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Serum Lipid Profile and Inflammatory Severity in Acne Vulgaris: Implications for Metabolic Health

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Abstract

Background: Acne vulgaris (AV) is a chronic inflammatory disorder of the pilosebaceous unit with varying degrees of severity. Increasing evidence suggests that metabolic disturbances, including dsylipidemia, may contribute to its inflammatory progression. This study aimed to investigate the association between serum lipid profiles and AV severity.

Methods: A cross-sectional study was conducted among 30 patients with AV aged 25–40 years at a tertiary hospital in Manado, Indonesia. Serum triglycerides, low-density lipoprotein (LDL), high-density lipoprotein (HDL), and total cholesterol levels were measured. Association between lipid parameters and AV severity were assessed using Spearman's rank correlation. Group differences were analyzed using one-way ANOVA or the Kruskal-Wallis test with post-hoc analysis.

Results: Participants were predominantly Minahasa (66.7%) and female (60.0%). Total cholesterol and LDL levels demonstrated strong positive correlations with AV severity (r=0.696 and 0.654, respectively; p<0.001). Patients with severe AV had significantly higher total cholesterol and LDL levels compared to those with mild and moderate AV (F=13.275; p<0.001 for total cholesterol; H=12.496; p=0.002 for LDL). Triglycerides levels showed a weak correlation with AV severity (r=0.375; p=0.041) but no significant intergroup difference (p=0.112). HDL levels were not significantly associated with AV severity (p=0.198).

Conclusions: Dyslipidemia, specifically elevated total cholesterol and LDL, is strongly associated with severe AV. These findings underscore the importance of metabolic evaluation and lifestyle-based interventions as part of comprehensive management to reduce inflammation and promote long-term metabolic health.

Keywords: Acne vulgaris, cholesterol, low-density lipoprotein, serum lipid profile

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Introduction

Acne vulgaris (AV) is a chronic inflammatory disorder of the pilosebaceous unit that present with varying degrees of severity.[1] It predominantly occurs during adolescence, affecting approximately 85% of adolescence, and may persist into adulthood.[2,3] Although the exact cause of AV remains unclear, multiple contributing factors have been proposed, including genetic predisposition, ethnicity, hormonal influences, psychological stress, environmental exposure, drug use, and nutritional habits.[1]

Dietary patterns rich in fats or lipids, such as triglycerides, cholesterol, and phospholipids, have been frequently linked to the development or exacerbation of AV.[4] The Western diet, characterized by a high intake of high glycemic index foods, dairy products, and fats, but deficient in essential unsaturated fatty acids, results in an imbalanced omega-6/

omega-3 ratio of approximately 20:1, markedly higher than the more balanced 1:1 ratio commonly observed in traditional diets. Furthermore, this dietary pattern is often low in vitamins and antioxidants, while being excessive in refined carbohydrates and trans fats. Inadequate intake of essential unsaturated fatty acids, including linoleic and alpha-linolenic acid, may lead to abnormal keratinization in sebaceous gland ducts and increased transepidermal water loss, thereby potentially exacerbating acne severity.[5,6]

North Sulawesi, a province in Indonesia, is home to diverse ethnic populations, including the Minahasan people, whose traditional diet is characterized by a high intake of animal-derived fats. A previous study found that elevated consumption of saturated fats was associated with an increased risk of coronary heart disease in this population.[7] However, data on the association of serum lipid profiles and the clinical severity of AV among patients in North Sulawesi remain limited. Therefore, this study aimed to investigate the association of serum lipid profiles (triglycerides, low-density lipoprotein [LDL], high-density lipoprotein [HDL], and total cholesterol) with the clinical severity of AV.

Methods

A cross-sectional study was conducted from March to September 2023 at the Outpatient Department of Dermatology, Venereology, and Aesthetics, Prof. Dr. R. D. Kandou General Hospital, Manado, Indonesia. The study enrolled patients aged 25–40 years who were clinically diagnosed with AV. Participants were selected using a purposive sampling technique.

The exclusion criteria were: 1) presence of hormonal conditions, including menstruating, pregnancy, use of hormonal contraceptives, or diagnosed endocrine disorder; 2) receipt of any topical or systemic AV treatment within one month prior to enrollment; 3) sleep duration of fewer than six hours per day, and 4) current use of corticosteroid, lipid-lowering agents (fibrates and/or statin), and or β-blocker therapy.

Data collection consisted of structured questionnaires, clinical assessments performed by two board-certified dermatovenereologists, and laboratory analysis of serum lipid profiles. The questionnaire collected demographic information, including age, gender, and ethnicity. A clinical dermatological examination was conducted to confirm the diagnosis of AV and to assess the clinical severity. Additionally, laboratory investigations included measurements of serum lipid profile, specifically triglycerides, LDL, HDL, and total cholesterol levels, were performed. Ethical approval was obtained from the Ethics Committee of the Faculty of Medicine, BLU RSUP Prof. Dr. R. D. Kandou Manado, North Sulawesi (approval number: PP 04 03/XIX 3/4770/2022). Written informed consent was obtained from all participants prior to enrollment, and all data were anonymized to ensure confidentiality.

For analysis, participants were categorized into three age groups: 25–29 years, 30–34 years, and 35–39 years. The clinical severity of AV was classified according to the Lehmann criteria into mild (fewer than 20 comedones, fewer than 15 inflammatory lesions, and fewer than 30 total lesions), moderate (20–100 comedones, 15–50 inflammatory lesions, and 30–125 total lesions); and severe (more than 5 nodules or cysts, more than 100 comedones, more than 50 inflammatory lesions, and more than 125 total lesions).

Serum lipid profiles parameters were analyzed as continuous variables and presented as mean±standard deviations or median with interquartile range, as appropriate. Questionnaire-derived data were systematically recorded in Microsoft Excel, and statistical analyses were conducted utilizing IBM SPSS Statistics for Windows, version 26.0. Descriptive statistics were used to summarize demographic characteristics. The Kolmogorov-Smirnov test was applied to assess data normality. Associations between serum lipid profile parameters and the clinical severity of AV were assessed using the Spearman's rank correlation test, while comparisons of continuous variables across severity groups were carried out using one-way analysis of variance (ANOVA) or the Kruskal-Wallis H test, as appropriate. A p-value <0.05 was considered statistically significant.

Results

A total of 30 patients with AV who met the inclusion criteria were recruited and evenly distributed according to clinical severity, with 10 patients in each group (mild, moderate, and severe). Most participants were Minahasan ethnicity (66.7%), female (60.0%), and belonged to 25–29 years age group (66.7%) (Table 1). Among male participants, the majority (58.3%) were classified as having moderate AV, whereas half of the female participants (50.0%) were classified as having

Table 1 Demographic Characteristics of Patients with Acne Vulgaris (n = 30)

Variable	n	%
Gender		
Female	18	60.0
Male	12	40.0
Ethnicity		
Minahasa	20	66.7
Chinese	4	13.3
Sangirese	3	10.0
Javanese	2	6.7
Gorontalese	1	3.3
Age group (years)		
25–29	20	66.7
30–34	9	30.0
35–39	1	3.3

mild AV. The mean age of the participants was 28.43 ± 3.04 years, with a median age of 28.5 years (range: 25–36 years).

Overall, serum lipid profile parameters showed an increasing trend with greater clinical severity of AV. Mean concentrations of total cholesterol, LDL, and triglycerides were highest in the severe AV group, whereas mean HDL levels were slightly lower in the moderate AV group compared with the mild and severe groups (Table 2).

Analysis of Spearman's rank correlation revealed strong and statistically significant positive correlations between AV severity and both LDL (r=0.654; p<0.001) and total cholesterol levels, (r=0.696; p<0.001), indicating that higher concentrations of these lipids parameters were associated with increased AV severity. Triglyceride levels showed a statistically significant but weak positive correlation with the clinical severity of AV (r=0.375; p=0.041). In contrast, HDL

Table 2 Correlation Between Lipid Profile Parameters and Clinical Severity of Acne Vulgaris

Lipid Profile Parameters	Degree of Severity			r	p-value
	Mild AV n=10	Moderate AV n=10	Severe AV n=10		
Triglycerides (mg/dL)[a]	65.6 ± 22.3	84.7 ± 29.7	117.6 ± 78.4	0.375	0.041
LDL (mg/dL)[a]	104.7 ± 18.8	126.1 ± 21.9	154.3 ± 32.2	0.654	0.000
HDL (mg/dL)[a]	58.1 ± 9	49.3 ± 9.6	57.9 ± 16.2	–	0.692
Total cholesterol (mg/dL)[a]	171.5 ± 19	184 ± 19	225.4 ± 32.6	0.696	0.000

Note: [a] Values are presented as mean ± standard deviation for descriptive purposes. AV= Acne vulgaris; LDL= Low-density lipoprotein; HDL= High-density lipoprotein., *Spearman's rank correlation test, r-value for HDL was not calculated

Table 3 Comparison of Lipid Profile Parameters Across Acne Severity Groups

Variable	Overall Value	Test Statistic	p-value	Significant Post-Hoc Comparison
Total cholesterol (mg/dL)[b]	193.67 ± 33.25	F=13.275	<0.001	Mild vs. Moderate, Severe
LDL (mg/dL)[c]	120.00 (109.25 – 149.25)	H=12.496	0.002	Mild vs. Moderate, Severe
HDL (mg/dL)[b]	55.10 ± 12.41	F=1.719	0.198	N/A
Triglycerides (mg/dL)[c]	73.00 (58.25 – 99.25)	H=4.383	0.112	N/A

Note. [b]Analyzed using one-way ANOVA; data presented as mean ± standard deviation.[c]Analyzed using Kruskal–Wallis H test; data presented as median (interquartile range). Post-hoc analyses were performed using Tukey's HSD test (ANOVA) and Dunn's test with Bonferroni correction (Kruskal–Wallis), where applicable. N/A indicates that no post-hoc analysis was conducted because the overall test was not statistically significant (p>0.05)

levels were not significantly associated with clinical severity of AV (p=0.692) (Table 2).

Comparative analysis of lipid profile parameters (triglycerides, LDL, HDL, and total cholesterol) across three levels of clinical severity of AV using one-way ANOVA showed a highly significant difference in total cholesterol ($F(2.27)=13.275$; $p<0.001$). No significant difference was observed for HDL levels ($F(2.27)=1.719$; $p=0.198$). The Kruskal-Wallis H test showed a significant difference in LDL levels across severity groups ($H(2)=12.496$; $p = 0.002$), whereas for triglycerides levels did not differ significantly ($H(2)=4.383$; $p=0.112$) (Table 3).

Post-hoc analysis using Tukey's honestly significant difference (HSD) revealed that the severe AV group had significantly higher mean total cholesterol compared with both the mild group (mean difference=53.90; 95% CI:26.77–81.03; $p<0.001$) and the moderate groups (mean difference=41.30; 95% CI:14.17–68.43; $p=0.002$). No significant difference was observed between the mild and moderate groups ($p=0.492$). For the LDL levels, Dunn's post-hoc test with Bonferroni correction revealed a significant difference between the mild and severe AV group ($Z= -3.519$, adjusted $p=0.001$), while differences between the mild and moderate groups, as well as between the moderate and severe groups were not significant (adjusted $p=0.422$ and 0.122, respectively).

Discussion

Acne vulgaris is a multifactorial dermatological disorder involving abnormal follicular keratinization, increased sebum production, colonization by *Cutibacterium acnes*, and inflammation.[1] One important contributor to lesion development is hormonal stimulation of sebaceous gland activity.[1,8] Furthermore, hormonal dysregulation may also influence systemic lipid metabolism, suggesting a potential link between serum lipid profiles and AV severity, particularly due to the key role of cholesterol in steroid hormone synthesis.[9] In our study, total cholesterol and LDL levels were significantly associated with the clinical severity of AV consistent with findings reported in prior studies.[9–13]

HDL is a complex and heterogeneous lipoprotein particle that plays a primary role in reverse cholesterol transport.[14] The molecular composition of HDL is inherently variable and subject to various physiological states, although it typically consists of phospholipids (~50%), cholesteryl esters (~25%), triglycerides (~20%), and apolipoproteins (~5%).[14] In our study, HDL showed a mixed trend across AV severity groups, with the lowest mean concentration in the moderate AV group and no overall association with AV severity. Several mechanism may explain this finding. First, systemic inflammation and infection can induce structural remodeling and functional impairment of HDL particles, characterized by decreased cholesteryl esters and increased free cholesterol, triglycerides, and free fatty acids.[15] Second, HDL may act as a negative acute-phase reactant, whereby inflammatory cytokines reduce circulating HDL levels through altered synthesis and lipid transfer protein activity.[16] Third, the non-linear progression of AV from microcomedones to inflammatory lesions involving distinct immune and cellular pathways may contribute to variability in HDL levels across clinical severity categories.[17]

From a physiological standpoint, sebum is a lipid-rich substance predominantly composed of triglycerides, wax esters, squalene, and cholesterol, produced and secreted by sebaceous glands.[18] These glands actively uptake circulating lipids through fatty acid transport proteins (FATPs), particularly FATP4 (solute carrier family 27 member 4 (SLC27A4)), and LDL receptors.[19] Sebocytes also express lipoprotein lipase, facilitating the hydrolysis of serum lipoproteins into free fatty acids.[19] Lipid metabolism within the skin is influenced by multiple factors,, including nutritional status, dietary intake, lifestyle behaviors, and genetic predisposition. Notably, fasting has been shown to lower the incorporation of free fatty acids into sebum by up to 20%.[18,19] Additionally, recent evidence suggests that the skin act as a primary target for dietary fat deposition, with triglycerides accumulating in the epidermis and dermal white adipose tissue for several weeks after ingestion, thereby influencing the skin surface lipidome.[20,21]

When stratified by the degree of AV severity, a clear trend toward higher total cholesterol was observed. Sebaceous glands function is strongly regulate by androgens, which are synthesized from cholesterol in the gonad and adrenal glands.[1,22] Furthermore, sebaceous glands are also capable of local androgen synthesis from dehydroepiandrosterone sulfate (DHEA-S).[1] Androgen receptors are expressed in sebaceous glands basal cell and in keratinocytes of the outer root sheath of hair follicles, enabling responsiveness to both

systemin and locally produced androgens.[1] Free testosterone is enzymatically converted into 5α-dihydrotestosterone by the action of 5α-reductase, an enzyme whose activity correlates positively with sebaceous gland volume.[23] Given cholesterol's role as a precursor for steroid hormone; dysregulation of cholesterol metabolism may contribute to AV pathogenesis.[9]

Although dietary habits were not directly investigated in this study, substantial evidence support a role for diet, particularly Westernized dietary patterns, in pathophysiology of AV.[24] Such diets are typically rich in saturated fats, red meats, dairy products, eggs, refined grains, and high-glycemic index foods.[25] This nutrient composition promotes elevation in insulin, insulin-like growth factor (IGF)-1, and branched-chain amino acids such as leucine, which collectively stimulate follicular keratinocyte proliferation, sebocyte lipogenesis, and inflammatory signaling.[19,26] These process initiates comedogenesis and exacerbate inflammatory lesions. In addition, linoleic acid, a peroxisome proliferator-activated receptor-γ (PPARγ) ligand commonly found in Western diets, further promotes keratinocyte proliferation and seboceous lipid synthesis.[26]

Dietary modification, particularly reducing high-glycemic foods, dairy intake, and processed products, should be emphasized as part of AV management.[24,27] Emerging evidence suggest that nutrient-rich dietary patterns, including plant-based, Mediterranean, and ketogenic diet, may improve AV outcomes by reducing of both systemic inflammation and sebaceous gland activity.[28,29] Although further controlled studies are needed, integrating personalized nutritional counseling with conventional pharmacological therapy may offer a more comprehensive approach to optimizing AV management.[28]

This study demonstrated a significant association of serum lipid profile with the clinical severity of AV, potentially mediated by increased lipid uptake by sebaceous glands or hormonal modulation, especially androgenic activity, or combination of both mechanism. However, several limitations should be acknowledged, including the cross-sectional design, the relatively small sample size, and the lack of data on lifestyle factors, such as dietary intake patterns and physical activity. These limitations restrict causal inference. Future multicenter studies with larger and more diverse populations, incorporating assessments of dietary patterns, lifestyle factors, lipid profiles, and AV severity are recommended.

In conclusion, there is an association between elevated serum lipid profiles, particularly LDL and total cholesterol and the clinical severity of AV. Total cholesterol and LDL are significantly higher in patients with severe AV compared with those with mild and moderate AV. Conversely, HDL levels shows no overall association with clinical severity of AV, although a mixed patterns is observed across severity groups. These findings suggest that serum lipid profile evaluation may relevant in the clinical management of AV. Given the multifactorial nature of disease, a comprehensive treatment strategy that includes metabolic and dietary considerations may contribute to improved clinical outcomes.

Author's Contributions

MGK contributed to conceptualization, validation, formal analysis, investigation, data curation, writing review and editing, supervision, and funding acquisition. NHK contributed to methodology, data analysis, writing review and editing, and supervision. TSK contributed to conceptualization, methodology, data curation, and writing review and editing. SMG contributed to conceptualization, validation, formal analysis, investigation, data curation, writing original draft, and project administration. PMC contributed to methodology, validation, data analysis, writing review and editing, visualization, and project administration.

Conflict of Interest

The authors declare no conflicts of interest.

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