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Exploring Vitamin K Deficiency in Psoriasis Vulgaris: A Pilot Investigation

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Abstract

Psoriasis vulgaris is a chronic inflammatory skin disorder characterized by dysregulated immune responses and abnormal keratinocyte proliferation. Emerging evidence suggests a potential link between vitamin K deficiency and the pathogenesis of psoriasis, highlighting the role of vitamin K in modulating inflammation, oxidative stress and tissue calcification. This pilot investigation aims to explore the prevalence of vitamin K deficiency in patients with psoriasis vulgaris and evaluate its association with disease severity and clinical outcomes. Serum levels of vitamin K and markers of inflammation, oxidative stress and vascular calcification will be measured in a cohort of patients with psoriasis vulgaris compared to healthy controls. Additionally, correlations between vitamin K status and disease activity, severity scores and treatment response will be analyzed. The findings from this pilot investigation may provide insights into the potential role of vitamin K in the pathophysiology of psoriasis vulgaris and inform future research directions and therapeutic strategies targeting vitamin K metabolism in the management of psoriasis.

Keywords: Psoriasis • Vitamin K deficiency • Inflammation • Oxidative stress

Introduction

Psoriasis vulgaris is a common inflammatory skin disorder characterized by erythematous plaques with silver scales, affecting approximately 2-3% of the global population. The pathogenesis of psoriasis is multifactorial, involving genetic predisposition, dysregulated immune responses, environmental triggers and abnormal keratinocyte proliferation. Despite advances in understanding the molecular mechanisms underlying psoriasis, the etiology and pathophysiology of the disease remain incompletely understood. Emerging evidence suggests that vitamin K, a fat-soluble vitamin known for its role in blood coagulation and bone metabolism, may also play a role in the pathogenesis of psoriasis. Vitamin K exists in multiple forms, including vitamin K1 (phylloquinone) and vitamin K2 (menaquinone), each with distinct biological activities. Beyond its classical functions, vitamin K has been implicated in modulating inflammation, oxidative stress and tissue calcification, processes that are known to contribute to the pathogenesis of psoriasis [1].

Preclinical studies have demonstrated anti-inflammatory effects of vitamin K, including inhibition of pro-inflammatory cytokine production and suppression of Nuclear Factor-kappa B (NF-κB) signaling pathways. Moreover, vitamin K has been shown to reduce oxidative stress and promote antioxidant enzyme activity, potentially mitigating the oxidative damage observed in psoriatic skin lesions. Additionally, vitamin K-dependent proteins such as Matrix Gla protein (MGP) play a crucial role in inhibiting vascular calcification, a process implicated in psoriasis-associated cardiovascular comorbidities. Despite these mechanistic insights, limited clinical data are available on the prevalence of vitamin K deficiency in patients with psoriasis vulgaris and its association with disease severity and clinical outcomes. This pilot investigation aims to address this gap in knowledge by examining serum levels of vitamin K and markers of inflammation, oxidative stress and vascular calcification in patients with psoriasis vulgaris compared to healthy controls. Furthermore, correlations between vitamin K status and disease activity, severity scores and treatment response will be evaluated to assess the potential clinical relevance of vitamin K deficiency in psoriasis [2].

Literature Review

Psoriasis vulgaris, a chronic inflammatory skin disorder, is characterized by aberrant immune responses and dysregulated keratinocyte proliferation, leading to the formation of erythematous plaques with silver scales. While the exact etiology of psoriasis remains incompletely understood, growing evidence suggests a potential association between vitamin K deficiency and the pathogenesis of the disease. Vitamin K, a fat-soluble vitamin, exerts diverse physiological effects beyond its classical roles in blood coagulation and bone metabolism. Preclinical studies have demonstrated anti-inflammatory properties of vitamin K, including inhibition of pro-inflammatory cytokine production and suppression of NF-κB signaling pathways implicated in psoriasis pathogenesis. Moreover, vitamin K has been shown to modulate oxidative stress and tissue calcification, processes that contribute to the development and progression of psoriasis. Vitamin K-dependent proteins such as Matrix Gla Protein (MGP) play a crucial role in inhibiting vascular calcification, a common feature of psoriasis-associated cardiovascular comorbidities. Despite these mechanistic insights, clinical data on the prevalence of vitamin K deficiency in patients with psoriasis vulgaris and its impact on disease severity and clinical outcomes are limited. Several observational studies have reported lower serum levels of vitamin K in patients with psoriasis compared to healthy controls, suggesting a potential link between vitamin K deficiency and the pathogenesis of psoriasis. However, the exact mechanisms underlying this association and the clinical implications of vitamin K deficiency in psoriasis remain to be elucidated. Furthermore, the effects of vitamin K supplementation on psoriasis outcomes have not been extensively studied, warranting further investigation into the potential therapeutic benefits of vitamin K in psoriasis management [3,4].

Discussion

The findings from the literature review suggest a plausible association between vitamin K deficiency and the pathogenesis of psoriasis vulgaris. Vitamin K's anti-inflammatory, antioxidant and anti-calcification properties make it a potential candidate for modulating key pathways involved in psoriasis pathophysiology. However, the clinical significance of vitamin K deficiency in psoriasis and the therapeutic potential of vitamin K supplementation require

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further investigation. Future research efforts should focus on elucidating the mechanisms by which vitamin K influences psoriasis development and progression, as well as evaluating the efficacy and safety of vitamin K supplementation as a therapeutic intervention for psoriasis management. Longitudinal studies examining the relationship between serum vitamin K levels, disease activity and treatment outcomes in patients with psoriasis are needed to establish causality and determine optimal supplementation strategies. Furthermore, exploring the interplay between vitamin K deficiency and other metabolic abnormalities commonly observed in psoriasis, such as obesity, insulin resistance and dyslipidemia, may provide additional insights into the complex pathophysiology of the disease. Additionally, investigating the impact of vitamin K supplementation on psoriasis-associated comorbidities, including cardiovascular disease and metabolic syndrome, may uncover potential benefits beyond skin manifestations [5,6].

Conclusion

In conclusion, vitamin K deficiency may contribute to the pathogenesis of psoriasis vulgaris through its effects on inflammation, oxidative stress and tissue calcification. While preclinical studies support the potential anti-psoriatic effects of vitamin K, clinical data on the prevalence of vitamin K deficiency in psoriasis and its clinical implications are limited. Further research is warranted to elucidate the role of vitamin K in psoriasis pathophysiology and evaluate the therapeutic potential of vitamin K supplementation in psoriasis management. By addressing these knowledge gaps, we may uncover novel approaches for optimizing psoriasis treatment and improving patient outcomes.

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Conflict of Interest

There are no conflicts of interest by author.

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