ORIGINAL ARTICLE

Dyslipidaemia in Patients with Acne Vulgaris: A Hospital-Based Case-Control Study

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ABSTRACT

Introduction: Acne vulgaris is a prevalent dermatological condition, with emerging evidence suggesting its association with metabolic comorbidities. **Aims:** To evaluate the serum lipid profile in patients with acne vulgaris, compare it with controls, and assess its correlation with disease severity and duration. **Methods:** A hospital-based case-control study was conducted, enrolling 100 patients with acne vulgaris and 100 healthy controls. Serum total cholesterol, triglycerides, high-density lipoprotein cholesterol, and low-density lipoprotein cholesterol were assessed. Acne severity was evaluated using the Global Acne Grading System. Statistical analyses employed the Mann-Whitney U test, Kruskal-Wallis test with post-hoc analysis, and Spearman rank correlation. **Results:** The cases and controls exhibited comparable sex distribution ($p = 0.109$). Serum total cholesterol (168.0 vs. 152.8 mg/dL, $p = 0.0002$) and triglycerides (148.0 vs. 116.1 mg/dL, $p < 0.001$) were significantly elevated in cases compared to controls. Low-density lipoprotein cholesterol was significantly lower in cases (78.0 vs. 86.2 mg/dL, $p = 0.040$), while high-density lipoprotein cholesterol was significantly elevated (52.0 vs. 38.2 mg/dL, $p < 0.001$). Total cholesterol, triglycerides, and low-density lipoprotein cholesterol increased progressively with acne severity (all $p < 0.001$), with the severe group differing significantly from milder groups. Both disease duration and acne severity correlated positively with total cholesterol, triglycerides, and low-density lipoprotein cholesterol; acne severity showed an inverse correlation with high-density lipoprotein cholesterol. **Conclusion:** Acne vulgaris is associated with a dyslipidemic pattern characterized by elevated total cholesterol and triglycerides, dependent on both disease severity and duration. Routine lipid screening may be advisable, particularly for patients presenting with moderate-to-severe manifestations of the disease.

Keywords: Acne vulgaris; Cholesterol; Dyslipidaemia; Global Acne Grading System; Lipid profile; Triglycerides

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INTRODUCTION

Acne vulgaris is a chronic inflammatory condition of the pilosebaceous unit, affecting approximately 9.4% of the worldwide population and 85% of young adults aged 12 to 25 years.[1,2] A recent Global Burden of Disease analysis indicated a 39.2% increase in prevalent cases among adolescents from 1990 to 2021.[3] In Nepal, acne vulgaris represents a significant portion of dermatology outpatient consultations; Dhakal et al reported a prevalence of 18.96% in a multicenter study, predominantly among adolescents.[4] The pathogenesis of acne vulgaris involves excessive sebum production, follicular hyperkeratinization, colonization by Cutibacterium acnes, and inflammation.[5] The sebaceous gland is pivotal in this process, as it secretes, a complex composition of triglycerides, wax esters, and cholesterol esters. Alteration in lipid metabolism, both within the sebaceous gland and in the systemic circulation, have been implicated in the pathogenesis of the disease.[6] As cholesterol is a precursor for androgen biosynthesis, elevated circulating cholesterol may enhance androgenic activity, thereby exacerbating acne.[7] Furthermore, signaling molecules such as Insulin-like growth factor-1 (IGF-1) and mechanistic target of rapamycin complex 1 (mTORC1) further stimulate lipogenesis via sterol regulatory element-binding protein 1 (SREBP1), linking systemic lipid metabolism to acne pathogenesis.[8] Numerous case-control studies have explored serum lipid profiles in patients with acne vulgaris. A 2023 study by Mohammed et al identified significantly elevated levels of total cholesterol, tri-

glycerides, and low-density lipoprotein cholesterol in acne patients compared to controls, with triglyceride levels positively correlating with disease severity.[9] Similarly, a clinicobiochemical study from India reported significant elevations in these parameters in moderate-to-severe acne, exhibiting a severity-dependent pattern.[10] These findings are supported by a recent meta-analysis examining lipid alterations in acne vulgaris patients.[7] However, data from Nepal remain sparse, and no hospital-based study has examined this association in a Nepalese cohort.

METHODS

This study employed a hospital-based case-control design and was conducted in the Department of Dermatology and Venereology at Nepalgunj Medical College and Teaching Hospital, Banke, Nepal, from July 2025 to May 2026. Ethical approval was obtained from the Institutional Review Committee (IRC) of Nepalgunj Medical College and Teaching Hospital, Banke (Approval No. 76 / 082- 083). Written informed consent was obtained from all participants, and for those under 18 years of age, assent was obtained alongside parental or guardian consent. The study adhered to the principles outlined in the Declaration of Helsinki.

A total of 100 cases and 100 controls were enrolled in the study. The cases comprised patients aged 13 to 25 years who presented to the Dermatology outpatient department with a clinical diagnosis of acne vulgaris. The controls were sex-matched healthy individuals within the same age range (13 to 25 years) who had no history of acne vulgaris and were attending the hospital for routine health check-ups. Both groups were recruited from the same hospital catchment area to ensure comparability in socioeconomic and dietary backgrounds.

Inclusion criteria for cases include: A clinical diagnosis of acne vulgaris confirmed by a qualified dermatologist; an age range of 13 to 25 years; and a willingness to participate, demonstrated by providing written informed consent (or assent with parental or guardian consent for minors). Inclusion criteria for controls comprise: healthy individuals within the same age range (13 to 25 years); sex-matched with the case group; no personal or family history of acne vulgaris; and no known lipid disorder or dyslipidemia.

Exclusion criteria applicable to both cohorts include: A diagnosis of diabetes mellitus, hypertension, thyroid disorder, polycystic ovarian syndrome (PCOS), or any other systemic illness; current pregnancy or lactation; the use of oral contraceptives, systemic corticosteroids, isotretinoin, lipid-lowering agents, or any medication known to affect lipid metabolism within the past three months; obesity, defined as a body mass index (BMI) of 30 kg/m² or greater; alcohol consumption or tobacco use; and refusal to participate or provide written informed consent.

The sample size was calculated using the formula pertinent to case-control studies, incorporating assumptions of a 95% confidence interval, 80% statistical power, and an expected prevalence of dyslipidaemia among acne patients as documented in prior research. A minimum of 100 cases and 100 controls was considered sufficient to detect a clinically significant difference in lipid parameters between the two groups. The diagnosis of acne vulgaris was conducted through clinical evaluation, identifying characteristic lesions such as comedones, papules, pustules, nodules, and/or cysts, predominantly appearing on the face, chest, and back. The Global Acne Grading System (GAGS) was employed to assess the severity of the condition, assigning a weighted score based on the type and distribution of lesions across six distinct facial and truncal regions. Severity was categorized into mild (score 1–18), moderate (score 19–30), and severe (score 31–38) levels. The duration of the disease was defined as the number of months from the initial onset of acne to the time of enrolment, as reported by the patient.

Following a minimum 12-hour overnight fast, five millilitres of venous blood were collected from each participant under sterile conditions. The samples were subsequently centrifuged at 3000 rpm for 10 minutes to separate the serum, which was analyzed within two hours using an automated biochemistry analyzer. The serum lipid parameters assessed included: Total cholesterol (TC) - enzymatic colorimetric method, Triglycerides (TG) - enzymatic colorimetric method, High-density lipoprotein cholesterol (HDL-C) — direct enzymatic method, Low-density lipoprotein cholesterol (LDL-C) — direct enzymatic method. Dyslipidemia was identified according to the NCEP ATP III criteria as follows: TC ≥ 200 mg/dL, TG ≥ 150 mg/dL, LDL-C ≥ 130 mg/dL, or HDL-C < 40 mg/dL in males and < 50 mg/dL in females.

Statistical Analysis

Data were analyzed utilizing IBM SPSS Statistics version 25. The Shapiro-Wilk test was employed to evaluate normality. Given that most lipid variables exhibited non-normal distribution in at least one group, results are presented as medians with interquartile ranges (IQR). Categorical variables are displayed as frequencies with percentages. The Mann-Whitney U test was applied for continuous comparisons between groups, while the Chi-square test was used for categorical comparisons. Differences in lipid levels across severity groups were assessed using the Kruskal-Wallis test, followed by Bonferroni-corrected pairwise comparisons (adjusted threshold: p < 0.017). Spearman rank correlation was utilized to examine associations between lipids and both disease duration and acne severity (coded: mild=1, moderate=2, severe=3). A p-value < 0.05 was considered statistically significant.

RESULTS

1. Baseline Characteristics

A total of 100 cases and 100 controls were enrolled in the study. Among the cases, 68 (68.0%) were female and 32 (32.0%) were male, while among the controls, 56 (56.0%) were female and 44 (44.0%) were male. The sex distribution was comparable between the two groups ($\chi^2 = 2.568$, df = 1, p = 0.109). The median age of the cases was 19.0 years (IQR: 6.0; range 13–25), which was significantly lower than that of the controls, whose median age was 25.0 years (IQR: 1.0; range 13–25) (U = 1215.5, p < 0.001). Among the cases, the severity of the condition was classified as mild in 39 (39.0%), moderate in 42 (42.0%), and severe in 19 (19.0%), with a median disease duration of 9.0 months (IQR: 9.0; range 2–18 months). Baseline characteristics are detailed in Table I.

Variable	Cases (n=100)	Controls (n=100)	p-value
Sex, n (%)			0.109[a]
Female	68 (68.0)	56 (56.0)	
Male	32 (32.0)	44 (44.0)	
Age (years), Median (IQR)	19.0 (6.0)	25.0 (1.0)	<0.001[b]
Acne Severity, n (%)			
Mild	39 (39.0)	—	—
Moderate	42 (42.0)	—	—
Severe	19 (19.0)	—	—
Disease Duration (months), Median (IQR)	9.0 (9.0)	—	—

[a]Chi-square test. [b]Mann-Whitney U test. Data as n (%) or Median (IQR). IQR = interquartile range.

Table I: Baseline Characteristics of Cases and Controls

2. Assessment of Normality

The Shapiro-Wilk test showed non-normal distribution of TC (p = 0.002), TG (p = 0.001), and HDL-C (p = 0.003) in cases; and TG (p = 0.001), HDL-C (p = 0.002), and LDL-C (p = 0.010) in controls. Non-parametric tests were therefore used throughout.

3. Comparison of Serum Lipid Profile between Cases and Controls

Comparative analysis of serum lipid levels, as presented in Table II and Figure 1, revealed significantly elevated total cholesterol (TC) in the case group [168.0 mg/dL (IQR: 35.0)] compared to the control group [152.8 mg/dL (IQR: 45.7)] (U = 6504.0, p = 0.0002). Additionally, triglyceride (TG) levels were markedly higher in cases [148.0 mg/dL (IQR: 24.0)] than in controls [116.1 mg/dL (IQR: 84.3)] (U = 6778.0, p < 0.001). High-density lipoprotein cholesterol (HDL-C) was also significantly elevated in cases [52.0 mg/dL (IQR: 11.0)] relative to controls [38.2 mg/dL (IQR: 10.7)] (U = 8599.5, p < 0.001). Conversely, low-density lipoprotein cholesterol (LDL-C) was significantly lower in cases [78.0 mg/dL (IQR: 17.0)] compared to controls [86.2 mg/dL (IQR: 37.5)] (U = 4157.5, p = 0.040).

Lipid (mg/dL)	Cases Median (IQR)	Controls Median (IQR)	U statistic	p-value
TC	168.0 (35.0)	152.8 (45.7)	6504.0	0.0002
TG	148.0 (24.0)	116.1 (84.3)	6778.0	<0.001
HDL-C	52.0 (11.0)	38.2 (10.7)	8599.5	<0.001
LDL-C	78.0 (17.0)	86.2 (37.5)	4157.5	0.040

Median (IQR). Mann-Whitney U test. TC = total cholesterol; TG = triglycerides; HDL-C = high-density lipoprotein cholesterol; LDL-C = low-density lipoprotein cholesterol.

Table II: Comparison of Serum Lipid Profile between Cases and Controls

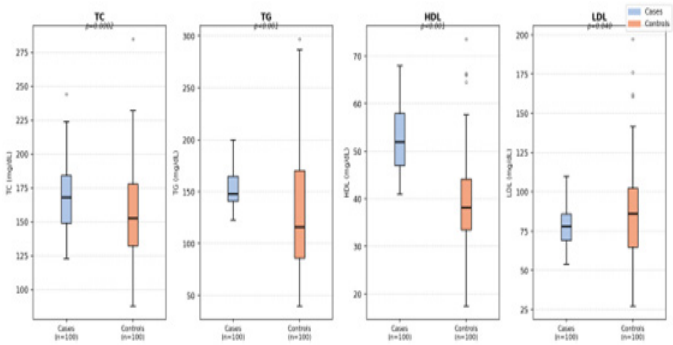

Figure 1: Box plots comparing serum lipid profiles between acne vulgaris cases (n=100, blue) and controls (n=100, orange). Horizontal lines indicate medians; boxes represent IQR; whiskers extend to 1.5×IQR. p-values from Mann-Whitney U test

4. Serum Lipid Profile by Acne Severity

The Kruskal-Wallis test (Table III) revealed significant inter-group differences for total cholesterol (TC) (H = 27.41, p < 0.001), triglycerides (TG) (H = 26.32, p < 0.001), and low-density lipoprotein cholesterol (LDL-C) (H = 17.39, p < 0.001). However, high-density lipoprotein cholesterol (HDL-C) did not achieve statistical significance (H = 5.40, p = 0.067). The TC levels increased progressively across groups: mild at 157.0 mg/dL (IQR: 23.5), moderate at 168.0 mg/dL (IQR: 29.8), and severe at 196.0 mg/dL (IQR: 25.0). Similarly, TG levels were recorded as follows: mild at 143.0 mg/dL (IQR: 15.0), moderate at 148.0 mg/dL (IQR: 22.5), and severe at 175.0 mg/dL (IQR: 18.0). For LDL-C, the values were mild at 76.0 mg/dL (IQR: 12.5), moderate at 77.0 mg/dL (IQR: 16.0), and severe at 86.0 mg/dL (IQR: 9.0).

Lipid (mg/dL)	Mild (n=39) Median (IQR)	Moderate (n=42) Median (IQR)	Severe (n=19) Median (IQR)	H	p-value
TC	157.0 (23.5)	168.0 (29.8)	196.0 (25.0)	27.41	<0.001
TG	143.0 (15.0)	148.0 (22.5)	175.0 (18.0)	26.32	<0.001
HDL-C	54.0 (9.5)	51.5 (9.8)	48.0 (12.0)	5.40	0.067
LDL-C	76.0 (12.5)	77.0 (16.0)	86.0 (9.0)	17.39	<0.001

Median (IQR). H = Kruskal-Wallis H statistic. NS = not significant

Table III: Serum Lipid Profile across Acne Severity Groups (Kruskal-Wallis Test)

4a. Post-hoc Pairwise Comparisons (Bonferroni-corrected)

The post-hoc analysis, as presented in Table IIIb, revealed statistically significant differences in total cholesterol (TC), triglycerides (TG), and low-density lipoprotein cholesterol (LDL-C) levels between the severe group and both milder groups, with all results being Bonferroni-corrected (p < 0.017). However, no significant differences were observed between the mild and

moderate groups for any lipid parameter.

Lipid (mg/dL)	Mild vs Moderate p-adj	Mild vs Severe p-adj	Moderate vs Severe p-adj	Pattern
TC	0.523 (NS)	<0.001***	<0.001***	Severe > Mild & Moderate
TG	0.307 (NS)	<0.001***	<0.001***	Severe > Mild & Moderate
HDL-C	0.869 (NS)	0.082 (NS)	0.313 (NS)	NS across all groups
LDL-C	0.596 (NS)	<0.001***	0.003**	Severe > Mild & Moderate

p-values adjusted by Bonferroni correction (×3 comparisons). NS = not significant. ** p < 0.01; *** p < 0.001

Table III(b): Post-hoc Pairwise Comparisons by Acne Severity (Bonferroni-corrected)

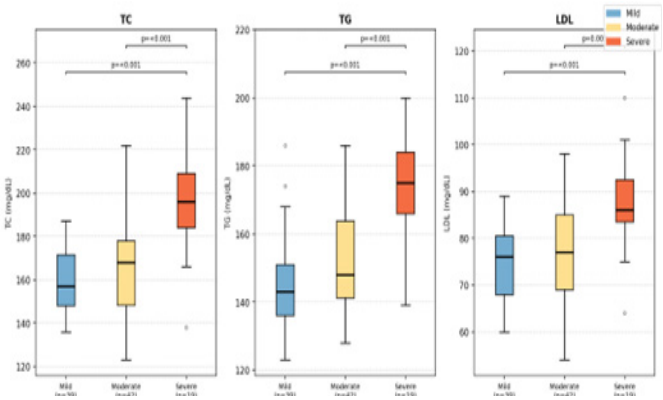

Figure 2: Box plots of TC, TG, and LDL-C by acne severity (Mild n=39, Moderate n=42, Severe n=19). Significance brackets show Bonferroni-corrected post-hoc p-values. HDL-C omitted (KW p = 0.067, NS)

5. Correlation of Serum Lipids with Disease Duration and Acne Severity

The duration of the disease exhibited a significant correlation with total cholesterol (TC) (rs = 0.446, p < 0.001), triglycerides (TG) (rs = 0.339, p = 0.001), and low-density lipoprotein cholesterol (LDL-C) (rs = 0.243, p = 0.015), while no significant correlation was observed with high-density lipoprotein cholesterol (HDL-C) (rs = −0.099, p = 0.326). Furthermore, the severity of acne demonstrated a positive correlation with TC (rs = 0.458, p < 0.001), TG (rs = 0.463, p < 0.001), and LDL-C (rs = 0.377, p < 0.001), and an inverse correlation with HDL-C (rs = −0.223, p = 0.026) (Table IV, Figure 3).

Lipid (mg/dL)	rs (Duration)	p (Duration)	rs (Severity)	p (Severity)	Interpretation
TC	0.446	<0.001	0.458	<0.001	Significant positive; both
TG	0.339	0.001	0.463	<0.001	Significant positive; both
HDL-C	-0.099	0.326	-0.223	0.026	Negative; significant with severity only
LDL-C	0.243	0.015	0.377	<0.001	Significant positive; both

rs = Spearman rank correlation coefficient. Duration in months. Severity: mild=1, moderate=2, severe=3

Table IV: Spearman Rank Correlations of Serum Lipids with Disease Duration and Acne Severity

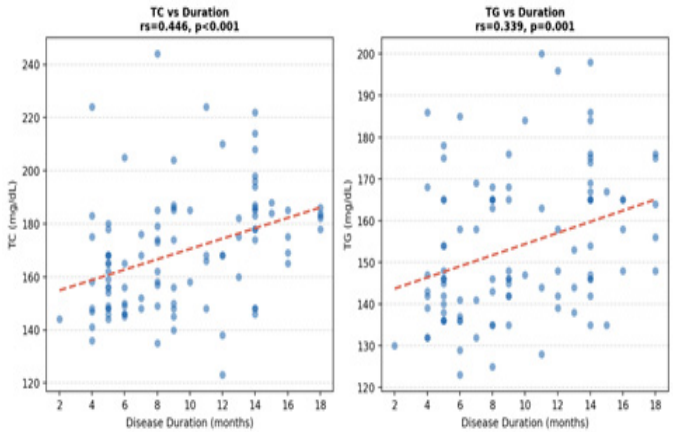

Figure 3: Scatter plots with Spearman correlation between disease duration (months) and TC (left, rs=0.446, p<0.001) and TG (right, rs=0.339, p=0.001) in cases (n=100). Dashed lines = linear trend

DISCUSSION

This hospital-based case-control study indicates that acne vulgaris is correlated with a dyslipidemic lipid profile. Serum total cholesterol (TC) levels (168.0 vs. 152.8 mg/dL, p = 0.0002) and triglycerides (TG) (148.0 vs. 116.1 mg/dL, p < 0.001) were significantly elevated in the cases, aligning with findings from Mohammed et al[9], a 2025 cross-sectional study conducted in Iraq,[11] and a 2025 meta-analysis encompassing 38 studies (n=2,485).[7] The mechanistic explanation involves cholesterol's role as a necessary precursor for androgen biosynthesis, with elevated androgen levels promoting sebaceous gland overactivity.[5] Additionally, the IGF-1/mTORC1/SREBP1 signaling pathway further enhances lipogenesis.[6]

A progressive increase in total cholesterol (TC), triglycerides (TG), and low-density lipoprotein cholesterol (LDL-C) was observed in relation to the severity of the condition (Kruskal-Wallis H = 27.41, 26.32, 17.39; all p < 0.001), with the severe group exhibiting significant differences from both milder groups in post-hoc analysis. This gradient aligns with the findings of Mohammed et al (p = 0.037 for TC in severe versus milder acne),[9] Mosa et al (elevated non-HDL-C with severity),[11] and a study based on the Global Acne Grading System

(GAGS) conducted in Bangladesh.[12] Additionally, disease duration showed a positive correlation with TC (rs=0.446), TG (rs=0.339), and LDL-C (rs=0.243), a finding of particular regional importance given the lack of comparable data from Nepal.

Serum LDL-C levels were unexpectedly lower in the case group compared to the control group (78.0 vs. 86.2 mg/dL, p = 0.040). This discrepancy is likely due to the significantly older age of the control group (19.0 vs. 25.0 years) and the broader interquartile range (IQR) of LDL-C in controls (37.5 vs. 17.0 mg/dL), indicating physiological age effects rather than metabolic changes specific to acne. Sobhan et al similarly observed non-significant differences in LDL levels.[13] The increase in LDL-C levels corresponding to acne severity within the case group (76→77→86 mg/dL) remains a clinically pertinent pattern. Serum HDL-C levels were higher in the case group (52.0 vs. 38.2 mg/dL), which can be attributed to the younger age and predominance of females (68%) in this group. The notably low HDL-C levels in the control group (median 38.2 mg/dL, below the NCEP male threshold of 40 mg/dL) suggest confounding factors related to age and sex. The negative correlation between acne severity and HDL-C levels (rs=−0.223, p = 0.026) supports a progressive atherogenic shift with increasing acne severity, aligning with emerging evidence that links severe acne to metabolic syndrome.[14]

The present findings underscore the necessity for fasting lipid profile screening in individuals with moderate-to-severe acne. Petca et al have documented metabolic abnormalities associated with severe acne and have advocated for the assessment of lipid levels and insulin resistance in these patients.[14] Our data offer regional evidence that supports this recommendation within the Nepalese context.

LIMITATIONS

When interpreting these findings, several limitations must be considered. The control group was significantly older than the case group (median age 25.0 vs. 19.0 years, p < 0.001); strict individual age-matching was not achieved, potentially confounding comparisons of HDL-C and LDL-C levels. This study was conducted at a single tertiary care center, which may limit the generalizability of the findings to the broader Nepalese population. The subgroup with severe acne included only 19 patients, which restricts the statistical power for analyses specific to severity. Key confounding variables, such as BMI, dietary history, physical activity, family history of dyslipidemia, and tobacco or alcohol use, were not systematically recorded. Hormonal parameters, including testosterone, DHEAS, insulin, and IGF-1, were not measured, precluding a direct mechanistic evaluation. The duration of the disease was based on patient recall, which is susceptible to recall bias. The cross-sectional nature of lipid measurement precludes causal inference; thus, longitudinal studies are warranted. There is a lack of published Nepalese data on acne-associated dyslipidemia for regional comparison, which simultaneously underscores the originality of this study.

CONCLUSION

This hospital-based case-control study reveals an association between acne vulgaris and a dyslipidemic lipid profile, particularly characterized by significantly elevated serum total cholesterol (TC) and triglycerides (TG) compared to healthy controls. A consistent and statistically significant severity-dependent gradient was observed for TC, TG, and low-density lipoprotein cholesterol (LDL-C), with patients exhibiting severe acne demonstrating significantly higher lipid levels than those with mild or moderate disease. Both disease duration and acne severity showed a positive correlation with TC, TG, and LDL-C, while acne severity was inversely correlated with high-density lipoprotein cholesterol (HDL-C), indicating a progressive pattern of lipid dysregulation that exacerbates with increased severity and chronicity.

Acne vulgaris should be regarded not merely as a dermatological disorder but as a condition with potential systemic metabolic implications. Routine fasting lipid profile assessment should be considered in patients with moderate-to-severe acne or prolonged disease duration, to enable timely dietary modification, lifestyle counselling, and metabolic evaluation where appropriate.

To the best of our knowledge, this study represents one of the initial hospital-based case-control investigations examining serum lipid profiles and their association with acne severity and disease duration within a Nepalese population. The research offers foundational regional data to guide clinical practice and inform future research endeavors. It is recommended that longitudinal multicenter studies with rigorous age-matching and comprehensive metabolic profiling be conducted to validate and expand upon these findings.

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