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CHRONIC IRRITANT CONTACT DERMATITIS MANAGED WITH BARRIER REPAIR: A CASE REPORT ON THE ROLE OF CERAMIDE AND HYALURONIC ACID

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ABSTRACT

Introduction: *Chronic irritant contact dermatitis (ICD) is a prevalent inflammatory skin condition driven by cumulative exposure to weak irritants, leading to profound epidermal barrier dysfunction. Management strategies are increasingly focused on restoring barrier integrity as a primary therapeutic goal. This report discusses the synergistic roles of exogenous ceramides in structurally replenishing the depleted intercellular lipid matrix and of hyaluronic acid in providing potent hydration, facilitating cellular repair, and modulating the inflammatory microenvironment.*

Case Illustration: *We present the case of a 38-year-old female healthcare worker with a multi-year history of chronic hand eczema. Clinical examination revealed erythema, scaling, lichenification, and painful fissures on the dorsal and interdigital aspects of her hands, consistent with chronic ICD. After failing to respond adequately to intermittent topical corticosteroids, she was managed with a strict irritant avoidance protocol and a twice-daily application of a topical emollient containing a physiological lipid mixture of ceramides and hyaluronic acid. Significant improvement in all clinical parameters, including resolution of fissures and reduction in erythema and pruritus, was observed within four weeks of follow-up.*

Conclusion: *This case highlights the efficacy of a targeted barrier repair strategy using ceramides and hyaluronic acid as a primary, pathophysiology-directed therapy for chronic ICD, offering a valuable alternative or adjunct to conventional anti-inflammatory treatments.*

Keywords: *Chronic irritant contact dermatitis; Ceramides; Hand eczema; Hyaluronic acid; Skin barrier; Stratum corneum; Topical therapy.*

INTRODUCTION

Irritant contact dermatitis (ICD) is a non-immunologic inflammatory skin disease that constitutes up to 80% of all cases of contact dermatitis.[1] It is particularly prevalent in occupations that involve frequent and repeated exposure to water and weak irritants, a condition often termed "wet work".[2] Occupations such as healthcare, food handling, cleaning, and hairdressing are associated with a high incidence of ICD, making it a significant occupational health concern.[3] While acute ICD results from a single exposure to a potent irritant, the chronic form develops insidiously over weeks to years of cumulative exposure to multiple, often weak, irritants like soaps, detergents, and solvents.[4] The clinical presentation of chronic ICD is characterized by erythema, scaling, hyperkeratosis, lichenification, and the development of painful fissures, which can significantly impair quality of life and work productivity.[5]

It is essential to distinguish ICD from allergic contact dermatitis (ACD). ICD is a direct, non-specific cytotoxic response of epidermal cells, primarily keratinocytes, to chemical or physical insults. This damage leads to the release of pro-inflammatory cytokines and the initiation of an innate immune response without the requirement of prior immunologic sensitization.[5] In contrast, ACD is a classic delayed, Type IV T-cell mediated hypersensitivity reaction that occurs only in previously sensitized individuals upon re-exposure to a specific allergen.[6] This fundamental pathophysiological difference has critical implications for management. While the cornerstone of ACD management is the identification and strict avoidance of a specific allergen, the triggers for chronic ICD are often ubiquitous and unavoidable elements of a person's daily or occupational environment, making complete avoidance impractical.[7]

Consequently, the pathogenesis of chronic ICD is fundamentally rooted in the progressive failure of the epidermal barrier.[8] The stratum corneum (SC), the outermost layer of the epidermis, serves as the primary protective barrier. Cumulative exposure to weak irritants gradually strips the SC of its essential intercellular lipids, disrupting its highly organized lamellar structure. This disruption leads to increased transepidermal water loss (TEWL), resulting in xerosis, and allows for the enhanced penetration of external insults, perpetuating the inflammatory cycle. This barrier-centric perspective shifts the therapeutic focus from merely suppressing inflammation to actively rebuilding and maintaining the skin's structural and functional integrity.[9]

This report aims to illustrate the successful clinical application of a pathophysiology-based therapeutic strategy. By presenting a case of severe chronic ICD that responded favorably to a topical formulation designed to replenish key components of the epidermal barrier—namely ceramides and hyaluronic acid—we highlight the importance of targeted barrier repair in the management of this challenging condition.

CASE ILLUSTRATION

A 38-year-old female presented to the dermatology clinic with a three-year history of a progressively worsening rash on both hands. Her occupation as a registered nurse in a busy hospital ward required frequent handwashing with antiseptic soaps and water, as well as the regular use of alcohol-based hand sanitizers, a routine she estimated performing over 20 times per shift. Her chief complaints were persistent and severe dryness, intense pruritus, and the presence of painful cracks in the skin, which significantly impaired her manual dexterity at work and at home. This condition not only caused significant physical discomfort but also led to emotional distress and self-consciousness, affecting her social interactions.

The patient had a history of mild atopic dermatitis in childhood, primarily affecting the flexural areas, which had been quiescent for over two decades. She reported no known contact allergies and had no personal or family history of psoriasis. Previous treatments for her hand dermatitis included various over-the-counter emollients, which provided only transient and minimal relief. She had also been prescribed a mid-potency topical corticosteroid (triamcinolone acetonide 0.1% ointment) by her primary care physician for use during flares. While this provided a temporary reduction in erythema and pruritus, the symptoms would rapidly relapse upon discontinuation of the steroid, a common cycle in chronic barrier-impaired conditions.

On physical examination, both hands exhibited diffuse, ill-defined erythematous plaques with overlying fine, silvery scaling, particularly prominent on the dorsal surfaces and extending into the interdigital web spaces.[10] The skin over the dorsal aspects of the proximal and distal interphalangeal joints was markedly lichenified, appearing thickened and leathery with accentuated skin markings, a classic sign of chronic rubbing and scratching.[11] Multiple painful, linear fissures, some with hemorrhagic crusting, were noted over the knuckles of the second, third, and fourth digits of her dominant right hand. The left thumb displayed a more acute-on-chronic picture, with scattered, tense vesicles on an erythematous and edematous base, alongside areas of post-inflammatory hyperpigmentation. The palmar surfaces were less inflamed but showed profound xerosis with a parched, glazed appearance and hyperlinearity (Figure 1).

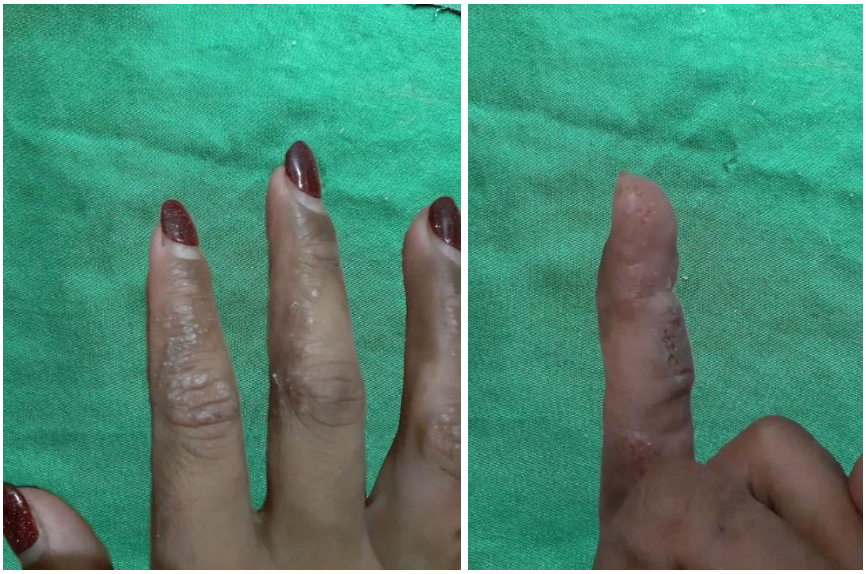

Figure 1. Clinical examination at the Patient's Initial Visit to the Polyclinic

ICD is a clinical diagnosis. Therefore, diagnosis of ICD can be established based on the clinical morphology, the chronic and relapsing nature of the condition, and a clear occupational history of cumulative exposure to known weak irritants.[12] The patient's history of atopy was noted as a significant predisposing factor, as individuals with an atopic diathesis often have a constitutionally impaired skin barrier, rendering them more susceptible to the damaging effects of irritants.

The patient received extensive counseling on a multi-faceted irritant avoidance strategy, including the 'soak and seal' method after handwashing, using lukewarm water followed by immediate application of the prescribed emollient to damp skin.[13] She was advised to switch to a gentle, soap-free, pH-balanced cleanser and to meticulously pat her hands dry with a soft towel, avoiding any rubbing friction.[14] For all occupational and domestic wet work, she was instructed to use a double-gloving technique: soft cotton liners under non-latex, waterproof vinyl gloves to absorb sweat and prevent direct contact with potential rubber allergens. The patient was instructed to discontinue the use of the topical corticosteroid to avoid potential side effects and the cycle of relapse, and was instead prescribed a specific barrier repair emollient containing a combination of ceramides and hyaluronic acid, to be applied liberally to both hands twice daily (morning and evening) and additionally after each instance of handwashing throughout the day.

The patient was reassessed at a four-week follow-up visit. She reported a significant reduction in pruritus and pain within the first two weeks of initiating the new regimen. She noted that the skin felt more comfortable and less "tight." By the four-week mark, she reported a near-complete resolution of her symptoms.

Clinical examination at the follow-up appointment revealed a remarkable transformation, as depicted in Figures 2A and 2B. The diffuse erythema and scaling had completely resolved, replaced by smooth skin with a healthy, supple texture. The previously painful and debilitating fissures had fully re-epithelialized without scarring. The lichenification over the knuckles was significantly reduced, and the skin's natural turgor and hydration appeared fully restored. The patient expressed high satisfaction with both the cosmetic outcome and the functional recovery, reporting a profound improvement in her quality of life. She was advised to continue the irritant avoidance measures and the regular use of the ceramide and hyaluronic acid-based emollient as a long-term maintenance strategy to prevent recurrence.

Figure 2. Clinical examination at the follow-up appointment.
A) Before receiving any therapy. B) After therapy, dorsal aspects of both hands showing complete resolution of diffuse erythema and scaling, with smooth skin.

DISCUSSION

This case highlights how a targeted, pathophysiology-based barrier repair strategy can offer an effective alternative to conventional, inflammation-centered management in severe, occupationally induced chronic ICD. The discussion will explore the underlying mechanisms of chronic ICD and the molecular rationale for using a combination of ceramides and hyaluronic acid.

Unlike acute ICD, which is caused by strong, overtly corrosive agents, chronic ICD is an insidious process initiated by repeated, cumulative exposure to weak, sub-toxic irritants.[15] The pathogenesis involves three main interconnected events: skin barrier disruption, epidermal cellular changes, and cytokine release.[16]

The process begins with the direct cytotoxic action of irritants on keratinocytes and the emulsification of intercellular lipids by surfactants found in soaps and detergents.[17] This 'degreasing' effect disrupts the highly organized lamellar structure of the stratum corneum, leading to a quantifiable increase in TEWL.[18] The compromised barrier then permits deeper penetration of irritants, which triggers damaged keratinocytes to release a cascade of pre-formed pro-inflammatory cytokines, primarily interleukin-1 alpha (IL−1α) and tumor necrosis factor-alpha (TNF−α).[19] This initiates an innate, non-allergic inflammatory response, recruiting neutrophils and lymphocytes and perpetuating a state of chronic inflammation that drives the clinical changes of erythema, hyperkeratosis, and lichenification.[20]

The patient's history of atopic dermatitis is a critical predisposing factor. Individuals with an atopic diathesis, particularly those with loss-of-function mutations in the filaggrin (FLG) gene, have an inherently defective epidermal barrier.[21] Filaggrin is a pivotal structural protein essential for the terminal differentiation of keratinocytes, the formation of the cornified envelope, and the generation of natural moisturizing factor (NMF), a collection of hygroscopic small molecules that hydrate the SC.[22] Its deficiency results in a disorganized, 'leaky' SC with reduced baseline hydration, creating a state of profound susceptibility to the damaging effects of external irritants.[23]

This process creates a self-perpetuating vicious cycle. The initial barrier breach caused by irritants leads to inflammation. The resulting inflammatory mediators can, in turn, further impair epidermal differentiation and downregulate the expression of key enzymes involved in the synthesis of barrier lipids, such as ceramides.[24] Therefore, the inflammation actively suppresses the skin's own ability to repair the very barrier whose disruption caused the inflammation in the first place. This explains the clinical course observed in this patient: topical corticosteroids could temporarily suppress the inflammation, but because they did not address the underlying structural defect, the barrier remained compromised, and the condition relapsed as soon as the anti-inflammatory pressure was removed. A successful long-term therapy must interrupt this cycle by addressing the primary barrier defect.

The molecular rationale for barrier repair therapy was to directly counteract the core pathophysiological defects of chronic ICD. This was achieved by providing the molecular building blocks necessary to restore barrier structure and function.

The stratum corneum is often described by the "brick and mortar" model, where the terminally differentiated corneocytes are the "bricks" and the intercellular lipid matrix serves as the "mortar".[25] This lipid mortar is paramount for the skin's barrier function. It is composed of an approximately equimolar mixture of ceramides, cholesterol, and free fatty acids, with ceramides being the most abundant and structurally critical component, constituting about 50% of the lipid mass by weight.[26] Ceramides are essential for forming the highly ordered, tightly packed lamellar structures that are largely impermeable to water, preventing excessive TEWL and blocking the entry of foreign substances.[27]

The intercellular lipid matrix is composed of an approximately equimolar mixture of ceramides (around 50% by weight), cholesterol, and free fatty acids.[28] These lipids are organized into highly ordered lamellar bilayers with a unique long periodicity phase (LPP) of ~13 nm, which is considered essential for barrier function.[29] Ceramides, with their long-chain fatty acid tails, are the primary architects of this structure.[30] In inflammatory skin diseases like atopic dermatitis and ICD, there is a marked reduction in total ceramide content, particularly long-chain ceramides like Ceramide 1 (Cer), and an alteration in the ceramide profile.[31] This deficiency leads to a disorganized, less dense lipid structure (a shift from orthorhombic to hexagonal packing), which directly correlates with increased TEWL and barrier incompetence.[32] The therapeutic principle of using ceramide-containing emollients is, therefore, the direct replenishment of these crucial structural lipids. When applied topically in a properly formulated vehicle, exogenous ceramides can integrate into the SC, help reorganize the disordered lipid lamellae, decrease TEWL, and restore the physical integrity of the barrier.[33]

Hyaluronic acid (HA) is a large, naturally occurring glycosaminoglycan (GAG) and a major component of the dermal and epidermal extracellular matrix (ECM).[34] Its primary and most well-known function is its remarkable capacity to bind and retain water molecules—it can hold up to 1000 times its own weight in water.[35] This makes it a powerful humectant, drawing moisture into the epidermis and maintaining skin hydration, turgor, and elasticity. In the context of chronic ICD, where elevated TEWL leads to severe xerosis and fissuring, this potent hydrating property is of paramount importance.

Beyond its role as a passive humectant, HA is a dynamic biological modulator. Its synthesis is carried out by three distinct membrane-bound enzymes (HAS-1, -2, -3), which produce HA chains of varying lengths, while its degradation is managed by a family of enzymes called hyaluronidases (HYALs).[36] This constant turnover is crucial, as the biological activity of

HA is highly dependent on its molecular weight. High-molecular-weight HA (>1000 kDa), found in healthy tissue, is generally anti-inflammatory, immunosuppressive, and anti-angiogenic. In contrast, low-molecular-weight fragments, often generated during tissue injury, can act as damage signals, promoting inflammation and angiogenesis, which are critical components of the wound healing cascade.[1] Therefore, the application of topical HA not only combats the dryness of ICD but also actively supports the healing of damaged skin and may help to downregulate the underlying inflammatory process.

The combination of ceramides and hyaluronic acid in a single formulation offers a synergistic, dual-pronged approach to managing chronic ICD. This strategy simultaneously addresses both the structural and functional deficits of the compromised epidermal barrier. Ceramides address the *structural deficit* by physically rebuilding the lipid barrier—in effect, "repairing the mortar between the bricks." Hyaluronic acid addresses the *functional deficit* by intensely hydrating the tissue and creating a pro-healing, anti-inflammatory microenvironment—"providing the water and raw materials for the repair crew to work effectively."

This dual-action approach was particularly relevant for the patient in this case, who presented with both signs of a severely compromised lipid barrier (scaling, xerosis) and signs of active tissue damage and inflammation (fissures, erythema). A clinical study directly comparing a hyaluronic acid-based foam to a ceramide-containing cream for atopic dermatitis found that while both were effective, the HA formulation achieved a statistically significant improvement in overall eczema severity earlier in the treatment course (by week 2). Furthermore, patient preference statistically and significantly favored the HA foam for its cosmetic attributes, including ease of spread, moisturization, and lack of odor, as well as for its perceived effectiveness and soothing ability. This suggests that the active biological properties of HA may accelerate clinical improvement beyond what is achieved by lipid replenishment alone, highlighting the benefit of a combined approach.

In barrier repair therapy for ICD, each component has a specific site of action and mechanism that contributes to its therapeutic benefit. Ceramides primarily act within the intercellular space of the stratum corneum as structural lipids that organize the intercellular lipid lamellae and form the primary permeability barrier. Through this role, ceramides restore barrier integrity, reduce TEWL, and prevent penetration of irritants. HA, on the other hand, acts mainly within the extracellular matrix of the epidermis and dermis as a potent humectant that binds water, provides a hydrated matrix for cellular processes, and modulates inflammation. These mechanisms allow HA to improve skin hydration and elasticity, create a moist environment that supports fissure healing, and soothe irritation.

The management of chronic hand eczema (CHE), a broad category that includes chronic ICD, typically follows a stepwise approach. The foundational step, and arguably the most important for long-term success, is patient education on irritant avoidance and the consistent, liberal use of emollients. First-line pharmacotherapy for inflammatory flares involves the use of topical corticosteroids, with potency tailored to disease severity. However, as demonstrated in this case, long-term or frequent use is limited by potential side effects, such as skin atrophy and telangiectasias, and the common phenomenon of rebound flares upon cessation.

For moderate-to-severe flares unresponsive to emollients, potent or super-potent topical corticosteroids (e.g., clobetasol propionate 0.05%) are first-line therapy, typically used for short courses of 2-4 weeks to regain control.[17] Topical calcineurin inhibitors (tacrolimus, pimecrolimus) are valuable steroid-sparing second-line agents, particularly for maintenance therapy in sensitive areas like the face or intertriginous zones.[12] For severe, refractory CHE, options escalate to phototherapy (PUVA or narrowband UVB) or systemic therapies.[5] Alitretinoin, an oral retinoid, is approved in Europe and Canada specifically for severe CHE unresponsive to potent topical steroids and has shown significant efficacy.[2] Other systemic options, often used off-label, include traditional immunosuppressants like methotrexate and cyclosporine, or biologics such as dupilumab, which targets the Type 2 inflammatory pathway.[5]

This case makes a strong argument for positioning advanced, pathophysiology-directed barrier repair emollients—those containing active ingredients like ceramides and hyaluronic acid—not merely as adjunctive "moisturizers," but as a distinct therapeutic class. They can be used as a primary monotherapy in mild-to-moderate cases, as a highly effective steroid-sparing maintenance strategy to prolong remission, or as an essential adjunct to pharmacotherapy in severe cases to help break the cycle of barrier damage and inflammation. By directly addressing the root cause of the disease, this approach can reduce the need for potent anti-inflammatory agents and improve long-term outcomes.

CONCLUSION

This case report demonstrates the successful rapid and sustained clinical resolution of severe, occupationally-induced chronic ICD by shifting the therapeutic focus from intermittent inflammation suppression to continuous, pathophysiology-directed barrier repair using a targeted therapy combining ceramides and hyaluronic acid. The discussion reinforces the understanding that chronic ICD is a disease driven by a self-perpetuating cycle of epidermal barrier disruption and subsequent non-allergic inflammation. A therapeutic approach that simultaneously addresses the structural lipid deficit with ceramides and the functional hydration and healing deficit with hyaluronic acid represents a rational, effective, and well-tolerated strategy. Such pathophysiology-directed emollients should be considered a cornerstone in the management of chronic ICD, serving to reduce reliance on topical corticosteroids, prevent disease flares, and ultimately improve long-term disease control and patient quality of life.

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