], Qiu et al.[25] and Rizvi et al.[26] who demonstrated significant associations with T2D in their cohorts. For CAPN10 rs3792267**,** the GG genotype was most frequent in both cases (63%) and controls (67%), and no significant association was detected (P=0.352). Our findings agree with studies by Horikawa et al.[18] and Weedon et al.[27] although results have varied across different populations. Analysis of PPAR-γ Pro12Ala revealed a very low frequency of the G (Ala) allele in both groups (2.01% in cases; 2.9% in controls), with the CC genotype being predominant. In contrast, studies in Caucasian populations[19,28] have reported G allele frequencies of 12–15% and demonstrated protective associations against T2D and obesity. The discrepancy underscores the influence of ethnicity on allele distribution. The ApoA1 G-75A polymorphism was nearly absent in our cohort, with only one heterozygous individual in cases (0.5%) and complete absence in controls. This contrasts with reports from North Indian[20] and Indian (Assam)[29] populations where the A allele has been reported with higher frequency, and in some populations has been associated with altered lipid profiles or cardiovascular disease. Similarly, the ApoA1 C+83T polymorphism showed only a few heterozygotes in controls (3%), with complete absence in cases.Our findings are comparable to those reported in North Indian[20] and Assam populations[29], though with even lower T allele frequency. For ApoE polymorphism, only the E3/E3 genotype was observed in both groups (100%), with complete absence of E2 and E4 alleles. Global studies consistently report ApoE allele diversity and E4 frequencies[30,31]. The absence of E4 may reflect a protective ethnic profile in this central Indian cohort. The ApoB –C7673T mutation revealed a predominance of the CC genotype in both cases (97.64%) and controls (100%), with the T allele being rare (1% in cases, absent in controls). These frequencies differ from previous ApoB association studies[22,32], where higher frequencies have been reported in several populations. The ApoCIII C-3238G variant also showed predominance of the C allele in our population (97.5% in cases; 98.99% in controls), with a very low G allele frequency. These findings are comparable to previous studies of ApoCIII polymorphisms[23], although the G allele frequency was comparatively lower in our study. Overall, the study highlights the importance of combining anthropometric indices (BMI, WC, HC, NC, WHR, WHtR) with lipid and genetic profiling to assess obesity-related risks. The low prevalence or absence of several risk-associated alleles in this central Indian population underscores the role of ethnic variation in genetic susceptibility. This work strengthens evidence that abdominal obesity, rather than general adiposity, drives dyslipidemia and cardiometabolic risk. However, the modest sample size and restriction to a single regional population are limitations. Future research with larger, multi-ethnic cohorts and functional genomic approaches will be essential to clarify the biological relevance of these polymorphisms in obesity and related disorders.

Limitations

The present study was limited by its modest sample size and single-centre design, which may restrict the generalizability of the findings.

Conclusion

Significant correlations between anthropometric indices and lipid profiles in adolescents underscore their value as predictors of dyslipidemia and cardiovascular risk. Gender-specific variations indicate the need for individualized clinical assessment. Analysis of *CAPN10 rs3792267, KCNJ11 rs5219, PPAR-γ Pro12Ala, ApoA1, ApoE, ApoB* and *ApoCIII p*olymorphisms revealed population-specific

distributions, though no consistent associations with obesity or diabetes were observed. These findings highlight the multifactorial nature of adolescent obesity and its related metabolic complications. Integration of anthropometric, biochemical and genetic parameters offers a comprehensive framework for early identification of at-risk adolescents and may aid in developing preventive and therapeutic strategies to mitigate long-term cardiometabolic risk.

Future Directions

Larger multi-ethnic and longitudinal studies are required to validate these observations and to elucidate gene–environment interactions influencing obesity and its sequelae. Incorporation of genetic profiling into routine screening may enable early detection and tailored interventions, ultimately improving clinical outcomes and quality of life in obese adolescents.

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Conflict of interest

The authors declare no conflict of interest.

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