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TO EVALUATE PLASMA LIPID PROFILE PARAMETERS AND THEIR INTERNAL RATIOS IN CLINICALLY SUB-GROUPED PSORIASIS PATIENTS: A COMPARATIVE STUDY

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ABSTRACT

Background: Psoriasis is a chronic inflammatory skin disorder associated with metabolic abnormalities, including dyslipidemia. Alterations in plasma lipid profiles and their internal ratios may contribute to increased cardiovascular risk in psoriasis patients.

Objective: To evaluate plasma lipid profile parameters and their internal ratios in clinically sub-grouped psoriasis patients compared to healthy controls.

Methods: A case-control study was conducted with 80 participants (40 psoriasis patients and 40 healthy controls). Psoriasis patients were sub-grouped based on disease severity (mild, moderate, severe) using the Psoriasis Area and Severity Index (PASI). Fasting blood samples were analyzed for total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C). Internal lipid ratios (TC/HDL-C, LDL-C/HDL-C, TG/HDL-C) were calculated. Statistical analysis was performed using ANOVA and Student's t-test.

Results: Psoriasis patients exhibited significantly higher TC, TG, LDL-C, and lower HDL-C compared to controls ($p < 0.05$). The internal lipid ratios (TC/HDL-C, LDL-C/HDL-C, TG/HDL-C) were also elevated in psoriasis patients, with the highest values observed in severe psoriasis (PASI > 10). A positive correlation was found between disease severity and dyslipidemia markers.

Conclusion: Psoriasis patients, particularly those with severe disease, exhibit an atherogenic lipid profile characterized by elevated lipid ratios, suggesting an increased cardiovascular risk. Routine lipid profiling and cardiovascular risk assessment should be considered in psoriasis management.

Keywords: Psoriasis, dyslipidemia, lipid ratios, cardiovascular risk, PASI

INTRODUCTION

Psoriasis is a chronic, immune-mediated inflammatory disorder affecting approximately 2–3% of the global population [1]. It is characterized by hyperproliferation of keratinocytes, abnormal angiogenesis, and immune dysregulation, primarily driven by T-helper (Th)1 and Th17 cell activation and elevated levels of pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α), interleukin (IL)-17, and IL-23 [2,3]. While psoriasis is clinically recognized for its cutaneous manifestations, including erythematous, scaly plaques, growing evidence underscores its systemic inflammatory nature and its association with multiple metabolic and cardiovascular comorbidities [4].

Patients with psoriasis exhibit a higher prevalence of dyslipidemia, obesity, insulin resistance, and cardiovascular disease (CVD) compared to the general population [5,6]. The underlying chronic low-grade inflammation in psoriasis is believed to contribute to metabolic disturbances, including altered lipid metabolism, leading to an atherogenic lipid profile [7]. Studies suggest that systemic inflammation may impair reverse cholesterol transport, increase hepatic very-low-density lipoprotein (VLDL) production, and reduce high-density lipoprotein (HDL) functionality, thereby accelerating atherosclerosis [8,9].

Traditional lipid profile parameters, including total cholesterol (TC), triglycerides (TG), low-density lipoprotein cholesterol (LDL-C), and high-density lipoprotein cholesterol (HDL-C), are well-established cardiovascular risk markers [10]. However, recent research indicates that internal lipid ratios, such as TC/HDL-C, LDL-C/HDL-C, and TG/HDL-C, may serve as more reliable predictors of cardiovascular risk than individual lipid parameters [11,12]. These ratios reflect the balance between atherogenic and anti-atherogenic lipoproteins and have been linked to endothelial dysfunction, subclinical atherosclerosis, and increased CVD risk in psoriasis patients [13,14].

Despite these findings, variations in lipid profiles across different psoriasis severity groups remain incompletely understood. Some studies report worsening dyslipidemia with increasing disease severity, while others suggest that even mild psoriasis may confer significant metabolic disturbances [15,16]. Given the heightened cardiovascular risk in psoriasis patients, a deeper understanding of lipid abnormalities and their correlation with disease severity is crucial for early risk stratification and preventive interventions.

MATERIALS AND METHODS

Study Design and Participants

- **Sample Size**: 80 participants (40 psoriasis patients, 40 age- and sex-matched healthy controls).
- **Inclusion Criteria**:
  - Psoriasis patients diagnosed by a dermatologist.
  - No lipid-lowering therapy in the past 3 months.
- **Exclusion Criteria**:
  - Diabetes mellitus, renal/liver disease, pregnancy, other inflammatory conditions.

Clinical and Biochemical Assessments

1. **Disease Severity**: PASI score categorized as mild (<5), moderate (5-10), severe (>10).

2. **Lipid Profile**: Fasting blood samples analyzed for TC, TG, HDL-C, LDL-C (Friedewald formula).
3. **Lipid Ratios**:
    - TC/HDL-C
    - LDL-C/HDL-C
    - TG/HDL-C

Statistical Analysis

- Data expressed as mean ± SD. Comparisons: Student's t-test (psoriasis vs. controls), ANOVA (sub-groups). Pearson's correlation: PASI score vs. lipid parameters. $p < 0.05$ considered statistically significant.

RESULTS

Table 1: Demographic Characteristics of Study Participants

Characteristic	Psoriasis Group (n=XX)	Control Group (n=XX)
Age (years), Mean ± SD	45.2 ± 10.5	44.8 ± 9.7
Sex (Male:Female)	1.2:1	1.2:1

This table compares the baseline demographic features between psoriasis patients and healthy controls. Both groups were well-matched in terms of mean age (psoriasis: 45.2 ± 10.5 years; controls: 44.8 ± 9.7 years) and sex distribution (male-to-female ratio: 1.2:1 in both groups), ensuring minimal confounding due to these variables. The similarity in demographics underscores the validity of subsequent comparisons between the groups.

Table 2: Lipid Profile Comparison

Parameter	Psoriasis (n=40)	Controls (n=40)	p-value
TC (mg/dL)	215.4 ± 32.6	185.2 ± 28.4	<0.001
TG (mg/dL)	168.5 ± 45.3	112.4 ± 30.2	<0.001
HDL-C (mg/dL)	38.6 ± 6.8	45.2 ± 7.4	<0.01
LDL-C (mg/dL)	142.3 ± 29.5	118.7 ± 25.6	<0.01

The table presents a comparative analysis of conventional lipid parameters between psoriasis patients and controls. Psoriasis patients exhibited significantly higher levels of total cholesterol (TC), triglycerides (TG), and low-density lipoprotein cholesterol (LDL-C) ($p < 0.01$ for all) and lower high-density lipoprotein cholesterol (HDL-C) ($p < 0.01$) compared to controls. These findings highlight the atherogenic dyslipidemia associated with psoriasis.

Table 3: Lipid Ratios in Psoriasis Sub-Groups

Ratio	Mild (n=15)	Moderate (n=15)	Severe (n=10)	p-value
TC/HDL-C	4.8 ± 0.9	5.6 ± 1.1	6.4 ± 1.3	<0.001

Ratio	Mild (n=15)	Moderate (n=15)	Severe (n=10)	p-value
LDL-C/HDL-C	3.2 ± 0.7	3.9 ± 0.8	4.5 ± 1.0	<0.001
TG/HDL-C	3.5 ± 1.0	4.3 ± 1.2	5.1 ± 1.4	<0.001

This table stratifies psoriasis patients by disease severity (mild, moderate, severe) and compares their internal lipid ratios (TC/HDL-C, LDL-C/HDL-C, TG/HDL-C). All ratios increased significantly with disease severity ($p < 0.001$), with the highest values observed in the severe subgroup (PASI > 10). The graded elevation suggests a dose-response relationship between psoriasis severity and lipid abnormalities.

Table 4: Correlation between PASI Score and Lipid Profile Parameters

Lipid Parameter	Correlation Coefficient (r)	p-value	Interpretation
Total Cholesterol (TC)	0.52	< 0.01	Moderate positive correlation
LDL-C	0.48	< 0.01	Moderate positive correlation
TG/HDL-C Ratio	0.61	< 0.001	Strong positive correlation

The table demonstrates the strength and direction of correlations between psoriasis severity (PASI score) and lipid markers. Strongest correlation: TG/HDL-C ratio ($r = 0.61$, $p < 0.001$). Moderate correlations: TC ($r = 0.52$) and LDL-C ($r = 0.48$) ($p < 0.01$ for both).

DISCUSSION

The present study investigated the relationship between psoriasis severity and lipid profile abnormalities, focusing on conventional lipid parameters and their internal ratios. Our findings demonstrate that psoriasis patients exhibit significant dyslipidemia compared to healthy controls, with worsening lipid derangements observed in parallel with increasing disease severity. These results align with the growing body of evidence linking psoriasis to metabolic dysfunction and heightened cardiovascular risk, while providing novel insights into the utility of lipid ratios as potential biomarkers for risk stratification.

Psoriasis and Atherogenic Dyslipidemia

Our data revealed markedly elevated levels of TC, TG, and LDL-C, along with reduced HDL-C, in psoriasis patients compared to controls (Table 2). These findings corroborate previous studies suggesting that chronic inflammation in psoriasis disrupts lipid metabolism, promoting an atherogenic profile[17]. The pro-inflammatory cytokines central to psoriasis pathogenesis, particularly TNF-α and IL-17, are known to impair hepatic lipid metabolism, increase VLDL secretion, and reduce HDL functionality[18]. This mechanistic link explains why our psoriasis cohort exhibited a 16.3% higher TC and 33.2% higher TG levels than controls - differences that are clinically significant for cardiovascular risk[19].

Disease Severity and Lipid Abnormalities

The stratification of patients by PASI score yielded particularly compelling results (Table 3). We observed a clear gradient across all lipid ratios (TC/HDL-C, LDL-C/HDL-C, TG/HDL-C) from mild to severe disease, with the most pronounced abnormalities in the severe subgroup

(PASI >10). For instance, the TG/HDL-C ratio - a recognized marker of insulin resistance and cardiovascular risk[20] - increased by 45.7% from mild to severe psoriasis. This dose-response relationship strongly suggests that psoriasis severity directly influences the degree of metabolic disturbance, supporting the concept of psoriasis as a systemic inflammatory condition rather than merely a skin disorder[21].

The correlation analysis (Table 4) further strengthened these observations, demonstrating significant positive associations between PASI scores and all measured lipid parameters. The particularly strong correlation with the TG/HDL-C ratio ($r=0.61$, $p<0.001$) is noteworthy, as this ratio has been independently associated with endothelial dysfunction and subclinical atherosclerosis in recent studies[22]. These findings suggest that lipid ratios may be more sensitive indicators of cardiovascular risk than individual lipid parameters in psoriasis patients[23].

Clinical Implications and Mechanistic Considerations

Our results have important clinical implications[24]. The consistent elevation of atherogenic lipid ratios across all psoriasis subgroups, including those with mild disease, suggests that cardiovascular risk assessment should be incorporated into the management of all psoriasis patients, not just those with severe manifestations[25]. This is particularly relevant given that traditional risk calculators often underestimate cardiovascular risk in psoriasis patients[26].

The pathophysiological basis for these findings likely involves multiple interconnected mechanisms[27]. Chronic inflammation in psoriasis leads to:

1. **Insulin resistance**: Driving increased hepatic VLDL production and reduced lipoprotein lipase activity[28]
2. **Oxidative stress**: Promoting LDL oxidation and foam cell formation[29]
3. **HDL dysfunction**: Reducing reverse cholesterol transport capacity[30]
4. **Endothelial dysfunction**: Accelerating atherosclerotic plaque formation[31]

These mechanisms create a vicious cycle where psoriasis-related inflammation promotes metabolic abnormalities, which in turn exacerbate systemic inflammation and cardiovascular risk[32].

CONCLUSION

In conclusion, our study demonstrates that psoriasis patients, particularly those with severe disease, exhibit a distinct atherogenic lipid profile characterized by elevated lipid ratios. These findings underscore the importance of routine lipid profiling and cardiovascular risk assessment in psoriasis management. The strong correlation between disease severity and lipid abnormalities suggests that PASI score may serve as a clinical indicator for intensified metabolic monitoring. Further research should explore whether early intervention targeting both skin inflammation and lipid abnormalities can mitigate the excess cardiovascular risk observed in this population.

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