Vol. 29, No. 2, 2025, Pages 225-228

Thi-Qar Medical Journal (TQMJ)

Thi-Qar Medical Journal was established in 2006 and is published by the College of Medicine, University of Thi-Qar, Iraq

ISSN (PRINT): 1992-9218 | ISSN (ONLINE): 3006-4791

www.jmed.utq.edu.iq utjmed@utq.edu.iq

Evaluation of serum Procalcitonin in Patients with Pustular Psoriasis

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Received: 2-11-2025

Revised: 19-12-2025

Accepted: 22-12-2025

DOI:

10.32792/jmed.2025.29.42

Keywords:

Corticosteroid

Pustular Psoriasis

Inflammation

Systemic Inflammation

How to cite

Ahmed Abdulhussein Kawen1, Saddam Sahib Atshan 2, Ahmed Sadek 3. Evaluation of serum Procalcitonin in Patients with Pustular Psoriasis. *Thi-Qar Medical Journal (TQMJ)*. 2025; Vol.(29):225-228.

Abstract

This study evaluates the effect of corticosteroids on procalcitonin levels in patients with pustular psoriasis compared to those with psoriasis vulgaris and healthy controls. The aim was to assess the potential role of procalcitonin as a marker for inflammation and disease severity. The study was conducted at Al-Nasiriyah Teaching Hospital from March 2023 to October 2024, with diagnoses confirmed by histopathological examination. The results demonstrated significant differences in procalcitonin levels among the groups, suggesting a potential role for corticosteroids in modulating systemic inflammation in psoriasis.

Introduction

Psoriasis is a chronic inflammatory disorder, with psoriasis vulgaris being the most common type. It is associated with hyperproliferation of keratinocytes and immune dysregulation. Pustular psoriasis, in contrast, is a severe form that often presents with pustules and systemic inflammation, leading to higher morbidity. Procalcitonin is a biomarker of systemic inflammation, often elevated in conditions like bacterial infections, but its role in psoriasis remains under-explored. Corticosteroids are commonly used in the management of psoriasis, but their effects on systemic markers like procalcitonin have not been thoroughly studied. This study aims to evaluate the impact of corticosteroid therapy on procalcitonin levels in patients with pustular psoriasis, psoriasis vulgaris, and healthy controls. Among its various clinical phenotypes, psoriasis vulgaris (plaque psoriasis) is the most prevalent, typically presenting with well-demarcated, erythematous plaques covered by silvery scales. In contrast, pustular psoriasis represents a rarer but more severe variant, marked by sterile pustules on erythematous skin, often accompanied by systemic symptoms such as fever, malaise, and leukocytosis. This form is associated with significant morbidity and can progress to life-threatening complications if untreated [1, 2, 3]. The underlying pathophysiology of psoriasis involves a complex interplay between genetic, environmental, and immunological factors. Dysregulation of both the innate and adaptive immune responses leads to the release of numerous pro-inflammatory cytokines, including TNF-α, IL-17, and IL-23, which drive keratinocyte proliferation and sustain chronic inflammation. These cytokines also have systemic effects, contributing to the concept of psoriasis as a systemic inflammatory disorder rather than a disease confined solely to

the skin. Procalcitonin (PCT) is a 116-amino-acid peptide precursor of the hormone calcitonin. In healthy individuals, it is produced at low levels by thyroid C-cells. However, under systemic inflammatory conditions—particularly bacterial infections—its expression increases markedly in extra-thyroidal tissues. Unlike traditional inflammatory markers such as CRP or ESR, PCT rises more rapidly and correlates closely with the severity of systemic inflammation. Although primarily used to differentiate bacterial from non-bacterial inflammation, recent studies suggest that PCT may also be elevated in non-infectious inflammatory conditions, including autoimmune and dermatologic diseases. However, its behavior and diagnostic significance in psoriasis, especially in pustular variants, remain insufficiently explored [2, 3]. Corticosteroids are potent anti-inflammatory and immunosuppressive agents that remain a cornerstone in the management of many inflammatory and autoimmune diseases. Their use in psoriasis, however, remains controversial, particularly in generalized pustular psoriasis (GPP), due to the risk of disease rebound or flare following withdrawal. Nonetheless, corticosteroids may be used in selected severe cases to rapidly control systemic inflammation. Despite their widespread use, little is known about their effect on biomarkers such as PCT in psoriasis patients. Understanding this relationship could help clarify whether PCT elevation in pustular psoriasis reflects true infection or merely systemic sterile inflammation, which has important implications for diagnosis and management [3]. Therefore, the present study aims to evaluate the impact of corticosteroid therapy on serum procalcitonin levels in patients with pustular psoriasis and psoriasis vulgaris compared with healthy controls. This investigation may provide insights into the inflammatory profile of different psoriasis phenotypes and the potential role of PCT as a biomarker for monitoring disease activity and treatment response.

Materials and Methods:

This observational study was conducted at Al-Nasiriyah Teaching Hospital from March 2023 to October 2024. The study included three groups:

1. Group 1: Patients diagnosed with pustular psoriasis.
2. Group 2: Patients diagnosed with psoriasis vulgaris.
3. Group 3: Healthy controls without any underlying risk factors.

Diagnosis was confirmed by histopathology, and all participants underwent blood tests to assess procalcitonin levels. Corticosteroid treatment was administered to patients with active disease, with changes in procalcitonin levels being monitored over time. Procalcitonin levels were measured using standard laboratory procedures [4, 5].

Results:

The study revealed significant differences in procalcitonin levels across the three groups. Pustular psoriasis patients had the highest procalcitonin levels, significantly higher than both psoriasis vulgaris patients and healthy controls (p-value < 0.05).

Table1: Demographics of Study Participants

Group	Number of Participants	Male(%)	Female(%)	Age Range
Group 1: Pustular Psoriasis	40	23 (57.5%)	17 (42.5%)	18-50
Group 2: Psoriasis Vulgaris	60	35 (58.3%)	25 (41.7%)	19-56
Group 3: Healthy Controls	65	35(53.8%)	30 (46.2%)	18-60

Table2: Procalcitonin Levels Across Groups

Group	Mean Procalcitonin Level (ng/mL)	p-value
Group 1: Pustular Psoriasis	2.75 ± 1.24	0.001
Group 2: Psoriasis Vulgaris	1.68 ± 0.99	0.023
Group 3: Healthy Controls	0.32 ± 0.21	-

The results show that procalcitonin levels were significantly higher in pustular psoriasis patients, suggesting an elevated systemic inflammatory response in these patients compared to psoriasis vulgaris and healthy controls. Furthermore, corticosteroid treatment appeared to lower procalcitonin levels in both the pustular psoriasis and psoriasis vulgaris groups, indicating a potential role for corticosteroids in reducing systemic inflammation [6, 7].

Discussion:

The findings of this study suggest that procalcitonin is a valuable biomarker for assessing systemic inflammation in patients with psoriasis, especially in more severe forms like pustular psoriasis. Elevated procalcitonin levels in pustular psoriasis patients are likely reflective of the higher intensity of systemic inflammation associated with this form of the disease. In contrast, psoriasis vulgaris patients showed moderate procalcitonin levels, which were significantly lower than those seen in pustular psoriasis patients but still higher than healthy controls [1, 5].

This supports the hypothesis that pustular psoriasis involves more systemic inflammation compared to psoriasis vulgaris, which is generally more localized. Elevated procalcitonin levels may also be an indicator of disease severity, as shown by the correlation between increased inflammatory markers and the presence of pustules [6, 8].

The effect of corticosteroids was also evident in the study, with a reduction in procalcitonin levels observed in both psoriasis groups. Corticosteroids are widely used to treat psoriasis and are known to suppress local inflammation; this study extends these findings to systemic markers, suggesting that corticosteroids may play a role in controlling overall inflammation. However, long-term corticosteroid use remains controversial due to potential side effects such as immunosuppression, which could lead to increased infection risk [4, 9].

Procalcitonin in Pustular Psoriasis vs. Psoriasis Vulgaris:

In this study, patients with pustular psoriasis demonstrated markedly elevated PCT levels, significantly higher than those with psoriasis vulgaris and healthy controls. This difference can be attributed to the extent and depth of inflammation characteristic of pustular psoriasis, which often involves widespread neutrophilic infiltration, cytokine storm, and acute systemic manifestations such as fever, malaise, and leukocytosis. These systemic features align with the biological pathways that stimulate PCT production—particularly IL-1β, TNF-α, and IL-6, which are markedly elevated in pustular variants [6, 8].

By contrast, psoriasis vulgaris is typically a chronic, localized inflammatory condition, dominated by Th1/Th17-mediated immune responses within the skin. The moderate elevation of PCT observed in psoriasis vulgaris patients reflects low-grade systemic inflammation, consistent with elevated C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) often seen in such cases, but not to the same degree as pustular disease [8,9].

These results support the hypothesis that pustular psoriasis represents a systemic inflammatory disorder, rather than merely a severe cutaneous variant. Elevated PCT levels may therefore serve as a quantitative marker of disease activity and systemic involvement, distinguishing it from the more localized inflammatory burden of psoriasis vulgaris.

Correlation Between PCT and Disease Severity:

The observed correlation between increased PCT levels and clinical severity—particularly the presence and extent of pustules—underscores PCT's potential as an objective biomarker for disease monitoring. Previous studies have shown that higher PCT levels correlate with elevated CRP, IL-6, and TNF-α, indicating a shared pathway in systemic inflammation [5, 8]. Therefore, monitoring PCT could provide clinicians with an additional, sensitive tool for assessing systemic inflammatory activity and evaluating treatment responses in severe psoriasis forms [9,10].

Effect of Corticosteroid Therapy:

A notable finding in this study is the reduction in PCT levels following corticosteroid therapy in both psoriasis groups. Corticosteroids exert their anti-inflammatory effects by inhibiting phospholipase A2, reducing the synthesis of pro-inflammatory cytokines such as IL-1, IL-6, and TNF-α—all of which are known to induce PCT synthesis. This suggests that the decline in PCT mirrors the attenuation of systemic inflammation under corticosteroid influence. [10].

However, it is important to interpret this cautiously. While corticosteroids can reduce systemic inflammatory markers and improve acute symptoms, long-term corticosteroid use in psoriasis is controversial. Chronic administration can result in rebound flares upon withdrawal, increased infection susceptibility, and metabolic side effects due to immunosuppression [4, 9]. Thus, while PCT reduction under corticosteroids reflects decreased inflammation, it should not be misconstrued as a sustainable therapeutic endpoint.

Clinical Implications.

These findings have several important implications:

1. Diagnostic Utility – PCT can help differentiate between severe systemic forms of psoriasis (e.g., pustular) and milder, localized variants (e.g., vulgaris).
2. Monitoring Disease Activity – Serial PCT measurements may serve as an adjunct to clinical scoring systems (e.g., PASI) to track systemic inflammatory burden.
3. Therapeutic Response – Changes in PCT levels may provide an early biochemical signal of treatment efficacy or flare risk.
4. Risk Stratification – Elevated baseline PCT could identify patients at higher risk of systemic complications, warranting closer clinical monitoring.

Limitations and Future Perspectives:

Although these findings are promising, several limitations should be noted. PCT levels can be influenced by subclinical infections, metabolic status, and comorbidities such as obesity or metabolic syndrome, which are prevalent among psoriasis patients. Future studies

should involve larger cohorts, longitudinal designs, and multimarker analyses (including IL-6, TNF-α, and CRP) to validate PCT's predictive value and establish disease-specific cutoff levels.
Additionally, exploring the impact of biologic therapies (e.g., TNF inhibitors, IL-17 blockers) on PCT dynamics could further elucidate its role as a biomarker of systemic inflammation control in the modern therapeutic era [5, 7].

Conclusion:

This study concludes that procalcitonin is a promising marker for evaluating systemic inflammation in psoriasis, particularly pustular psoriasis. Corticosteroid treatment appears to reduce systemic inflammation, as evidenced by a decrease in procalcitonin levels. Further studies should focus on validating procalcitonin as a reliable biomarker for assessing psoriasis severity and treatment response [8, 9].

References:

1. Liu, L., et al. (2020). Procalcitonin as a biomarker of inflammation in autoimmune diseases. Clinical and Experimental Dermatology, 45(6), 877-883.
2. Papadavid, E., et al. (2022). Effect of corticosteroids on inflammatory markers in psoriasis: A systematic review. Journal of Dermatological Treatment, 33(6), 845-852.
3. Gupta, A., et al. (2021). Effect of systemic corticosteroids on serum markers in psoriasis: A randomized trial. International Journal of Dermatology, 60(7), 804-811.
4. Gonzalez, M., et al. (2020). Systemic effects of psoriasis and the role of corticosteroids in inflammatory modulation. Archives of Dermatological Research, 313(5), 257-265.
5. Chao, C., & Lee, L. (2023). Psoriasis and inflammation: A systematic review of serum biomarkers and clinical outcomes. Dermatology Clinics, 41(2), 207-215.
6. Nash, P., & Sorensen, L. (2021). Procalcitonin as a potential marker for systemic inflammation in dermatologic diseases. Journal of Autoimmunity and Inflammation, 36(3), 78-85.
7. Varga, J., et al. (2021). Corticosteroids and their impact on biomarkers in dermatological diseases: An evidence-based approach. Dermatology and Therapy, 11(2), 128-134.
8. Bastien, M., et al. (2022). The effect of systemic treatment on biomarkers in psoriasis: Corticosteroids and beyond. Journal of Clinical Dermatology, 57(5), 789-798.
9. Kawen, A. A., & Jaaz, M. H. (2024). Ocular manifestations in psoriasis of Iraqi patients. Journal of Pakistan Association of Dermatologists, 34(2), 453-457.
10. Wu, L., et al. (2023). Effect of corticosteroids on procalcitonin levels in patients with inflammatory skin diseases. Journal of Dermatological Science, 92(4), 303-310.