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CASE REPORT

Delving into Wolf's Isotopic Phenomenon: Tale of Two Cases of Psoriasis Localized to the site of Scars

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ABSTRACT

Wolf's isotopic response refers to the occurrence of a new dermatosis at the site of a previously healed and unrelated skin condition. While commonly associated with viral triggers such as herpes simplex or zoster, non-viral initiators are rarely reported. We present two unique cases of psoriasis developing exclusively at sites of old scars from non-viral causes. The first case involved a 33-year-old male who developed a psoriatic plaque over a thermal burn scar on the dorsum of the hand. The second case featured a 28-year-old male with psoriatic lesions localized to an atrophic scar following a traumatic injury to the shin. Both cases lacked involvement of other body areas. Diagnosis was confirmed by clinical features, dermoscopy, and histopathology. These cases highlight an atypical presentation of Wolf's isotopic phenomenon and underscore the potential role of localized immune dysregulation and scar-mediated vulnerability in the development of secondary dermatoses.

KEYWORDS

• Wolf's Isotopic Response • Psoriasis • Burn Scar • Post-Traumatic Scar • Locus Minoris Resistentiae • Non-Viral Primary Dermatosis Isotopic Phenomenon • Scar-Associated Dermatosis

INTRODUCTION

"Wolf's isotopic response" refers to the occurrence of a new dermatosis at the site of previously healed, unrelated dermatosis. A number of factors including viral, neural, vascular, and immunologic, have been implicated in the causation of this response.[1] The commonly described initiator skin diseases

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are either herpes simplex or herpes zoster, both of which have viral etiology.[2] There exists scarcity of literature describing few anecdotal reports of Wolf's isotopic response, where the first dermatosis is non-viral. We hereby report two cases, wherein the primary dermatoses were non-viral, followed by scarring. The first case describes a middle-aged male, who developed psoriasis at the site of burn scar and the second describes development of psoriasis at the site of previous trauma. Both the cases didn't portray affliction of any other body sites.

Case 1

A 33-year-old male presented to dermatology outpatient department with, single, minimally itchy, red raised lesion over dorsum of his right hand since 2 months. On enquiry, a past history of second-degree, hot oil induced thermal burn, eight months ago, which was followed by scarring at the same site, was unveiled. Patient denied presence of similar lesions over the other body sites, photo exacerbation, topical irritant application, arthritis, nail changes or any other systemic complaints. He denied history of manipulation of the lesion. Patient did not give history suggestive of herpes infection, at the same site, prior to onset of current lesion. He denied any significant medical, surgical or past treatment history. Cutaneous examination revealed single, erythematous plaque of size 3 centimeters (cm) by 2 cm, associated with minimal scaling, and surrounding rim of atrophic burn scar (Figure 1 A). Upon scraping the lesion with blunt edge of a glass slide, multiple pin-point bleeding spots were appreciated, indicative of positive Auspitz's sign. With the highest clinical surmise of psoriasis, the other clinical differential included eczema. Dermoscopy (DinoLite II, Pro HR dermatoscope) at 10x magnification, in polarized mode revealed, regularly arranged red dots indicative of dotted vessels, on diffuse pink background (Figure 1 B). Routine investigations including complete hemogram, liver and renal function tests and serum electrolytes, were unremarkable.

A skin punch biopsy from the lesion depicted, regular acanthosis with club shaped, elongated rete ridges, focal parakeratosis, hypogranulosis, suprapapillary thinning and dilated blood vessels in the papillary dermis. This helped us clinch the diagnosis of Psoriasis, based upon clinical, dermoscopic and histopathological correlation. Owing to the localized nature of disease, the patient was treated with topical mometasone furoate 0.1% cream, to be applied twice a day, and was advised ample use of topical emollients. After three weeks of the above treatment, he portrayed significant improvement and is currently under follow up.

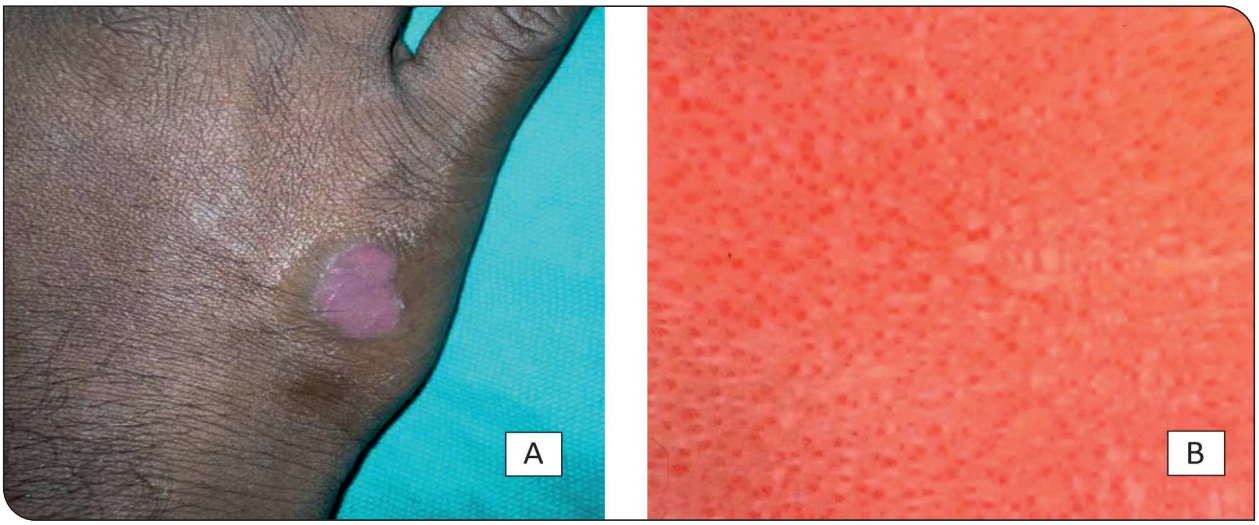


Figure 1: (A) Clinical image depicting erythematous plaque (3 cm x 2cm) with minimal scaling, present over dorsum of right hand, over an atrophic burn scar, **(B)** Dermoscopy (DinoLite II, Pro HR dermatoscope) at 10x magnification, in polarized mode revealed, regularly arranged red dots indicative of dotted vessels, on diffuse pink background

Case 2

A 28-year-old male presented with minimally itchy, red raised skin lesions, associated with flaking, over left lower leg in the past 8 months. On enquiry, the patient informed about history of trauma (road traffic accident) to the affected

area, 15 years ago. There is neither any history of similar lesions elsewhere on the body nor any systemic complaints. Patient did not give history suggestive of herpes infection at the same site, prior to the onset of current lesions. History of winter exacerbation of the lesions was prominent. Cutaneous examination depicted multiple erythematous plaques ranging in size from 1.5 cm x 1 cm to 3 cm x 2 cm with minimal scaling, present over left lower shin, overlying an atrophic post-traumatic scar [**Figure 3 (A and B)**] Treatment history was unremarkable. Upon scraping the lesion with blunt edge of a glass slide, multiple pin-point bleeding spots were observed, indicative of positive Auspitz's sign. Polarised dermoscopy (DinoLite II pro HR dermatoscope) at 10x magnification highlighted regularly arranged red dots, reminiscent of dotted vessels, on diffuse pink background (**Figure 3 C**)

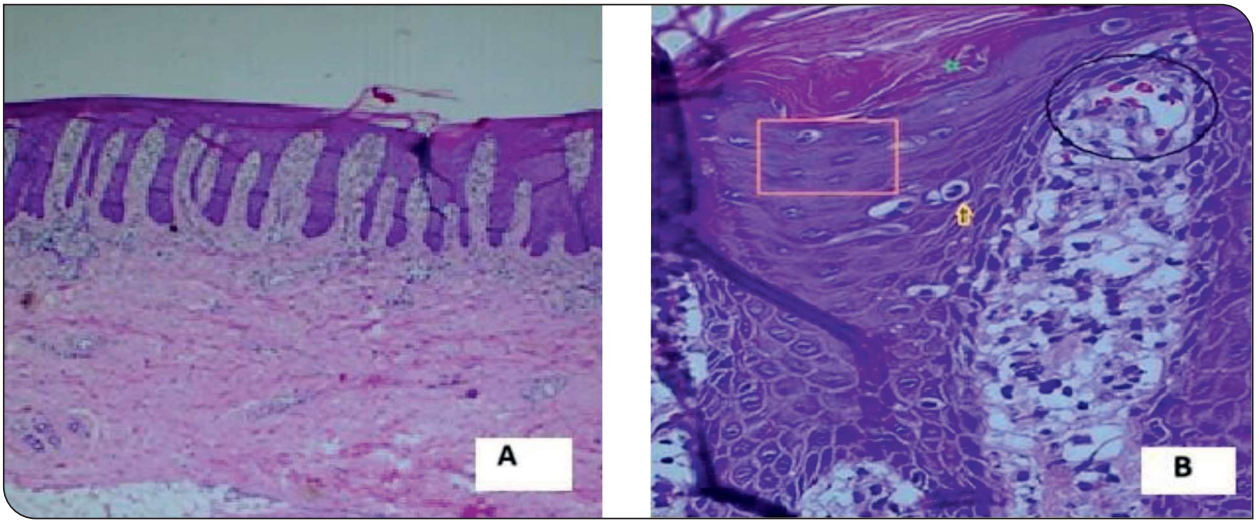


Figure 2: [**A**] Regular acanthosis with club-shaped rete ridges and superficial dermal, perivascular infiltrate (H & E, 4X), **[B]** Parakeratosis (green star), hypogranulosis (orange rectangle), neutrophilic exocytosis (yellow arrow) and dilated papillary dermal blood vessels (black circle) (H &E, 40 X) .

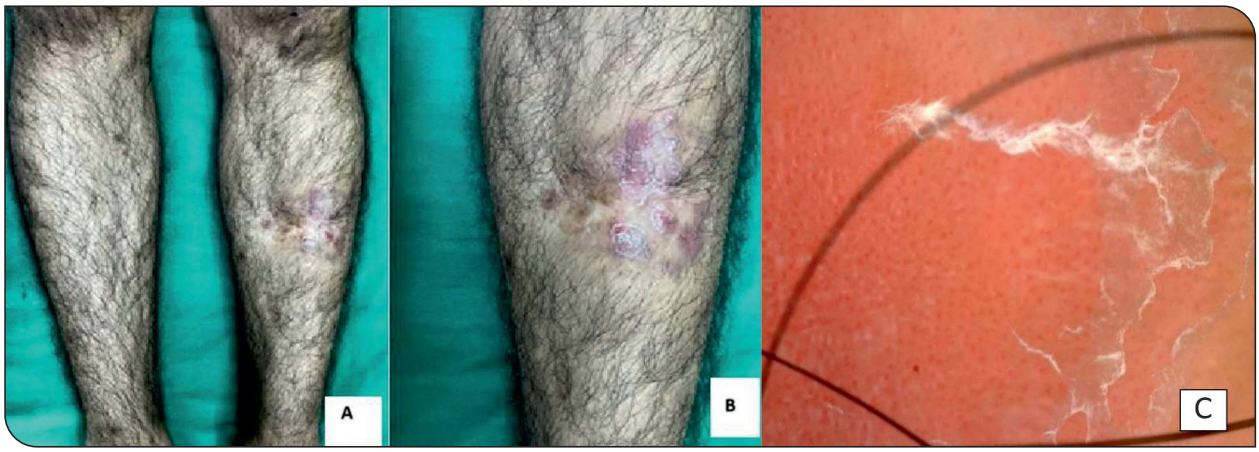


Figure 3: (A & B) Clinical image depicting multiple erythematous plaques ranging in size from 1.5 cm x 1 cm to 3 cm x 2cm with minimal scaling, present over left lower shin, overlying an atrophic post- traumatic scar, Polarised dermoscopy (DinoLite II pro HR dermatoscope) at 10x magnification highlighted regularly arranged red dots, (**C**) reminiscent of dotted vessels, on diffuse pink background

Provisional diagnosis of psoriasis was considered. Routine haematological evaluation including hemogram and complete metabolic panel including liver and renal function tests were within normal limits.

Histopathological analysis of skin punch biopsy revealed regular acanthosis, elongated rete pegs, Munro's microabscesses, dilated superficial dermal vessels with perivascular infiltrate (Figure 4 A & B). This confirmed the diagnosis of psoriasis at the site of traumatic scar.

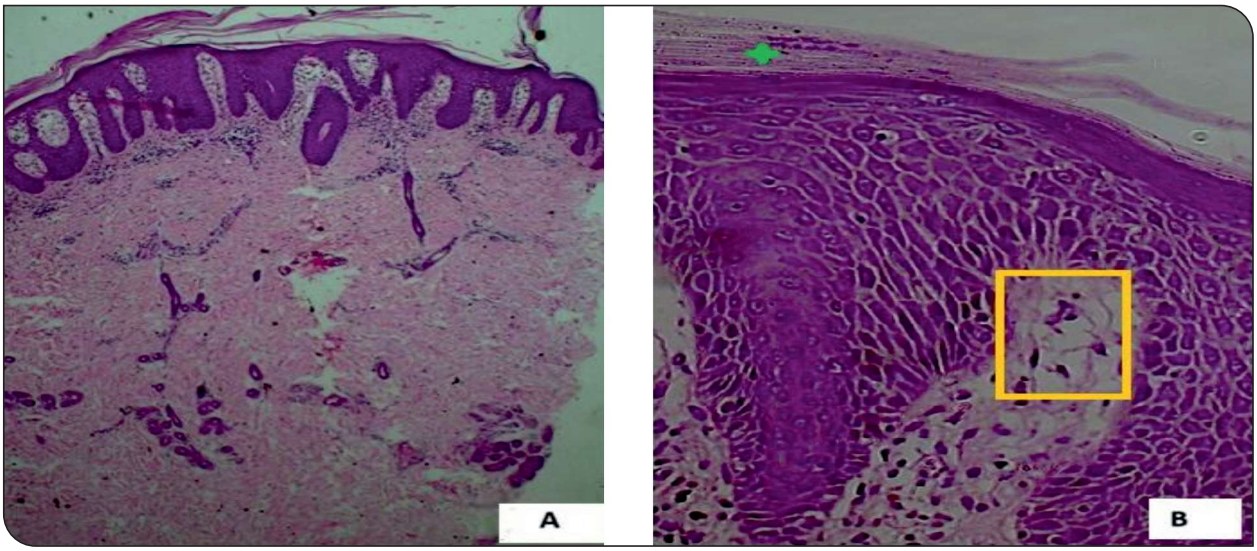
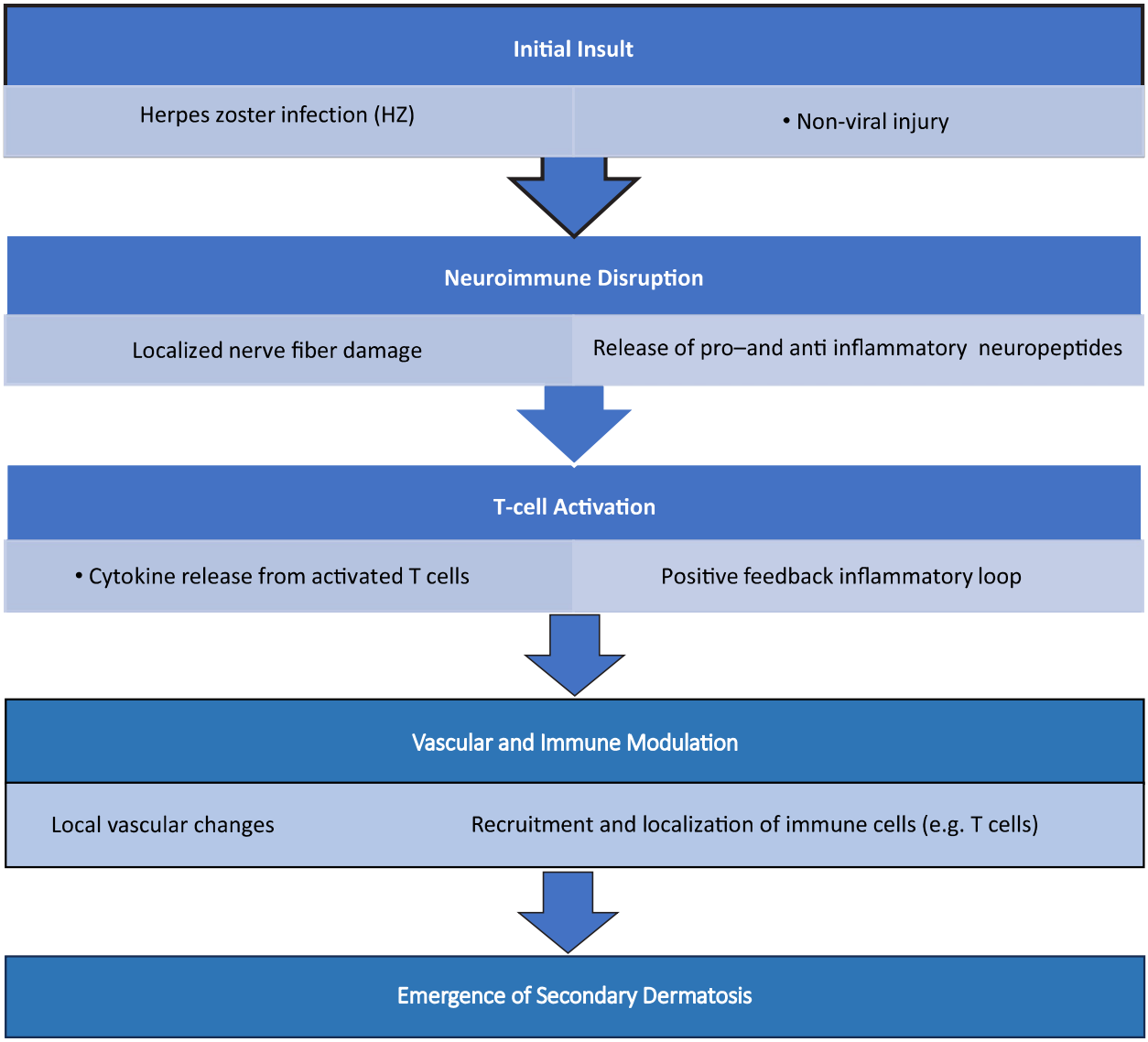


Figure 4: [A] Regular acanthosis with club-shaped rete ridges and superficial dermal, perivascular infiltrate (H & E, 4X), **[B]** Munro's microabscess (green star) and dilated papillary dermal blood vessels with perivascular infiltrate (yellow rectangle) (H &E, 40 X).



Considering the focal involvement, the patient was advised topical emollients along with mometasone furoate 0.1 % cream for twice daily application. Oral antihistamines were recommended (need-based) for controlling the pruritus and weekly Vitamin D3 (60,000 IU) supplementation was considered. After a month of above treatment, he displayed significant improvement and is under routine follow up.

DISCUSSION

In 1955, Wyburn-Mason, for the first time described the occurrence of a new skin disease at the site of another skin disease, that had already healed.[3] Such cases continued to mark their presence in literature till Wolf and Wolf in 1985 gave it a term; "isoloci response" (same locus).

This nomenclature was modified to "isotopic response" (same place) by Wolf *et al.*, and finally reframed as Wolf's isotopic response by Ruocco *et al.*[4] Multitudinous etiological factors such as viral, immunological, neural, vascular or locus minoris resistentiae (place of less resistance) have been implicated in the pathogenesis of isotopic response.[5]

Pathophysiological cascade involved in Wolf's isotopic response is depicted in Table 1.[1]

The association of Wolf's isotopic phenomenon at the site of healed herpes zoster is well established. However, the descriptions of non-viral primary dermatoses have been anecdotal and are included in the table below (Table 2).

Table 1: Pathophysiological cascade involved in Wolf's isotopic response

Serial No.	Body Region	Primary Condition	Subsequent Condition	Reference
1	Upper arm	Injection-induced trauma	Urticaria	[6]
2	Cheek	Cutaneous leishmaniasis	Discoid lupus erythematosus	[7]
3	Abdomen	Striae distensae	Leukemia cutis	[8]
4	Back and arm	Burns	Scar sarcoidosis	[9]
5	Back	Burns	Molluscum contagiosum	[10]
6	Shoulder	Scrofuloderma	HSV infection	[11]
7	Supraclavicular area	Scrofuloderma	HSV infection	[12]
8	Arm	BCG vaccination scar	Positive vesicular response on patch test	[13]

The occurrence of isotopic response at the burn scar is underreported, with the available case report describing molluscum contagiosum at the burn site.[10]

In both our cases, we hypothesize that, thermal or physical trauma, may have led to the altered local immunological resistance, leading to a "weakened" focus (locus minoris resistentiae). This postulated regional immune-weakening can involve both cellular and humoral responses, depending on the reduction of phagocytosis and lymphocytic function. The other potential contributor is variation in loco-regional micro-circulation.[14]

Herpesviruses have an ability to elicit prolonged T cell mediated responses, via helper, cytotoxic and memory T-cell recruitment. This ability can be attributed to pro-inflammatory cytokines, neuropeptides, release of nerve growth factors and chemotactic substances. The mechanisms behind evocation of such enduring immune responses, in cases of non-viral primary dermatoses (our cases) seem elusive.

Wolfs isotopic phenomenon requires distinction from isomorphic (Koebner's phenomenon).

The **isomorphic response** denotes the emergence of identical dermatoses at sites of trauma, typically observed in conditions such as psoriasis, lichen planus, and vitiligo. This phenomenon is also presumably mediated by immunologic mechanisms and illustrates how mechanical or inflammatory insult to previously unaffected skin can precipitate the same pathological process.

In contrast, the **isotopic response** refers to the development of a novel and etiologically unrelated dermatosis at the site of a previously healed skin disorder. The secondary lesion arises in an area that appears clinically normal following resolution of the initial condition. Crucially, the second disease must be histologically and pathophysiologically

distinct from the first. Telltale examples of secondary dermatoses include plethora of inflammatory, infectious, granulomatous diseases and lymphomas or other neoplasms at the site of resolved herpes zoster infection.[15]

CONCLUSION

Our cases highlight psoriasis arising as a Wolf's isotopic response, occurring at the site of a prior thermal burn or physical trauma, with no other cutaneous involvement. This underscores the role of localized immune dysregulation and locus minoris resistentiae in post-traumatic dermatoses. Recognition of such atypical presentations is essential for accurate diagnosis and appropriate management. Further studies are warranted to elucidate the pathomechanisms involved in isotopic responses, where initiator condition is non-viral.

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