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Revisiting Palmoplantar Inflammatory Dermatoses

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Introduction
Palmoplantar inflammatory dermatoses—chronic hand eczema (CHE), hyperkeratotic hand eczema (HHE), palmoplantar pustulosis (PPP), palmar psoriasis, and pompholyx—often show overlapping features, leading to diagnostic uncertainty and treatment selection. Because classification is largely morphology-based, biologically distinct entities may be grouped under broad labels such as “CHE.” Since 2020, a single referral center has conducted a series of studies using consistent histopathological criteria and immunohistochemical (IHC) methods to clarify whether key palmoplantar conditions align with the eczematous axis or a psoriasis-related spectrum. Here, we synthesize four investigations and propose a pathophysiology-oriented spectrum model integrating keratinocyte differentiation, inflammatory signatures, and real-world systemic treatment outcomes.

Materials and Methods
This narrative review consolidates four studies from one tertiary center using harmonized diagnostic frameworks. Palmar psoriasis, CHE/HHE, PPP, and pompholyx were assessed by routine histopathology and targeted IHC. Differentiation markers included basal keratins (K5/K14), palmoplantar keratin (K9), and involucrin, and psoriasis-associated mediators included β-defensin 2 and IL-36γ. Analyses compared marker distribution and intensity across diagnostic groups alongside histologic patterns (e.g., psoriasiform hyperplasia, granular layer changes, and parakeratosis). A retrospective outcomes study compared systemic therapy in refractory CHE, evaluating oral alitretinoin versus cyclosporine using 24-week response and drug survival.

Results

Across the included studies, HHE repeatedly exhibited psoriasis-like rather than eczematous biology. Histologically, HHE shared psoriasiform epidermal hyperplasia, a diminished or absent granular layer, and confluent parakeratosis with palmar psoriasis. Immunohistochemistry showed increased β-defensin 2 and IL-36γ expression in HHE, comparable to palmar psoriasis and higher than CHE, supporting a psoriasis-spectrum interpretation. Differentiation-marker profiling also demonstrated suprabasal extension of K5/K14 with reduced K9 and premature involucrin expression in HHE and palmar psoriasis, indicating disrupted palmoplantar differentiation and early terminal differentiation.
Using a similar IHC framework, PPP and pompholyx were distinguished despite clinically similar vesiculopustular presentations. PPP showed keratinocyte differentiation abnormalities consistent with a psoriasis-adjacent profile, including K5 extension beyond the basal layer, reduced K9 around pustules, and involucrin extension into basal layers. In contrast, pompholyx largely preserved palmoplantar differentiation, with relatively maintained K9 expression and a more physiologic involucrin pattern.
In a retrospective cohort of refractory CHE, oral alitretinoin achieved a higher 24-week responder rate than cyclosporine, while drug survival did not significantly differ. Together with the IHC findings, this underscores biological heterogeneity within CHE and suggests that hyperkeratotic phenotypes overlapping with psoriasis-like differentiation may show distinct systemic treatment responsiveness.

Conclusions
Collectively, these four studies support reframing palmoplantar inflammatory dermatoses as a continuous spectrum

from eczema-dominant disorders to psoriasis-adjacent and psoriasis-spectrum disease. HHE consistently demonstrates psoriasis-like inflammatory signaling and keratinocyte differentiation disruption, arguing against its placement as a simple subtype of CHE. PPP similarly aligns with psoriasis-adjacent differentiation abnormalities while retaining distinctive clinicopathological features, whereas pompholyx and typical CHE more closely follow an eczematous differentiation pattern. Integrating IHC signatures with real-world systemic treatment outcomes provides a biologically grounded framework that may improve diagnostic precision and guide individualized systemic treatment selection in palmoplantar disease.