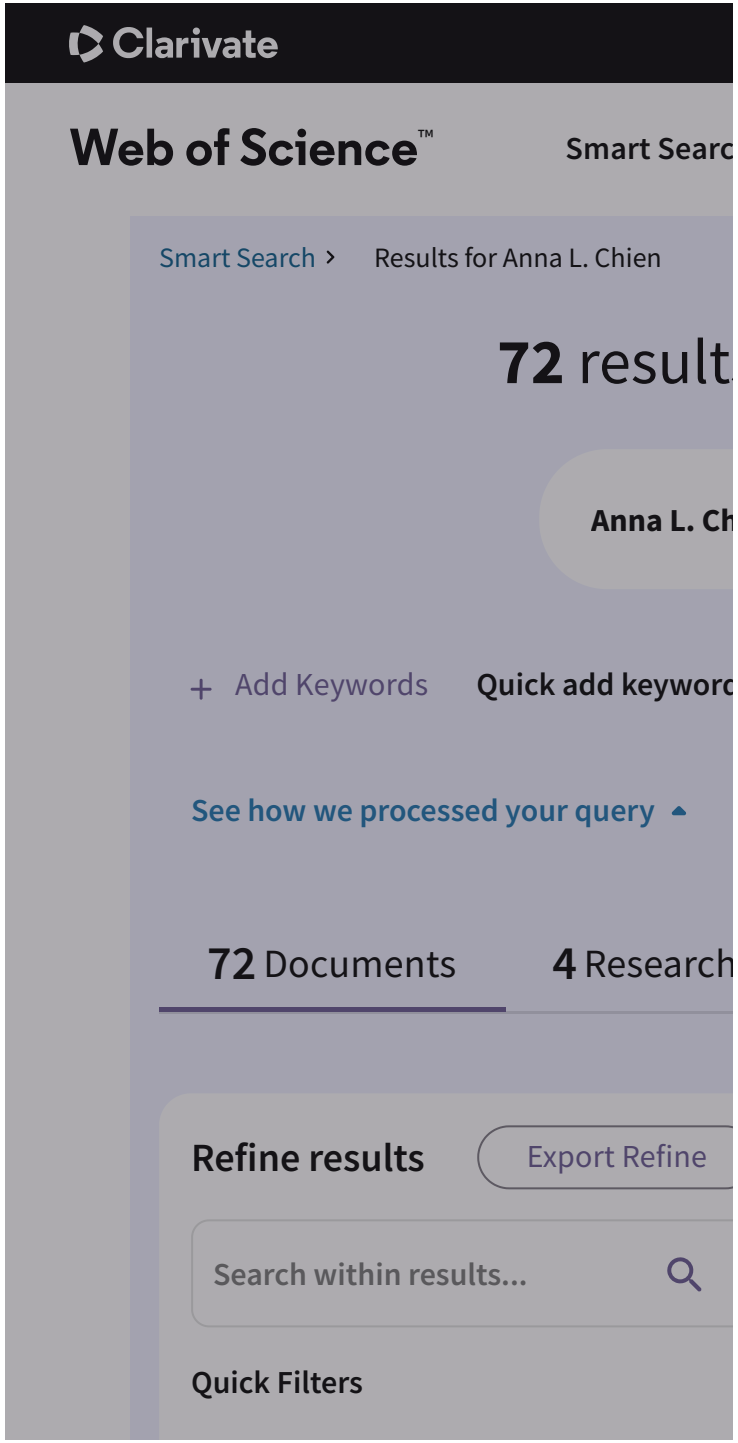


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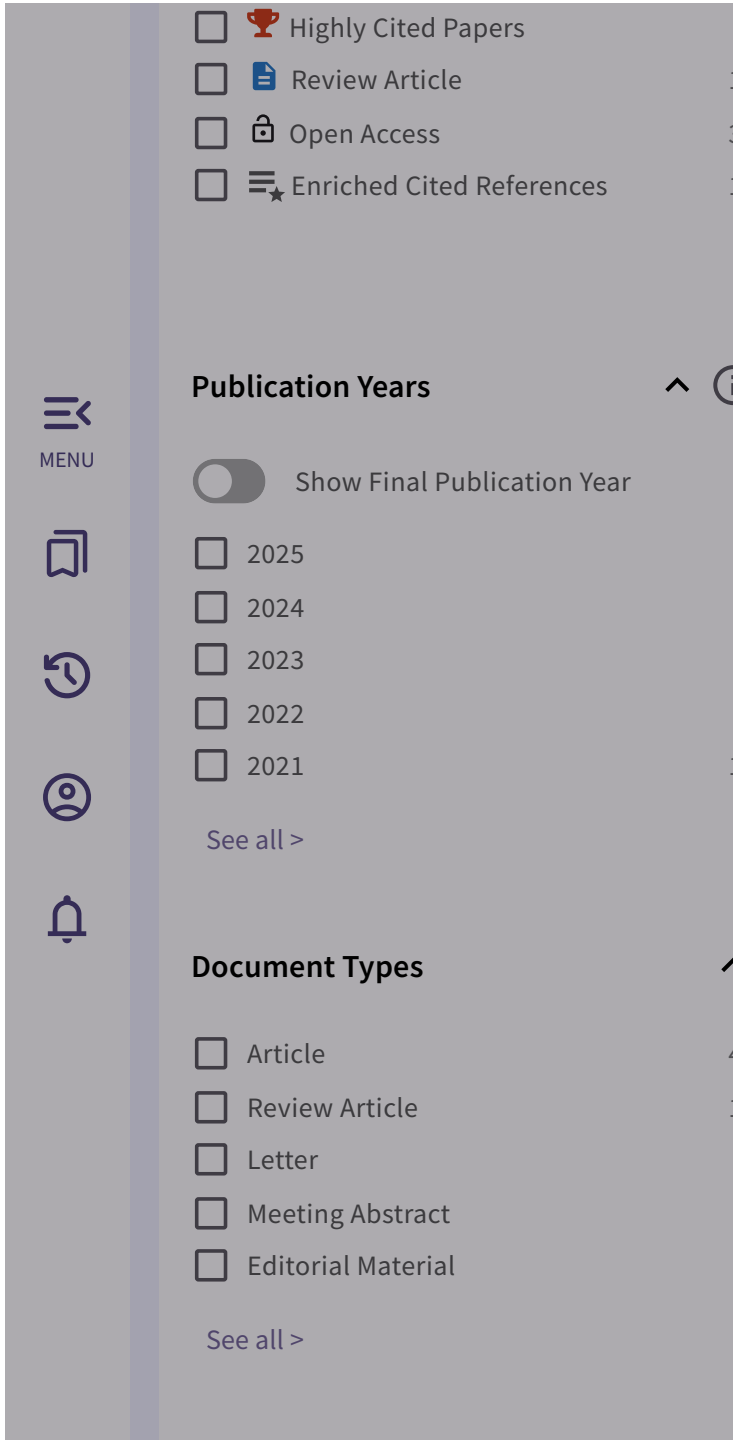
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Photodermatology Photoimmunology & Photomedicine



THE OFFICIAL PUBLICATION OF THE PHOTOMEDICINE SOCIETY, THE BRITISH PHOTODERMATOLOGY GROUP, THE EUROPEAN SOCIETY FOR PHOTODERMATOLOGY, THE KOREAN SOCIETY FOR PHOTOMEDICINE AND THE TAIWANESE PHOTOMEDICINE SOCIETY

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LETTER

Selective Involvement of Photoprotected Skin in an IL-36-Driven Pustular Eruption: A Possible Reverse Recall Phenomenon

Evren Odyakmaz Demirsoy[1] | Semanur Çakır Serinbaş[1] | Dilek Bayramgürler[1] | Busra Yaprak Bayrak[2] | Rebiay Kıran[1]

[1]Department of Dermatology, Faculty of Medicine, Kocaeli University, Kocaeli, Turkey | [2]Department of Pathology, Faculty of Medicine, Kocaeli University, Kocaeli, Turkey

Correspondence: Evren Odyakmaz Demirsoy (evrenodyakmaz@yahoo.com)

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Ultraviolet radiation (UV) is one of the most powerful modulators of cutaneous immunity and has long been used to treat psoriasis by suppressing IL23/IL17-mediated inflammation and restoring immune homeostasis. Beyond its immediate anti-inflammatory effects, UV exposure induces long-lasting changes in the cutaneous immune microenvironment through depletion of antigen presenting cells, induction of regulatory T cells, and modulation of tissue-resident immune populations. These effects may persist for weeks or even months and contribute to a sustained increase in the inflammatory threshold of previously irradiated skin [1, 2]. Because both generalized pustular psoriasis (GPP) and acute generalized exanthematous pustulosis (AGEP) are increasingly recognized as IL-36 driven neutrophilic disorders, it is conceivable that prior UV exposure may also influence the phenotype and anatomical distribution of subsequent pustular eruptions. However, clinical evidence demonstrating that antecedent UV exposure can induce site specific photobiological imprinting remains scarce. Here we describe a child with IL36RN associated GPP who developed a mild drug induced pustular eruption selectively affecting photoprotected skin, whereas previously sun exposed areas remained strikingly spared.

A 7-year-old girl with known pustular psoriasis presented with a two-day history of fever and the sudden onset of a generalized erythematous rash. The patient had been diagnosed with generalized pustular psoriasis (GPP) at 4 months of age and had remained stable for 6 months on acitretin (10 mg/day). One day prior to the onset of the eruption, she received amoxicillin–clavulanic acid for upper respiratory tract symptoms, including rhinorrhoea and mild cough, along with mild neutrophilia. She had experienced a similar eruption one year earlier after receiving the same antibiotic.

On examination, erythema was present on the eyelids, submental region, bilateral flexures, and the trunk areas previously covered by a bathing suit worn during a seaside vacation approximately 6 weeks before the onset of the eruption (Figure 1a–f). The eruption was predominantly erythematous, with only sparse, barely perceptible pustules on the lumbar region, the elbow and arms (Figure 2a). Geographic tongue was also noted on oral examination (Figure 2b).

Laboratory investigations revealed leukocytosis (17,000/mm^3), neutrophilia (14,300/mm^3), elevated C-reactive protein (77.32 mg/L), and erythrocyte sedimentation rate (85 mm/h). Histopathological examination revealed irregular psoriasiform acanthosis with elongation and partial fusion of rete ridges, prominent spongiosis, and focal intraepidermal neutrophilic aggregates consistent with early pustule formation. The superficial dermis showed papillary edema and a predominantly perivascular inflammatory infiltrate composed of lymphocytes with admixed neutrophils. These findings were compatible with a spongiotic neutrophilic dermatitis in the appropriate clinical context (Figure 2c,d). Genetic analysis revealed a heterozygous IL36RN variant of uncertain significance (NM_012275.3:c.91C>A).

Despite the limited number of pustules, the presence of geographic tongue, neutrophilia, elevated acute-phase reactants,

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FIGURE 1 | Selective involvement of photoprotected skin corresponding to prior bathing suit coverage. (a) Erythema involving the eyelids, submental region, and lateral neck areas fully shielded from sunlight during the recent seaside vacation. (b) Generalized erythematous eruption confined to UV-protected regions of the trunk and upper limbs, with clear sparing of previously sun-exposed areas. (c) The bathing suit worn 6 weeks earlier, illustrating the borders of prior UV exposure on the anterior trunk. (d) Posterior view showing marked erythema of the buttocks, lower back, and posterior thighs, sharply demarcated at the bathing suit line. (e) Complete sparing of the sun-exposed upper back and shoulders, with erythema limited to photoprotected skin. (f) Posterior view of the bathing suit demonstrating precise correlation between previously UV-exposed areas and the spared regions during the eruption.

spongiotic dermatitis, and a prior pustular reaction to the same drug supported a drug-induced IL-36-mediated pustular eruption.

Although the clinical distinction between AGEP and drug-triggered generalized pustular psoriasis could not be definitively established in our patient, both entities share IL-36-dependent neutrophilic inflammation as a central pathogenic mechanism [3]. Remarkably, the eruption of our patient was strictly limited to areas of the skin that had been shielded from sun exposure during a seaside vacation. Previously sun-exposed areas were completely spared. We speculated that the peculiar distribution of the eruption reflected a long-lasting UV-induced photohardening effect. Exposure to UV radiation initiates multiple molecular

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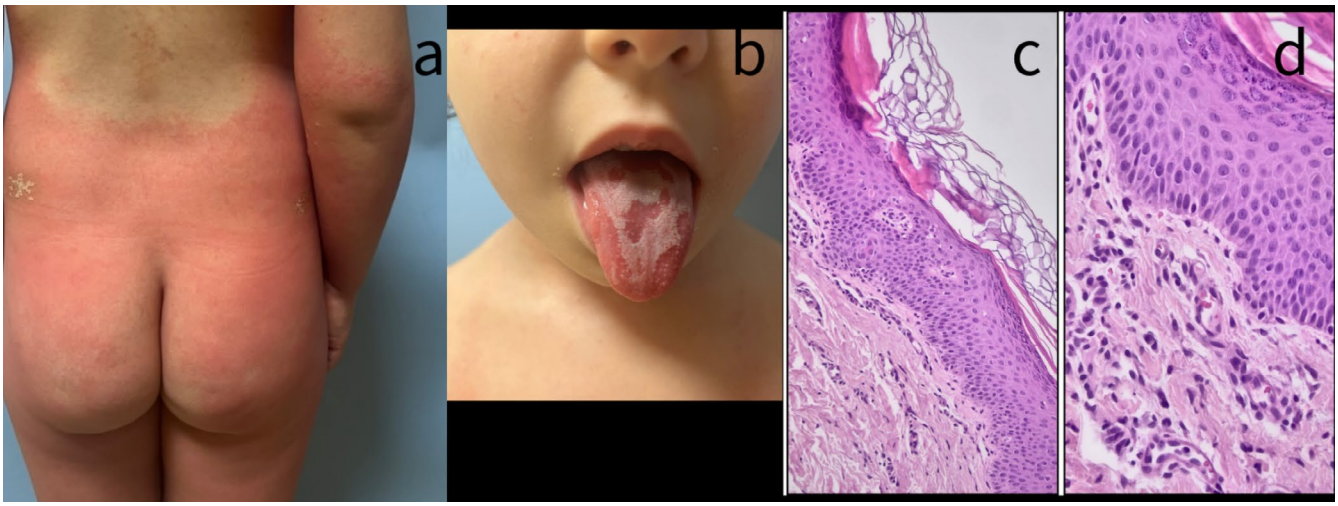


FIGURE 2 | (a) Postpustular desquamation on the lumbar region, sparse, tiny nonfollicular pustules around the elbows. (b) Geographic tongue. (c, d) Superficial perivascular spongiotic dermatitis with irregular acanthosis, spongiosis, elongation and partial fusion of rete ridges, and focal neutrophilic aggregates within the epidermis (H&E ×200, ×400).

events, including DNA photoproduct formation, cis-urocanic acid isomerization, platelet-activating factor production, and aryl hydrocarbon receptor activation, which subsequently induce a tolerogenic cutaneous microenvironment characterized by depletion and functional impairment of antigen-presenting cells, expansion of regulatory T cells, increased IL-10 production, and downregulation of the IL-23/Th17 pathway [1, 4]. These local immunomodulatory effects are known to persist beyond the immediate post-exposure period and are largely confined to previously irradiated skin. Accordingly, 6 weeks after sun exposure, the uncovered areas may have remained relatively protected from IL-36-driven neutrophilic inflammation, whereas the bathing suit-covered skin, lacking this UV-induced photobiological imprint, remained susceptible to the drug-triggered inflammatory cascade. Moreover, because UV-mediated immune regulation appears to modify rather than completely abolish inflammatory responses, partial suppression of neutrophil recruitment may explain the remarkably sparse and barely perceptible pustules observed in our patient. Thus, antecedent UV exposure may have acted as a local disease modifier, producing a reverse recall-like phenomenon characterized by selective sparing of previously sun-exposed skin and attenuation of pustule formation.

In addition, antecedent UV exposure may have interfered with the reactivation of previously primed drug-specific memory T cells [5]. By reducing antigen-presenting cell function and promoting a tolerogenic milieu enriched in regulatory T cells and IL-10, UV-exposed skin may have become relatively refractory to local recall responses upon reexposure to amoxicillin-clavulanate. Consequently, the inflammatory cascade could be preferentially amplified in photoprotected skin, resulting in a reverse recall-like distribution and only sparse pustulation. While AGEP and symmetrical drug-related intertriginous and flexural exanthema (SDRIFE) frequently involve UV-protected sites, this distribution has traditionally been attributed to occlusion, sweating, friction, or flexural anatomy. Our case raises the possibility that the relative absence of UV-induced immunoregulation may represent an additional and biologically plausible explanation.

Contact allergy to the bathing suit was considered unlikely for several reasons. The swimsuit had last been worn 6 weeks before the onset of the eruption and had not been used thereafter, making a causal relationship temporally implausible. In addition, the distribution of the lesions was not confined to areas of direct contact. Erythematous involvement was also observed on photoprotected, noncontact sites, including the eyelids and the submental (chin shadow) region. Taken together, the lack of temporal association and the involvement of noncontact photoprotected skin argues strongly against a contact-mediated mechanism.

Patch and intradermal tests were not performed because of parental refusal and the potential risk of disease reactivation. Therefore, the culprit drug was identified on the basis of the temporal relationship, clinical course, reproducibility with reexposure, and rapid resolution after drug withdrawal.

Overall, this case provides in vivo evidence that antecedent UV exposure can leave a durable site-specific photobiologic imprint capable of modifying both the severity and anatomical distribution of subsequent IL-36-driven drug eruptions. Such an effect may represent a previously unrecognized form of reverse recall phenomenon.

Author Contributions

Evren Odyakmaz Demirsoy: conceptualization; clinical evaluation; writing original draft; writing – review and editing; supervision. Semanur Çakır Serinbaş: case documentation; writing. Dilek Bayramgürler: diagnostic evaluation; supervision, review. Busra Yaprak Bayrak: histopathologic evaluation; interpretation of microscopic findings. Rebiay Kıran: review and editing.

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Consent

Written informed consent was obtained from the patient's legal guardian.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

Data sharing not applicable to this article as no datasets were generated or analysed during the current study.

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