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Postherpetic Psoriasis: A Rare Case Report

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Abstract

Wolf's isotopic response describes the phenomenon where a new, different skin disorder occurs at the site of a previously healed skin lesion. The most common primary and secondary diseases are herpes zoster and granuloma annulare, respectively. Psoriasis vulgaris is rare as a secondary disease, with only two cases reported. Here, we report another rare case of psoriasis vulgaris following a herpes zoster.

Keywords: Herpes zoster; Psoriasis vulgaris; Wolf's isotopic response

Introduction

Wolf's isotopic response, also known as the "isoloci response," describes the phenomenon where a new, different skin disorder occurs at the site of a previously healed skin lesion.[1] The precise pathogenesis of the phenomenon is yet unclear. Various hypotheses have been formulated to explain the etiopathogenesis of the phenomenon, including viral, vascular, neural, and immunological factors. A literature review revealed 176 reported cases of Wolf's isotopic response, with the primary diseases being herpes zoster and herpes simplex in 88.6% and 11.4% of cases, respectively. Granuloma annulare was the most common secondary disease comprising 18% of the total cases.[2] There are about 200 reported cases of Wolf's phenomenon, with only two cases of psoriasis vulgaris as the secondary disease.[3,4] We here report another rare case of psoriasis vulgaris following herpes zoster.

Case Report

A 68-year-old lady presented with multiple erythematous papules and plaques with silver-colored scales on the left T5-6 thoracic dermatomal area for 1 week (Figure 1a, b). It was associated with mild pruritus. She gave a history of herpes zoster 4 weeks back in the same dermatomal area. There was no history of psoriasis, no family history of psoriasis, and no history of any medications. The patient did not have hypertension, dyslipidemia, or diabetes mellitus. On scraping, the plaque surface with a blunt glass side, scales fell off as layers, revealing pinpoint bleeding on further scarping. Multiple hypo-pigmented macules and scars from healed herpes zoster lesions were present. Examining the scalp, nail, and skin at other sites was normal. A biopsy was sent from the plaque with a provisional diagnosis of psoriasis vulgaris (Figure 2 a, b).

Histopathological examination showed confluent parakeratosis with neutrophilic collection, orthokeratosis, hypo to agranular layer, regular acanthosis, and perivascular infiltration with lymphocytes and histiocytes (Figure 2a, b).

Discussion

The "isotopic response" refers to a new, unrelated skin disorder occurring at the site of a previously healed skin condition, whereas the "isomorphic response"

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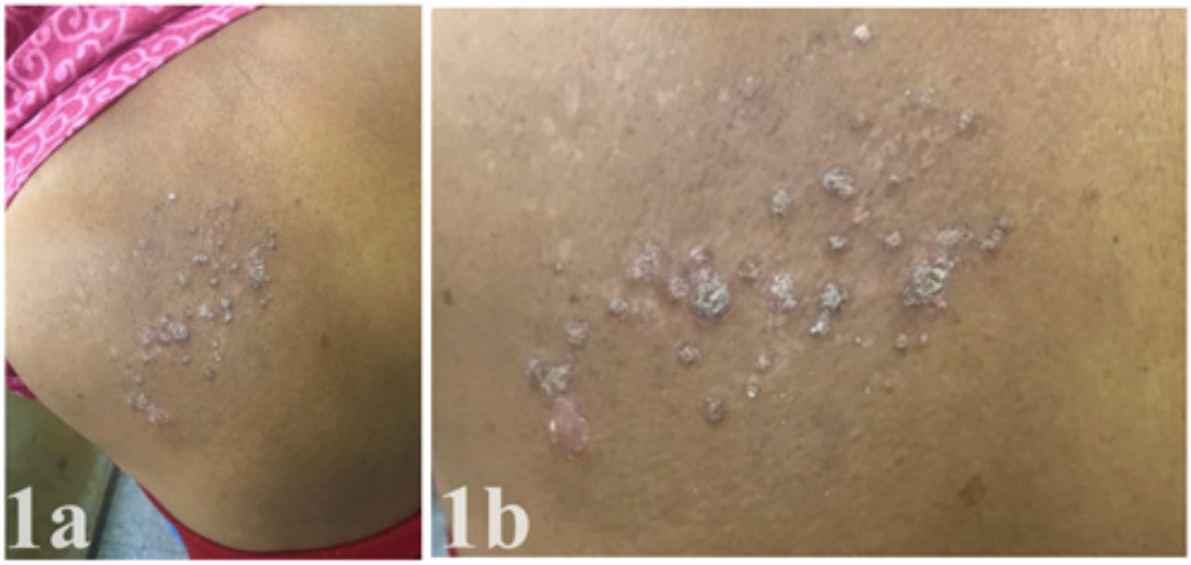

Figure 1(a, b): Multiple erythematous papules and plaques with silver-colored scales on the left T5-6 thoracic dermatomal area

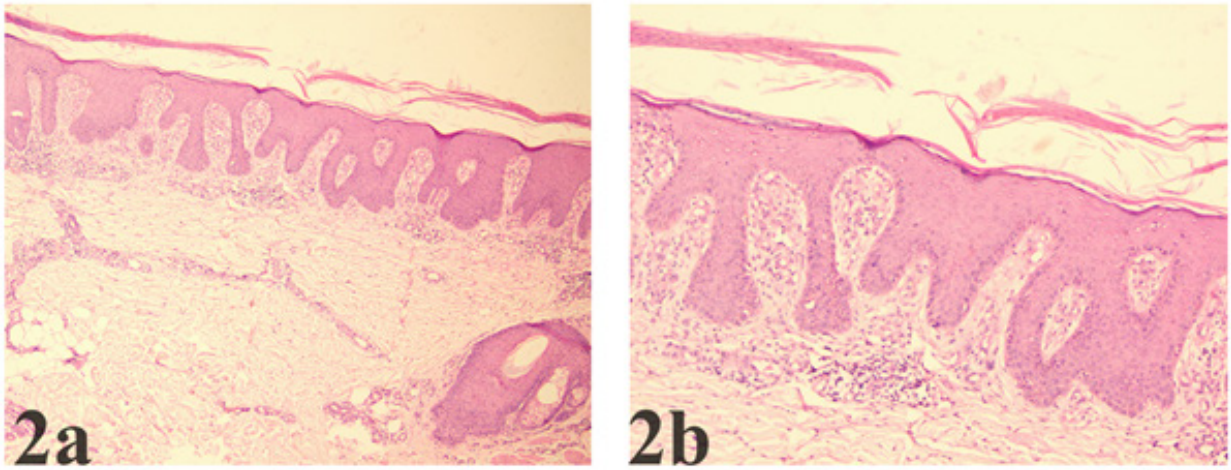

Figure 2 a (10X) b (40X) showing confluent parakeratosis with neutrophilic collection, orthokeratosis, hypo to agranular layer, regular acanthosis, and perivascular infiltrate with lymphocytes and histiocytes (H and E stain)

involves a skin lesion similar to an existing disease appearing at the site of an injury. To clarify, the term "isotopic response" should only apply to new skin diseases that appear at the site of a previously healed skin condition, where the skin looks clinically normal or has minimal scarring. It should not include conditions like carcinoma developing on scarred tissue from lupus vulgaris. Additionally, the term should be restricted to skin diseases and not used for conditions caused by external factors like chemicals, radiation, or other traumas.[1]

Varicella-zoster virus (VZV) or herpes simplex virus (HSV) infections are the most frequent underlying skin conditions leading to an isotopic response. A quick isotopic response might be mistakenly identified as a recurrence of herpes zoster. Among the secondary skin diseases that follow a VZV/HSV infection, granulomatous reactions are most common (primarily granuloma annulare), malignancies (ranging from single tumors to leukemic or lymphomatous infiltrations), immune disorders (such as lichen planus and allergic contact dermatitis), infections (viral, bacterial, and fungal), and other conditions like acneiform lesions and reactive perforating collagenosis.[5-8] Isotopic responses are rare, and psoriasis as an isotopic response is even rarer, with only a few reported cases.[3,4] The time between the initial herpetic infection and the onset of the second condition, known as Wolf's isotopic response, varies widely, from 15 days to 7 months.[9]

The exact cause of isotopic response remains unclear, though several theories, such as viral, vascular, immunological, and neural, have been proposed. The most widely accepted is the neural hypothesis. During herpes zoster, the varicella-zoster virus damages the A-delta and C nerve fibers. Additionally, neurohumoral factors trigger the development of a second skin

condition by releasing substances like substance P, bradykinin, serotonin, vasoactive intestinal peptide, calcitonin gene-related peptide, and α-melanocyte-stimulating hormone. These neuropeptides start the immune response that leads to the second disease. Furthermore, neurohumoral factors cause unchecked immune system activation, contributing to the isotopic response.[10] Allegue et al., suggested that tumor necrosis factor-alpha (TNF-alpha) plays a role in this response. TNF-alpha, known for its antiviral properties, is elevated in herpes zoster and psoriatic skin lesions. Therefore, it's hypothesized that increased TNF-alpha levels during herpes zoster could trigger psoriasis at the healed sites.[4]

In our study, a 68-year-old woman developed psoriasis just 4 weeks after recovering from herpes zoster, indicating a relatively quick onset. This contrasts with a 35-year-old woman from another study who developed psoriasis 6 months after her shingles had healed, suggesting age-related differences in the timing of psoriasis following herpes zoster. Another study involving a 41-year-old man further highlights this variability, emphasizing that the link between herpes zoster and psoriasis onset can differ significantly depending on age and gender.[3,4] This variability suggests that the mechanisms connecting herpes zoster and psoriasis are complex and influenced by individual health and immune status.

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