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Role of hydrogel-based systems in the management of lamellar ichthyosis: A comprehensive review

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Abstract

A rare autosomal recessive congenital skin condition called lamellar ichthyosis is typified by aberrant keratinization and compromised epidermal barrier function. Mutations in genes including TGM1, ABCA12, ALOX12B, ALOXE3, and NIPAL4, which are essential for protein cross-linking and lipid metabolism in the stratum corneum, are the main cause of the disorder. Clinically, affected people frequently have a collodion membrane at birth that eventually sheds, exposing the body's massive, black, plate-like scales. Ectropion, eclabium, hypohidrosis, and heightened vulnerability to infections as a result of weakened skin integrity are other characteristics. According to epidemiology, lamellar ichthyosis is an uncommon condition that affects both sexes equally and has a global prevalence of between 1 in 100,000 and 300,000 people. Genetic testing may be used to assist the diagnosis, which is mostly clinical. In extreme cases, emollients, humectants, keratolytic agents, and systemic retinoids are used to relieve symptoms. The potential use of hydrogels as a successful therapeutic strategy for treating this illness has been brought to light by recent developments. Because of their high water content and biocompatibility, hydrogels improve medication penetration into the stratum corneum, decrease scaling, and offer constant hydration. Treatment results are further enhanced by their capacity to provide sustained and regulated drug release. All things considered, lamellar ichthyosis necessitates lifelong care, and new treatments like hydrogel-based formulations offer encouraging opportunities to enhance patient care and quality of life.

Keywords: *Lamellar ichthyosis*; Mutations; Emollients; Humectants; Keratolytic agents

1. Introduction

Lamellar ichthyosis (LI) is a rare autosomal recessive genetic condition that falls within the larger category of ichthyosis, which is characterized by excessive skin scaling and aberrant epidermal differentiation. When doctors started recording individuals with unusually dry, scaly skin in the 18th and 19th centuries, ichthyosis-like conditions were first described.[1] The primary cause of this congenital disorder, which is present from birth, is abnormalities in the genes that generate and maintain the skin barrier, most notably the TGM1 (transglutaminase-1) gene. This enzyme is crucial for the cross-linking of proteins that forms the cornified cell envelope, which is a vital part of the stratum corneum, the skin's outermost layer. Increased transepidermal water loss, aberrant keratinization, and compromised barrier function are the results of defects in this process.

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Table 1 Classification, pathophysiology and Management of LI

Type of Ichthyosis	Pathophysiology	Management	Drug of Choice	Ref.
Ichthyosis vulgaris	Defect in filaggrin leading to impaired skin barrier and abnormal keratinization	Regular emollients, keratolytics, mild topical steroids if needed	Urea, Lactic acid	[2-5]
X-linked ichthyosis	Steroid sulfatase deficiency causing accumulation of cholesterol sulfate and scaling	Moisturizers, keratolytic agents	Urea, Salicylic acid	
Lamellar Ichthyosis	Mutation in genes such as TGM1 causing defective cornified envelope formation and severe hyperkeratosis	Hydration therapy, keratolytic agents, topical retinoids, systemic retinoids in severe cases	Acitretin	
Congenital ichthyosiform erythroderma	Defective epidermal lipid metabolism leading to erythema and scaling	Emollients, keratolytics, retinoids	Acitretin	
Harlequin ichthyosis	Mutation in ABCA12 gene causing abnormal lipid transport in epidermis	Intensive neonatal care, systemic retinoids, supportive therapy	Acitretin	

Clinically, a tight, glossy, translucent membrane called a collodion membrane is often present at birth in newborns with lamellar ichthyosis. During the first several weeks of birth, this membrane usually sheds, exposing the disorder's distinctive phenotype. The majority of the skin's surface is then covered with massive, thick, plate-like scales that are frequently dark brown in color. The disorder gets its name from the lamellar (layered) look of the skin caused by these scales, which are typically adhesive and arranged in a mosaic-like pattern. Lamellar ichthyosis typically shows little erythema (skin redness), however minor inflammation may occasionally be present, in contrast to some other types of ichthyosis, such as ichthyosis vulgaris. [6]

From a pathophysiological standpoint, lamellar ichthyosis is linked to abnormalities in the epidermis's protein cross-linking and lipid metabolism. Chronic dryness and scaling result from the skin's permeability barrier being compromised by the breakdown of lipid lamellae in the stratum corneum. Apart from TGM1, other genes including ABCA12, NIPAL4, ALOX12B, and ALOXE3 have also been linked to the illness, indicating its genetic variability. [7]

The primary method of diagnosis is clinical, with the identification of causal mutations through genetic testing supporting the diagnosis. Hyperkeratosis, or thickening of the stratum corneum, is usually seen on histopathological examination in the absence of severe epidermal inflammation. Families with known mutations may be eligible for prenatal diagnostics.

Lamellar ichthyosis is mostly treated symptomatically, with an emphasis on increasing skin moisture and minimizing scaling. This includes using emollients on a regular basis, using keratolytic agents like lactic acid or urea, and using systemic retinoids in more serious situations. In order to avoid consequences like infections, fissuring, and reduced thermoregulation, early and regular therapy is crucial. In order to maximize patient results, lamellar ichthyosis is a chronic dermatological disorder that necessitates multidisciplinary management and lifetime care. [8]

2. Disease profile

The word "ichthyosis" refers to a broad category of keratinizing conditions with various causes, but they are all characterized by dry, scaly skin and more or less widespread epidermal hyperkeratosis. The severity of the skin symptoms can vary from mild xerosis and scaling on the extremities, which appear primarily in the winter months (as in ichthyosis vulgaris), to massive hyperkeratosis and scaling throughout the body (as in lamellar ichthyosis), depending on the type of ichthyosis and the variable influences of individual and environmental factors.

A genetic autosomal recessive skin condition called lamellar ichthyosis manifests at birth as a collodion membrane covering the entire baby's body. One of the three hereditary skin conditions known as Autosomal Recessive Congenital Ichthyosis (ARCI) is *lamellar ichthyosis.* [9,10]

2.1. Epidemiology

From the 18th century to the present, the epidemiology of Lamellar ichthyosis shows a shift from sparse descriptive findings to accurate genetic and population-based data.

There was very little knowledge about epidemiology throughout the 18th and early 19th centuries. In the medical literature, ichthyosis cases were infrequently reported and sometimes jumbled together without a clear differentiation between subgroups. Because the diagnosis was based only on obvious skin abnormalities and many instances were either misclassified or unreported, there were no accurate estimates of prevalence. Dermatology started to emerge as a specialized field in the late 19th and early 20th centuries. Based on clinical appearance, doctors began differentiating between several types of ichthyosis. However, case reports and tiny clinical series continued to provide the majority of the anecdotal epidemiological data. [11]

Lamellar ichthyosis was identified as a member of the group known as autosomal recessive congenital ichthyoses (ARCI) around the middle to late 20th century due to improved clinical classification. Studies on epidemiology started to appear, especially in North America and Europe. The prevalence of the condition was estimated to be between 1 in 100,000 and 300,000 people. Males and females were distributed equally, which is consistent with its autosomal. It was found to be equally distributed among males and females, which is consistent with autosomal recessive inheritance. [12,13] Molecular genetics developments have increased epidemiological accuracy in the late 20th and early 21st centuries. Genotype-based diagnosis was made possible by the discovery of mutations in genes like TGM1, ABCA12, and others, which decreased misclassification. Prevalence estimates were further improved by databases of rare diseases and population-based registries. Regional differences became apparent, with communities with genetic isolation or consanguineous marriages reporting somewhat greater frequency.

Lamellar ichthyosis is currently acknowledged as a rare, worldwide illness with comparatively stable frequency estimates across various groups. Genetic screening, better neonatal diagnosis (collodion newborn presentation), and international cooperation in tracking rare diseases are all beneficial to modern epidemiology. [14]

Everything considered, the epidemiology of lamellar ichthyosis has progressed from qualitative historical descriptions to quantitative genetic epidemiology, offering a better grasp of its worldwide distribution, rarity, and risk factors.

2.2. Signs and symptoms:

2.2.1. At Birth (Neonatal Features)

- Presence of collodion membrane (tight, shiny, transparent covering over the skin)
- Membrane sheds within 2–4 weeks after birth

2.2.2. Skin Changes

- Large, thick, plate-like scales (brown or dark-colored)
- Scales are firmly attached and cover the whole body
- Skin appears dry (xerosis) and rough
- Minimal or no redness (erythema)

2.2.3. Facial Abnormalities

- Ectropion – outward turning of eyelids
- Eclabium – outward turning of lips
- Tight skin causing restricted facial movements [15]

2.2.4. Sweating and Temperature Issues

- Hypohidrosis (reduced sweating)
- Leads to heat intolerance and risk of overheating

2.2.5. Skin Integrity Problems

- Cracks and fissures in skin
- Increased risk of secondary infections [16,17]

2.2.6. Hair and Nail Changes

- Sparse or coarse hair
- Thickened or dystrophic nails (in some cases)

2.2.7. Palms and Soles

- Palmar and plantar hyperkeratosis (thickening of palms and soles)
- May cause discomfort while walking or using hands

2.2.8. General Condition

- Usually normal intelligence and growth
- Chronic condition requiring lifelong management [18]

3. Mechanism

3.1. Cornified Envelope Formation Pathway

Transglutaminase enzymes (TGM1, TGM3) cross-link proteins such involucrin, loricrin, and short proline-rich proteins (SPRs) in the cornified envelope production pathway. These proteins are stabilized by calcium-dependent dimerization, which creates a stiff structure beneath the cell membrane. In the stratum corneum, this envelope offers barrier protection and mechanical strength.

3.2. Lipid Envelope Formation Pathway

The creation of a lipid-rich barrier that is crucial for stopping water loss is the main goal of this pathway. Endoplasmic reticulum-synthesised lipids are transported and transformed into fatty acids and ceramides. The hydrophobic barrier of the skin is formed by these lipids, which are produced by lamellar bodies and arranged into extracellular layers. [19]

3.3. Sphingolipid Metabolism Pathway

Serine and palmitoyl-CoA are the starting points of sphingolipid metabolism. Serine palmitoyltransferase (SPT) then forms sphinganine. Ceramides are produced by subsequent enzymatic events and are essential for preserving the integrity of the epidermal barrier. Defects in this process, which is regulated by enzymes including CERS3 and KDSR, result in aberrant lipid composition and compromised epidermal function.

3.4. Lamellar Body Secretion Pathway

Specialized organelles called lamellar bodies carry lipids and enzymes to the stratum corneum. Lipid loading into these vesicles is facilitated by proteins such as ABCA12. Their contents create extracellular lipid layers upon secretion, which are crucial for barrier function. Severe scaling and poor lipid delivery are the outcomes of dysfunction. [20]

3.5. Lipoxygenase Pathway (ALOX12B–ALOXE3 Pathway)

The enzymes ALOX12B and ALOXE3, which oxidize fatty acids to produce lipid mediators required for epidermal development, are involved in this process. These altered lipids support the healthy development of the skin barrier. Mutations cause aberrant lipid buildup and improper desquamation in lamellar ichthyosis by interfering with lipid processing.

3.6. Filaggrin Metabolism Pathway

The first step in the metabolism of filaggrin is the storage of filaggrin in keratohyalin granules. It is broken down into filaggrin monomers by proteolytic enzymes such furin and caspases. They reinforce the skin by aggregating keratin filaments. Natural moisturizing factors (NMF), which are necessary for hydration and preserving skin suppleness, are produced by further breakdown. [21]

3.7. Keratin–Filaggrin Interaction Pathway

Filaggrin binds to keratin intermediate filaments (KRT1/2/10) in this pathway, causing them to aggregate into dense bundles. Corneocytes are strengthened and structural integrity is supported by this process. Increased fragility and scaling in keratinization diseases are caused by disruption, which also weakens the architecture of the skin. [22]

3.8. Natural Moisturizing Factor (NMF) Formation Pathway

The breakdown of filaggrin yields amino acids like histidine, which are then transformed into substances like pyrrolidone carboxylic acid (PCA) and urocanic acid (UCA). These molecules combine to generate NMF, which controls pH, preserves hydration, and guards against environmental stress. Dry, scaly skin is the result of deficiency.[23]

3.9. Cell Junction and Adhesion Pathway

Tight junctions (claudins, occludins), desmosomes (DSG, DSC), and corneodesmosomes are involved in this system, which keeps keratinocytes cohesive. These structures guarantee controlled desquamation and the integrity of the epidermis. Skin fragility and aberrant scaling result from mutations in adhesion proteins that decrease intercellular connections.

3.10. Keratinocyte Differentiation Pathway

Keratinocytes express particular keratins (KRT5/14 to KRT1/10) as they differentiate from the basal layer to the stratum corneum. Lipid buildup, structural protein production, and programmed cell death are all part of this process. Barrier creation is ensured by proper differentiation, whereas disruption leads to hyperkeratosis and impaired skin function. [24,25]

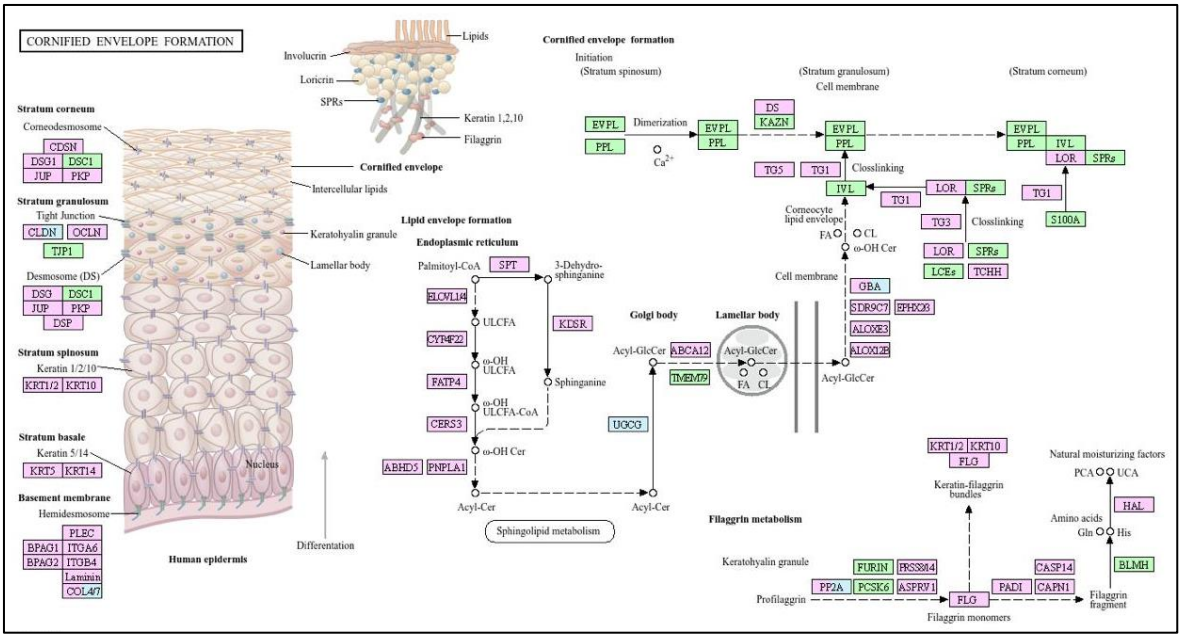

Figure 1 Overall mechanism of Lamellar ichthyosis

4. Causes

4.1. Gene Mutations In Lamellar Ichthyosis

Because they directly change the structure, stability, and function of proteins necessary for the proper development of the skin barrier, protein mutations play a key role in the disease process of lamellar ichthyosis. These mutations result from variations in the DNA sequence of particular genes, which impact the synthesis, folding, and functionality of proteins within epidermalcells.

A gene mutation modifies a protein's amino acid sequence at the molecular level. Even a single amino acid alteration can interfere with normal functioning since protein structure dictates function. For instance, mutations in the TGM1

gene affect transglutaminase-1, an enzyme that typically catalyzes the cross-linking of structural proteins like involucrin, loricrin, and small proline-rich proteins. These proteins make up the stratum corneum's protective layer, the cornified cell envelope. [26]

When mutations occur

- The enzyme may misfold, losing its correct three-dimensional shape.
- It may become unstable and degrade quickly inside the cell.
- Its active site may be altered, preventing it from binding substrates or catalyzing reactions.
- A weak and poorly constructed epidermal barrier results from improper protein cross-linking.

Various kinds of protein mutations have distinct effects: Missense mutations can interfere with folding or function by substituting one amino acid for another. A truncated, nonfunctional protein is produced when nonsense mutations produce an early stop codon. The downstream amino acid sequence is significantly changed by frameshift mutations (caused by insertions or deletions), which frequently result in total loss of function. Splice-site mutations cause mRNA to be processed improperly, resulting in aberrant proteins. Splice-site mutations cause mRNA to be processed improperly, resulting in aberrant proteins. Mutations impact protein processing and localization in addition to structural flaws. For example, mutations in ALOX12B and ALOXE3 affect enzymes that modify lipids necessary for skin maturation, whereas the mutant protein in ABCA12 is unable to transport lipids into lamellar granules. These modified proteins are unable to effectively carry out their biological functions. [27]

4.1.1. ABCA12 gene (ATP-binding cassette transporter A12)

A lipid transporter protein that is necessary for transferring lipids, particularly ceramides, into specialized organelles known as lamellar granules in keratinocytes is encoded by the ABCA12 gene. A protective lipid barrier is created when these lipids are secreted into the stratum corneum's extracellular area. Mutation effect: Defective ABCA12 protein prevents proper lipid transport. Result: Thick scaling, excessive water loss, and a seriously damaged barrier result from lipids failing to reach the skin's surface. More serious disorders like harlequin ichthyosis are also linked to severe mutations in ABCA12.

4.1.2. ALOX12B gene (Arachidonate 12-lipoxygenase, 12R type)

A lipoxygenase enzyme that is involved in the oxidation of fatty acids—a critical stage in the processing of epidermal lipids—is encoded by the ALOX12B gene. Mutation effect: Lipid oxidation pathways are disrupted when enzyme function is lost. Result: Inadequate lipid modification causes aberrant shedding (desquamation) and poor skin barrier development, which leads to scaling. [28]

4.1.3. ALOXE3 gene (Arachidonate lipoxygenase 3)

Another lipoxygenase enzyme participating in the same metabolic pathway is encoded by the ALOXE3 gene, which collaborates closely with ALOX12B. Impaired conversion of lipid intermediates necessary for proper epidermal development is the result of mutations. Result: Contributes to dry, scaly skin by causing aberrant lipid accumulation and poor cornified layer production.

4.1.4. NIPAL4 gene (NIPA-like domain containing 4 / ichthyin)

A protein that is thought to be involved in lipid metabolism and magnesium transport in the epidermis is encoded by the NIPAL4 gene. During keratinocyte differentiation, it contributes to the preservation of cellular homeostasis. Ion balance and lipid processing in skin cells are disrupted by the mutation effect. Impaired skin barrier function and aberrant keratinization are the results. Overall, aberrant keratinization, excessive scaling, and a damaged skin barrier are caused by protein mutations in lamellar ichthyosis, which result in loss of enzymatic activity, faulty structural assembly, and altered lipid-protein interactions.

4.2. Autosomal recessive inheritance:

A hereditary pattern where a problem only manifests when a person receives two mutant copies of a gene—one from each parent. The genes in question are found on autosomes, or non-sex chromosomes, and loss-of-function mutations that reduce or eliminate the ability to produce a functioning protein are the main source of this inheritance pattern. These mutations can be missense, nonsense, frameshift, or splice-site changes, all of which have distinct effects on the synthesis or activity of proteins. Since one normal allele is usually enough to sustain normal physiological function, people with only one mutant allele are usually asymptomatic carriers.

A child born to two carrier parents has a 50% chance of becoming a carrier, a 25% chance of inheriting two normal alleles, and a 25% chance of getting both faulty alleles and expressing the disease. The total or almost total loss of functional protein, which interferes with regular cellular or biochemical processes, is what causes sickness to emerge. Additionally, because related people are more likely to have the same harmful mutation, consanguinity greatly raises the risk of autosomal recessive illnesses. Certain recessive disorders may also be more common in particular populations due to population-specific genetic variables such founder effects and genetic drift. Lamellar ichthyosis is one of the genetic disorders that exhibit this method of inheritance; for the ailment to manifest clinically, mutations must be acquired from both parents. [29]

5. Treatment plan

Table 2 Treatment plan of LI

S.NO	Treatment Category	Drug / Therapy	Purpose	Ref.
1	Emollients	Petrolatum, Paraffin, Lanolin	Hydrate the skin and restore the skin barrier	[30-36]
2	Humectants	Urea, Glycerol	Increase water retention and soften the stratum corneum	
3	Keratolytic agents	Salicylic acid	Remove thick scales and reduce hyperkeratosis	
4	Keratolytic agents	Lactic acid, Glycolic acid	Promote desquamation and improve skin texture	
5	Keratolytic agents	Propylene glycol (10–25%)	Hydrate and soften keratin, aiding scale removal	
6	Thiol-based keratolytic	N-Acetylcysteine (NAC)	Break disulfide bonds in keratin and reduce scaling	
7	Topical retinoids	Tazarotene, Tretinoin, Adapalene	Normalize keratinocyte differentiation	
8	Vitamin D analogues	Calcipotriol	Regulate keratinocyte proliferation and differentiation	
9	Systemic retinoids	Acitretin, Isotretinoin	Reduce severe hyperkeratosis and scaling	
10	Antimicrobial therapy	Topical or oral antibiotics	Treat secondary skin infections	
11	Supportive therapy	Salt bath, oil bath	Hydrate skin and help loosen scales	
12	Eye care	Lubricating eye drops	Prevent dryness due to ectropion	
13	Advanced therapy	Gene therapy	Correct underlying genetic mutation	

5.1. Marketed product for lamellar ichthyosis:

Table 3 Marketed product for LI

S.No	Type of Formulation	Drug / Active Ingredient	Example (Marketed Product)	Purpose	Ref.
1	Cream	Ammonium lactate / Lactic acid	AmLactin cream	Moisturizes skin and reduces scaling	[37-42]
2	Cream / Lotion	Urea (10–40%)	Carmol, Keralac	Hydrates skin and removes excess keratin	

3	Ointment	Petrolatum / Mineral oil	White petrolatum ointment	Skin barrier protection and hydration	
4	Gel	Tazarotene 0.05–0.1%	Tazorac gel	Reduces hyperkeratosis and scaling	
5	Cream / Gel	Tretinoin	Retin-A	Regulates keratinocyte differentiation	
6	Cream	Calcipotriene (Vitamin D analogue)	Dovonex cream	Controls keratinocyte proliferation	
7	Capsule (Oral)	Acitretin	Soriatane	Used for severe lamellar ichthyosis	
8	Capsule (Oral)	Isotretinoin	Accutane / Claravis	Reduces excessive keratinization	
9	Topical emulsion	N-Acetylcysteine 10%	Compounded NAC emulsion	Breaks disulfide bonds in keratin and reduces scaling	
10	Cream	Trifarotene 0.005%	Aklief cream	Retinoid used for congenital ichthyosis therapy	
11	Lotion / Cream	Salicylic acid	Salex lotion	Keratolytic action to remove scales	
12	Combination cream	Lactic acid + Propylene glycol	Locobase / Diprobase formulations	Improves hydration and reduces scaling	

5.2. Hydrogel for the management of lamellar ichthyosis:

Table 4 Characteristics of hydrogel

S. No	Characteristic	Description	Ref.
1	High water content	Hydrogels can absorb and retain large amounts of water (70–90%), which helps maintain a moist environment.	[43-46]
2	Three-dimensional polymer network	Hydrogels consist of cross-linked hydrophilic polymer chains that form a stable 3-D structure.	
3	Biocompatibility	Hydrogels are generally non-toxic and non-irritant, making them suitable for biomedical and topical applications.	
4	Swelling ability	They can swell in aqueous media without dissolving due to cross-linking.	
5	Controlled drug release	Hydrogels can release drugs in a sustained or controlled manner.	
6	Soft and flexible nature	Their structure resembles biological tissues, giving them good flexibility and comfort when applied to skin.	
7	Permeability	Hydrogels allow diffusion of oxygen, nutrients, and drugs through the polymer network.	
8	Stimuli responsiveness	Some hydrogels respond to temperature, pH, light, or ionic strength.	
9	Good adhesion	Hydrogels can adhere well to biological tissues, improving drug delivery efficiency.	

6. Jutification for using hydrogel in lamellar ichthyosis

Hydrogel's many therapeutic benefits for treating dry, scaly skin make its usage in lamellar ichthyosis well warranted. Because of their high water content (about 70–90%), hydrogels give the skin considerable hydration and help combat the extreme dryness linked to the condition. Their capacity to hold onto and progressively release moisture guarantees ongoing skin hydration, which lessens scaling and enhances skin texture. Furthermore, by allowing for the regulated and prolonged release of topical drugs, hydrogels function as efficient drug delivery systems that improve overall therapeutic efficacy. Additionally, the hydrated polymer network enhances medication penetration through the stratum corneum, improving therapeutic agent absorption. [47]

Hydrogels are also ideal for long-term usage in sensitive skin disorders because they are non-toxic, biocompatible, and non-irritating. Additionally, they provide a calming and cooling impact that lessens inflammation and discomfort. Hydrogels are a great choice for topical treatment of lamellar ichthyosis because of their simplicity of administration and capacity to create a protective layer over the skin, which further enhances patient comfort and compliance.

Table 5 Marketed hydrogel for LI

S.No	Marketed Hydrogel Brand	Manufacturer	Components / Composition	Medical Use	Ref.
1	Intrasite Gel	Smith & Nephew Healthcare	Modified carboxymethylcellulose polymer, propylene glycol, purified water	Autolytic debridement, pressure ulcers, burns, diabetic foot ulcers	[48-50]
2	Purilon Gel	Coloplast Corp	Sodium carboxymethylcellulose, calcium alginate, purified water	Necrotic wounds, leg ulcers, diabetic ulcers, burns	
3	SoloSite Gel	Smith & Nephew Healthcare	Sodium carboxymethylcellulose, glycerol, water	Minor burns, superficial wounds, abrasions, skin tears	
4	NU-GEL Hydrogel	Systagenix	Sodium alginate hydrogel with purified water	Chronic wounds, pressure ulcers, moist wound healing	
5	Suprasorb G Hydrogel	Lohmann & Rauscher	Acrylic polymer derivatives, polyethylene glycol, phenoxyethanol	Dry wounds, burns, pressure ulcers	
6	AquaDerm Hydrogel	DermaRite	Acrylic polymer derivatives, propylene glycol, purified water	Ulcers, minor burns, wound healing	
7	Restore Hydrogel Dressing	Hollister Inc.	Hyaluronic acid hydrogel base, purified water	Chronic wounds, tissue regeneration	
8	Prontosan Wound Gel	B. Braun Medical	Hydroxyethyl cellulose, glycerol, betaine	Burns, infected wounds, surgical wounds	
9	Regenecare Hydrogel	MPM Medical	Collagen, aloe vera gel base, sodium alginate	Pain relief and wound healing	
10	DermaSyn Ag Hydrogel	DermaRite	Polypropylene glycol hydrogel base with silver	Burns, diabetic ulcer	

7. Conclusion

In conclusion, Lamellar ichthyosis is a chronic genetic disorder resulting from mutations that disrupt normal skin barrier function and keratinization. Although it is not life-threatening, the condition significantly affects the quality of life due to persistent scaling, dryness, and associated complications. Early diagnosis and continuous management are essential to control symptoms and prevent secondary infections. Conventional therapies, including emollients, keratolytics, and retinoids, remain the mainstay of treatment. However, the incorporation of advanced drug delivery systems such as hydrogels offers a promising improvement in therapeutic outcomes. Hydrogels not only enhance skin hydration but also facilitate controlled drug release and better skin penetration, making them highly suitable for long-term topical application. Future research focusing on gene-based therapies and innovative formulations may further improve the management and prognosis of this condition.

Compliance with ethical standards

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Disclosure of conflict of interest

The authors have no conflicts of interest regarding this investigation.

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