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Psoriasis Vulgaris Complicated by Secondary Tinea Pedis Infection: A Case Report

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ABSTRACT

Background: The coexistence of psoriasis vulgaris and tinea pedis presents significant diagnostic challenges due to similar clinical presentations. Chronic topical corticosteroid therapy may predispose psoriatic patients to secondary fungal infections through local immunosuppression. Case report: A 34-year-old female presented with a one-year history of erythematous, scaly, thickened lesions on bilateral feet extending above ankles, knees, elbows, and inguinal areas. Initial psoriasis vulgaris diagnosis led to treatment with topical desoximetasone 0.25% twice daily and oral cetirizine once 10mg daily. While other body sites improved significantly, bilateral foot lesions showed minimal response. Skin biopsy revealed characteristic psoriatic features including parakeratosis, Munro microabscesses, and epidermal acanthosis. Lactophenol cotton blue staining demonstrated septate hyphae with conidia consistent with Trichophyton species, confirming concurrent tinea pedis. Treatment was modified to fluconazole 150 mg weekly, topical ketoconazole 2% twice daily, and temporary corticosteroid discontinuation, resulting in significant improvement of foot lesions after three weeks.Summary: This case demonstrates the importance of comprehensive diagnostic evaluation when standard psoriasis treatment fails where the differential treatment response across anatomical sites served as a crucial indicator for further investigation emphasizing systematic approaches incorporating histopathological and microbiological examinations.

1. INTRODUCTION

Psoriasis defined as an immune-mediated, genetic disease manifesting in the skin, joints, or both (Boehncke and Schön, 2015). The disease has varied clinical manifestations: plaque psoriasis, characterized by erythematous plaques covered with white scaly scales, represents 90% of cases found in daily clinical practice; guttate psoriasis, observed as scaly teardrop-shaped spots; inverse psoriasis, usually found in skin folds; pustular psoriasis, characterized by pustules on erythematous skin; and erythrodermic psoriasis, which is marked by more than 90% of body surface area being covered with scaly erythematous skin (Boehncke and Schön, 2015). Currently, determining the precise global prevalence and incidence of psoriasis remains challenging due to significant variations across age groups, geographic regions, ethnicities, patient populations, and genetic backgrounds (Sylviningrum et al., 2019). According to previous study, European populations demonstrated a prevalence of 4.6%, while Asia exhibited the highest prevalence at 5.7%. In contrast, Latin America showed a significantly lower prevalence of 3.1%. Africa and North America demonstrated the lowest prevalence rates at 1.7% and 3.7%, meanwhile, Australia and the Middle East showed comparable prevalence rates of 4.6% and 4.9% (Skayem et al., 2025).

The disease burden was highest in the 60-69 years age group, with no significant gender predilection observed (Bartosińska et al., 2025). This autoimmune condition can affect various body sites, including the feet, where diagnosis may be complicated by the presence of other dermatological conditions (Maronese et al., 2024). The altered barrier function and local immunological changes in psoriatic skin create an environment that may predispose patients to secondary infections.

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Tinea pedis is a dermatophyte infection of the foot (Kaushik, et al., 2015). There are three dermatophyte species including Trichophyton rubrum, Trichophyton mentagrophytes, and Epidermophyton floccosum which Trichophyton rubrum as the common cause of epidermal dermatophyte infection (Barac et al., 2024). Tinea pedis affects an estimated 3% of the global population, with prevalence rates demonstrating distinct demographic patterns. The condition predominantly affects adolescents and adults, with children showing lower infection rates. Peak incidence occurs within the 16-45 years age group, indicating a predilection for the economically active population. Gender distribution shows a male predominance, with males experiencing higher infection rates compared to females (Leung et al., 2023).

Tinea pedis manifests in several distinct clinical forms: the hyperkeratotic (moccasin) variant is characterized by chronic hyperkeratotic scaling plaques with varying degrees of underlying erythema, typically affecting the heels, soles, lateral and medial aspects, posterior regions, and distal dorsum of the foot. Vesiculobullous tinea pedis presents with intensely pruritic vesicles and/or bullae overlying an erythematous base. The occult form of tinea pedis represents an asymptomatic presentation without visible clinical manifestations. Ulcerative tinea pedis is characterized by rapidly progressive interdigital erosions and ulcerations. Additionally, tinea incognito may develop when tinea pedis is inadvertently treated with immunosuppressive agents, particularly topical corticosteroids, resulting in atypical lesion morphology and loss of characteristic clinical features (Leung et al., 2023).

The moccasin type of tinea pedis presents particular diagnostic challenges as it shares remarkable morphological similarities with psoriasis vulgaris. Both conditions manifest as well-demarcated, erythematous plaques with silvery-white scales that can be virtually indistinguishable on clinical examination alone. The hyperkeratotic scaling pattern in chronic tinea pedis, particularly affecting the plantar surfaces, lateral foot margins, and dorsal aspects, closely resembles the characteristic scaling seen in plantar psoriasis (Całus et al., 2023). The clinical relevance of psoriasis-tinea pedis coexistence extends beyond diagnostic complexity to encompass significant therapeutic implications and patient safety concerns. Misdiagnosis represents a critical clinical risk, with studies indicating that cutaneous fungal infections are commonly misdiagnosed. Such diagnostic errors lead to inappropriate monotherapy with immunosuppressive agents in psoriasis patients, potentially exacerbating fungal proliferation and creating treatment-resistant infections. Treatment delays resulting from misdiagnosis can extend disease duration significantly, with patients experiencing prolonged symptoms and increased risk of secondary bacterial superinfection (Całus et al., 2023).

The shared clinical features of psoriasis vulgaris and tinea pedis create a diagnostic dilemma that cannot be resolved through clinical examination alone, making histopathological examination essential to identify psoriatic markers such as parakeratosis, acanthosis, Munro microabscesses, and the characteristic inflammatory infiltrate pattern (Kimmel and Lebwohl, 2018). Concurrently, mycological investigations including potassium hydroxide (KOH) preparation, fungal culture, and dermoscopy become indispensable for detecting dermatophyte elements such as hyphae, spores, and arthroconidia that are pathognomonic for fungal infection (Ridzuan et al., 2019). Furthermore, chronic topical corticosteroid therapy commonly used in psoriasis management may suppress local immune responses and facilitate fungal proliferation, creating a complex pathophysiological interaction between the two diseases (Sookdar et al., 2024).

Despite the theoretical predisposition for psoriatic patients to develop secondary fungal infections, documented cases of confirmed coexistence between psoriasis vulgaris and tinea pedis remain remarkably scarce in the medical literature, likely due to morphological similarity leading to presumptive diagnosis without confirmatory testing and the traditional approach of seeking single unifying diagnoses (Sookdar et al., 2024). This knowledge gap has significant clinical implications, as unrecognized coexistence can lead to treatment failure and disease progression, particularly with the increasing use of immunosuppressive therapies that may predispose patients to fungal superinfection (Całus et al., 2023). Accurate diagnosis of coexisting conditions requires systematic application of both histopathological and microbiological diagnostic methods.

We present an ongoing case of a 34-year-old female with histopathologically confirmed psoriasis vulgaris who subsequently developed culture-proven tinea pedis infection, demonstrating the diagnostic complexity encountered when these conditions coexist. This case report aims to highlight the critical importance of comprehensive diagnostic evaluation incorporating both histopathological and mycological examinations when standard psoriasis treatment fails to achieve expected therapeutic outcomes, and to emphasize the necessity of implementing appropriate therapeutic strategies that address both pathological processes while considering potential drug interactions and treatment priorities in managing such complex dermatoses

2. METHOD

Case Report

A 34-year-old female presented with a one-year history of erythematous, scaly, and thickened skin lesions affecting the dorsal aspects of both feet extending above the ankle joints. The patient complained of severe pruritus that significantly interfered with her daily activities. Similar lesions were observed on both knees, elbows, and inguinal areas **(Figure 1)**. Based on the clinical presentation and distribution pattern, the patient was diagnosed with psoriasis vulgaris. Initial treatment consisted of topical desoximetasone 0.25% cream applied twice daily to all affected areas except the inguinal region, along with oral cetirizine 10 mg once daily, considering the limited extent of the skin lesions. After several weeks of treatment, the lesions on both elbows, knees, and inguinal areas demonstrated significant improvement, resolving into hyperpigmented macules. However, the lesions on both lower extremities, particularly the dorsal feet and ankle areas, showed minimal to no therapeutic response despite adherence to the prescribed treatment regimen. The persistent lack of improvement in the lower extremity lesions, despite adequate response in other body sites, prompted further diagnostic evaluation to rule out concurrent conditions or alternative diagnoses.

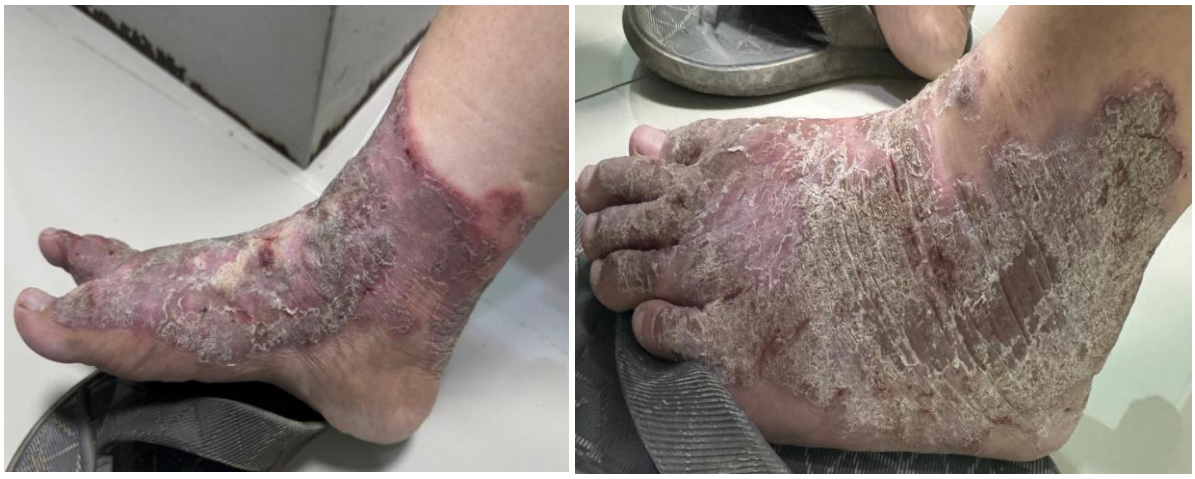

Figure 1. Erythematous, scaly, and thickened skin lesions affecting the dorsal aspects of both feet

Due to the lack of significant improvement in the bilateral foot lesions, a skin biopsy was performed for histopathological examination, along with acid-fast bacilli (AFB) staining and fungal examination. The differential diagnoses considered were psoriasis vulgaris, tinea pedis, and cutaneous tuberculosis variants. Histopathological examination of the skin lesion supported the diagnosis of psoriasis vulgaris, revealing characteristic features including parakeratosis, Munro microabscesses, and epidermal acanthosis. Acid-fast bacilli staining showed negative results for mycobacteria. However, lactophenol cotton blue staining demonstrated septate hyphae with conidia consistent with *Trichophyton* species.

Based on these diagnostic findings, the patient was diagnosed with psoriasis vulgaris with coincidental tinea pedis. The treatment regimen was modified to include fluconazole 150 mg once weekly as the available antifungal option, topical ketoconazole 2% cream applied twice daily, and

continued oral cetirizine 10 mg once daily. Topical corticosteroid therapy was temporarily discontinued. After three weeks of antifungal treatment, the bilateral foot lesions began showing improvement with reduced thickness of the skin lesions and decreased pruritus (Figure 2). The patient continues with the current treatment regimen for ongoing evaluation of therapeutic response and monitoring for potential adverse effects.

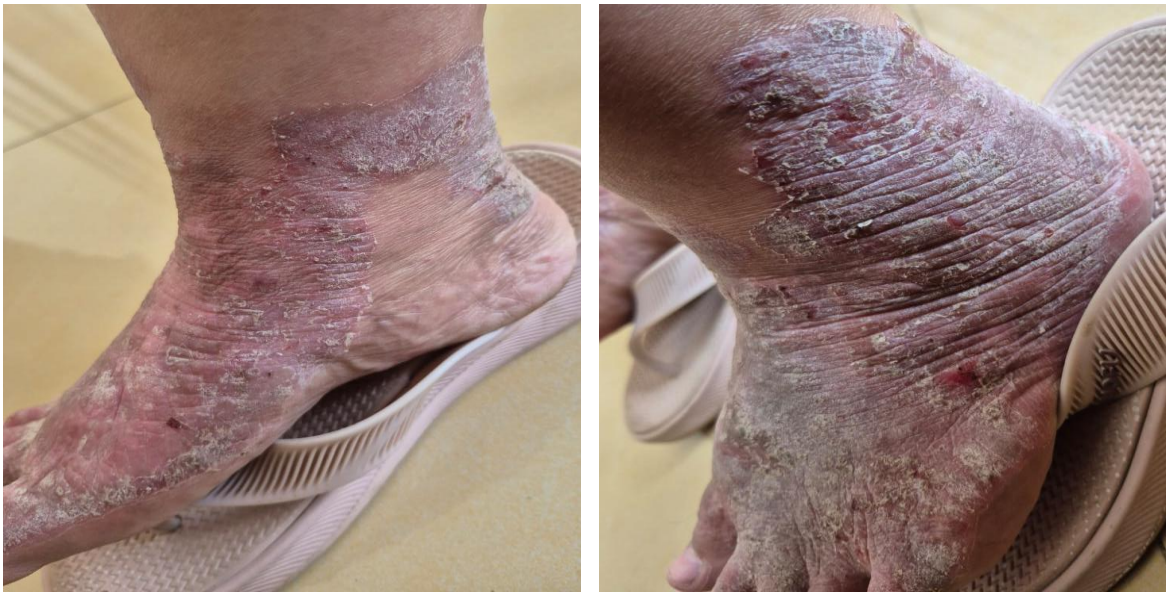

Figure 2. Reduction of erythema and skin thickness after treatment with oral and topical antifungal therapy

3. RESULT AND DISCUSSION

This case illustrates the diagnostic complexity encountered when psoriasis vulgaris coexists with tinea pedis, highlighting the importance of comprehensive diagnostic evaluation when standard treatment fails to achieve expected therapeutic outcomes. The patient's differential response to initial psoriasis therapy across anatomical sites served as a crucial clinical indicator necessitating further investigation. The clinical presentation of hyperkeratotic tinea pedis can closely mimic psoriasis, particularly when affecting the dorsal feet and ankles, as demonstrated in our case. Both conditions present with erythematous, scaly, and hyperkeratotic lesions that can be morphologically indistinguishable on clinical examination alone (Sookdar et al., 2024). This diagnostic challenge is further compounded when psoriasis genuinely coexists with fungal infection, as confirmed through our systematic diagnostic approach combining histopathological and microbiological examinations.

The pathophysiological interaction between psoriasis and dermatophyte infections creates a complex clinical scenario. Psoriatic skin exhibits compromised barrier function and altered local immunity, potentially predisposing patients to secondary fungal infections. The characteristic epidermal hyperproliferation and abnormal keratinocyte differentiation in psoriasis result in defective stratum corneum formation, leading to increased transepidermal water loss and compromised antimicrobial barrier function (Orsmond et al., 2021). The altered lipid composition and reduced ceramide content in psoriatic lesions further impair the skin's protective capabilities, creating microenvironmental conditions favorable for microbes especially dermatophyte colonization and proliferation (Knox and O'Boyle, 2021). Furthermore, psoriatic skin demonstrates significant immunological alterations that create a paradoxical predisposition to fungal infections. Under normal circumstances, effective antifungal immunity requires coordinated responses from multiple T-helper cell subsets, including Th1, Th2, and particularly Th17 cells, which play a crucial role in antifungal defense through the production of interleukin-17 (IL-17) (Bartemes and Kita, 2018). IL-17 promotes neutrophil recruitment, enhances

antimicrobial peptide production, and facilitates fungal clearance in acute infections. However, the chronic inflammatory state in psoriasis presents a paradoxical scenario where elevated Th17 responses and IL-17 levels, despite their inherent antifungal properties, may actually predispose patients to secondary fungal infections. This apparent contradiction can be explained by several interconnected mechanisms. First, the chronic and dysregulated nature of Th17 activation in psoriasis leads to functional immune exhaustion, where the prolonged inflammatory state compromises the effectiveness of antifungal responses despite elevated cytokine levels (McGeachy, et al., 2019). Second, the chronic IL-17-mediated inflammation causes significant epidermal barrier disruption and tissue damage, creating microenvironmental conditions that favor fungal colonization and override the protective antifungal effects of IL-17 (McGeachy, et al., 2019). This phenomenon likely contributed to the persistence and possible exacerbation of the tinea pedis infection in our patient's foot lesions.

The chronic use of topical corticosteroids, which are first-line therapy for localized psoriasis (Oon, et al., 2024), compounds these predisposing factors by further suppressing local immune responses, inhibiting keratinocyte proliferation, and altering the skin microbiome composition. Corticosteroids provide anti-inflammatory effects by reducing vasodilation, vascular permeability, and leukocyte recruitment to inflamed tissues. These agents suppress immune responses primarily through glucocorticoid receptor-mediated inhibition of NF-κB and other pro-inflammatory transcription factors. Additionally, corticosteroids decrease circulating macrophages and monocytes while impairing macrophage antimicrobial function by inhibiting phagolysosomal fusion (Li and Denning, 2023). This multifactorial immunosuppressive effect from corticosteroids, particularly the inhibition of phagolysosomal fusion in macrophages and suppression of local antifungal immunity, likely facilitated fungal colonization and contributed to the persistence and possible exacerbation of the tinea pedis infection in our patient's foot lesions, explaining the differential treatment response observed between corticosteroid-treated and untreated anatomical sites.

The therapeutic approach in this case demonstrates several unique aspects when compared to similar cases reported in the literature. While Sookdar et al (2024) reported two cases of psoriasis masked by tinea pedis in unhoused patients, their management focused primarily on antifungal therapy without detailed consideration of corticosteroid discontinuation timing or dual pathology management. In contrast, our case emphasizes the critical decision to temporarily discontinue topical corticosteroids while implementing concurrent systemic and topical antifungal therapy. Similarly, Calus et al, (2023) described misdiagnosis between psoriasis and fungal skin infection leading to inappropriate therapy that offered temporary remission. However, as a consequence, this made it difficult to establish the correct diagnosis at a later stage. The systematic diagnostic approach in our case, utilizing both histopathological confirmation of psoriasis and mycological identification of Trichophyton species, provides a simpler and more cost-effective method that successfully confirmed the coexistence of tinea pedis and psoriasis, compared to previous reports that used more expensive molecular identification methods for fungal infection (Chadeganipour, et al., 2021). Furthermore, unlike previous studies that focused primarily on diagnostic challenges (Leibovici et al., 2014), (Yadgar, et al., 2017), this case contributes to the literature by demonstrating the temporal relationship between corticosteroid-induced immunosuppression and fungal proliferation, supported by the differential treatment response across anatomical sites. The scientific contribution lies in establishing a systematic approach to diagnosis and management of such complex cases, providing clinicians with evidence-based rationale for comprehensive evaluation and targeted therapeutic modification in treatment-resistant psoriatic lesions.

Concurrently, the identification of septate hyphae with conidia consistent with Trichophyton species through lactophenol cotton blue staining confirmed the presence of dermatophyte infection. This dual diagnostic confirmation validated the clinical suspicion of coexisting conditions and guided appropriate therapeutic intervention. The therapeutic management of coincidental psoriasis and tinea pedis requires careful consideration of drug interactions and treatment priorities. In our case, temporary discontinuation of topical corticosteroids was essential to prevent further immunosuppression that could exacerbate the

fungal infection. The implementation of systemic antifungal therapy with fluconazole, combined with topical ketoconazole addressed the dermatophyte infection therapy while maintaining symptomatic control with oral antihistamines.

The observed clinical improvement after three weeks of antifungal therapy, characterized by reduced erythema, decreased skin thickness, and alleviated pruritus, supports the significant contribution of tinea pedis to the patient's clinical presentation. This therapeutic response validates our diagnostic approach and treatment modification, demonstrating the importance of addressing both pathological processes in patients with coexisting conditions. This case underscores several important clinical considerations. First, healthcare providers should maintain high clinical suspicion for secondary infections in psoriatic patients, particularly when treatment response is suboptimal or varies across anatomical sites. Second, comprehensive diagnostic evaluation incorporating both histopathological and microbiological methods is essential for accurate diagnosis of complex dermatological presentations. Third, therapeutic management must be tailored to address all identified pathological processes while considering potential drug interactions and contraindications (Diksis et al., 2019).

This ongoing case provides important preliminary lessons for clinicians: differential treatment response across anatomical sites should serve as a red flag for secondary infections, necessitating mycological examination before intensifying immunosuppressive therapy in treatment-resistant psoriatic lesions. The early clinical improvement observed after three weeks of antifungal therapy with temporary corticosteroid discontinuation supports the critical importance of prioritizing fungal eradication over psoriasis control in cases of confirmed coexistence, though long-term outcomes and optimal treatment duration remain to be determined through continued follow-up.

4. CONCLUSION

This case report demonstrates the diagnostic and therapeutic challenges associated with coexisting psoriasis vulgaris and tinea pedis, where differential treatment response across anatomical sites served as a critical indicator for comprehensive diagnostic evaluation. The successful management required objective confirmation through histopathological examination and fungal culture, temporary discontinuation of topical corticosteroids, and implementation of targeted antifungal therapy, resulting in significant clinical improvement. This case emphasizes the importance of maintaining high clinical suspicion for secondary infections in psoriatic patients with suboptimal treatment response and highlights the necessity of systematic diagnostic evaluation incorporating multiple modalities for optimal patient outcomes in complex dermatological presentations.

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