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Case Report

Tinea corporis overlying post-herpetic neuralgia: a novel manifestation of wolf's isotopic response

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ABSTRACT

Wolf's isotopic response is characterized by the occurrence of a new dermatosis at the site of a previously healed and unrelated skin disorder, most commonly following herpes zoster. A wide spectrum of secondary dermatoses has been described, including granulomatous, lichenoid, autoimmune, and neoplastic conditions. However, infectious etiologies-particularly superficial fungal infections-are exceedingly rare in this setting. We report the case of a 58-year-old female who developed tinea corporis localized strictly to the same dermatome previously affected by herpes zoster and persistent post-herpetic neuralgia. Clinical examination revealed an annular erythematous scaly plaque over the left T11 dermatome, corresponding to the healed zoster site. Potassium hydroxide examination demonstrated septate branching hyphae, confirming dermatophytosis. The strict dermatomal localization, along with the temporal sequence of events, supports the diagnosis of Wolf's isotopic response. To the best of our knowledge, this is the first reported case of dermatophytosis occurring over a site affected by post-herpetic neuralgia. This case highlights the role of persistent neuroimmune dysregulation in creating a localized area of immune vulnerability, predisposing to secondary infections.

Keywords: Wolf's isotopic response, Herpes zoster, Dermatophytosis, Persistent post-herpetic neuralgia

INTRODUCTION

Wolf's isotopic response, first described by Wolf et al., refers to the occurrence of a new, unrelated dermatosis at the site of a previously healed skin disease, most frequently herpes zoster.[1] This phenomenon is distinct from the Koebner response, where lesions of the same disease occur at sites of trauma, and instead represents a unique site-specific susceptibility to unrelated dermatological conditions.

Herpes zoster is the most commonly implicated primary dermatosis, owing to its profound impact on both cutaneous nerves and local immune responses.[2,3] Following viral reactivation, significant structural and functional alterations occur within the affected dermatome, including neuronal damage and immune dysregulation. A wide range of secondary dermatoses has been reported in this context, including granulomatous conditions such as granuloma annulare, lichenoid eruptions, autoimmune diseases, and malignancies such as cutaneous lymphomas.[2]

Despite the diversity of reported isotopic responses, infectious dermatoses remain relatively uncommon. Among these, bacterial and viral infections have occasionally been described, whereas fungal infections such as dermatophytosis are exceedingly rare.[3,4] This rarity is notable given the high prevalence of dermatophytosis in the general population, suggesting that specific local factors may influence susceptibility in isotopic responses.

Post-herpetic neuralgia, a chronic complication of herpes zoster, is characterized by persistent neuropathic pain due to irreversible nerve injury. The resulting neuroimmune alterations may persist long after resolution of the primary infection, creating a localized "immunocompromised district," which predisposes the affected skin to secondary dermatoses.[5,6]

We report a rare case of tinea corporis occurring over a dermatome affected by herpes zoster and persistent post-herpetic neuralgia, representing a novel manifestation of Wolf's isotopic response.

CASE REPORT

A 58-year-old female presented with complaints of an itchy, erythematous, scaly lesion over the left side of her abdomen for one month. The itching was insidious in onset, mild to moderate in intensity, and gradually progressive. There was no associated pain, discharge, or systemic symptoms.

The patient had a documented history of herpes zoster involving the same region four months prior. She had received appropriate antiviral therapy during the acute phase, following which the lesions resolved with residual hyperpigmentation. However, she continued to experience persistent burning pain and dysesthesia over same dermatome, consistent with post-herpetic neuralgia.

There was no history of similar lesions elsewhere on the body. The patient denied any history of diabetes mellitus, immunosuppression, or use of systemic or topical corticosteroids. There was no history suggestive of contact exposure/similar complaints in family members.

On cutaneous examination, a single, well-defined annular erythematous plaque measuring approximately 5×4 cm was present over the left flank. The lesion showed central clearing with active erythematous, scaly margins. The distribution was strictly dermatomal, corresponding to the T11 dermatome, and did not cross the midline. A healed, slightly atrophic scar corresponding to the previous herpes zoster eruption was noted within the same region.

No satellite lesions were observed, and examination of hair, nails, and mucosae was unremarkable. There was no regional lymphadenopathy.

Based on the clinical morphology, differential diagnoses included dermatophytosis, annular psoriasis, nummular eczema, and erythema annulare centrifugum.

Potassium hydroxide examination of skin scrapings obtained from the active margin of the lesion demonstrated multiple septate branching hyphae, confirming dermatophytosis.

Considering the strict dermatomal localization, temporal relationship with prior herpes zoster, and absence of lesions elsewhere, a diagnosis of tinea corporis occurring as Wolf's isotopic response over a site of post-herpetic neuralgia was established.

Treatment and follow-up

The patient was initiated on oral antifungal therapy along with topical antifungal agents. Neuropathic pain was managed with gabapentin.

At 2-week follow-up, there was a significant reduction in erythema, scaling, and pruritus, indicating a favorable therapeutic response.

At 4 weeks, the lesion showed near-complete resolution with residual post-inflammatory hyperpigmentation. Repeat potassium hydroxide examination at this stage was negative for fungal elements, confirming mycological cure.

At 8 weeks, there was no evidence of recurrence of dermatophytosis. However, the patient continued to experience mild residual neuropathic symptoms, suggesting persistence of post-herpetic neuralgia.

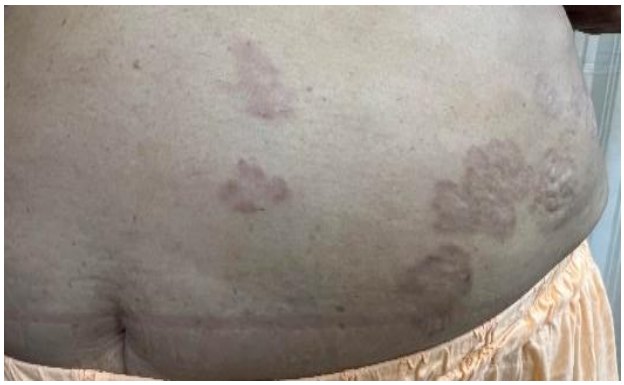

Figure 1: Tinea corporis overlying post-herpetic neuralgia.
*Well-demarcated, erythematous, annular scaly plaque, crossing midline present over left flank, atrophic scar present over left side of the flank.

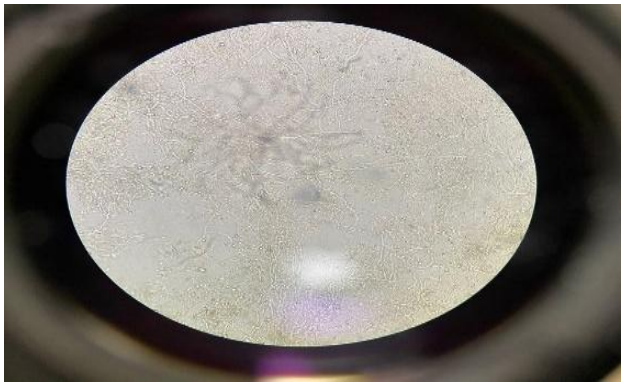

Figure 2: Tinea corporis overlying post-herpetic neuralgia.
*KOH mount showing septate hyphae.

DISCUSSION

Wolf's isotopic response is most frequently associated with prior herpes zoster infection, likely due to long-lasting alterations in both neural and immune components of the skin.[1,2]

In a study by Wang et al a variety of secondary dermatoses were reported following herpes zoster, including granulomatous and neoplastic conditions; however, dermatophytosis was notably absent.[2] Similarly, Neema et al. reported psoriasis arising over healed zoster lesions, highlighting the heterogeneous nature of isotopic responses.[7]

The pathogenesis of Wolf's isotopic response is complex and involves a combination of immune dysregulation and neural damage. Herpes zoster infection leads to depletion of Langerhans cells, impaired antigen presentation, and alterations in cytokine expression.[4] These changes compromise local immune surveillance and may persist even after clinical resolution of the infection.

Additionally, nerve injury plays a crucial role in this phenomenon. Damage to sensory nerves results in altered release of neuropeptides such as substance P, calcitonin gene-related peptide, and vasoactive intestinal peptide.[5] These neuropeptides regulate cutaneous immune responses and inflammation, and their dysregulation may lead to a localized imbalance in immune homeostasis.

The concept of an "immunocompromised district" provides a unifying explanation for such phenomena.[3,8] According to this concept, areas of the skin subjected to prior injury or insult become selectively vulnerable to secondary diseases due to persistent immune dysregulation.

In the present case, the coexistence of post-herpetic neuralgia suggests ongoing neural dysfunction, which may further perpetuate this localized immunocompromised state.

Dermatophyte infections are typically controlled by cell-mediated immunity, particularly Th1 and Th17 pathways.[9] A shift toward a Th2-dominant immune response, as may occur in chronically altered neuroimmune environments, can impair antifungal defense mechanisms and facilitate dermatophyte infection.[9,10]

The strict dermatomal localization of the lesion, absence of involvement of other body sites, and clear temporal relationship with prior herpes zoster strongly support the diagnosis of Wolf's isotopic response in this case.

Although infectious isotopic responses have been described, fungal infections remain extremely rare, making this case a unique addition to existing literature.[11]

CONCLUSION

This case represents a rare and possibly first reported instance of tinea corporis occurring over a site affected by herpes zoster and persistent post-herpetic neuralgia as part of Wolf's isotopic response.

It emphasizes the importance of recognizing infectious dermatoses within this spectrum and highlights the role of persistent neuroimmune dysregulation in predisposing previously affected skin to secondary infections. Early recognition and appropriate management are essential to prevent misdiagnosis and ensure optimal patient outcomes.

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