XEROSIS: ALL YOU NEED TO KNOW

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Xerosis, or dry skin, is a common presenting complaint in Dermatology. It doesn't usually have serious implications but can be uncomfortable and aesthetically displeasing to patients.

Skin owes its soft, pliable texture to its water, lipid and natural moisturising factor (NMF) content. These are mediated by the skin's epidermal permeability barrier (EPB), which restricts transepidermal water loss. The organised arrangement of cells and lipids in the stratum corneum, the outermost layer of the epidermis, forms this barrier. Any alteration in the ratio of water, lipids and NMF in the epidermis can cause EPB dysfunction and subsequent cutaneous dehydration, which presents as dry skin.

The stratum corneum consists of corneocytes - cells that mature from keratinocytes of the deeper layers of the epidermis. Corneocytes are surrounded by an insoluble cornified envelope. The intercellular space between these corneocytes constitutes lipids in the form of cholesterol, ceramides and fatty acids. These lipids are the primary barrier to water loss. The skin requires a water content of 10%-15% to remain supple and intact.

Natural moisturising factor is present within corneocytes. NMF is a collective term for water-soluble compounds (urea, lactate, amino acids and pyrrolidone carboxylic acid) in the epidermis, produced by breakdown of the protein filaggrin. These compounds are highly effective endogenous humectants with a significant water-binding capacity. They attract and bind water from the atmosphere, drawing it into corneocytes. Sebum produced by sebaceous glands also contributes to stratum corneum hydration.

Although cells of the stratum corneum are non-viable, they are biochemically active as numerous enzymes still function in this layer and require adequate liquid water to perform optimally. Desmosomes are junctional bridges that hold the cells of the stratum corneum together. Enzyme degradation of desmosomes allows for shedding of superficial skin cells (desquamation).

CAUSES OF XEROSIS

Several factors, both endogenous and exogenous are known to affect the ratio of water, lipids and NMF in the skin. Most commonly, extremes of temperature and low air humidity are known to cause a reduction in skin water content. Although often an occupational necessity, excessive use of harsh detergents and excessive hand washing both remove NMF from the superficial layers of the epidermis and influence the skin's natural water binding capacity.

Other exogenous factors that might cause epidermal barrier dysfunction include frictional irritation from wool or synthetic fibres in clothing, as well as medications such as retinoids, diuretics, epidermal growth factor inhibitors, nicotinic acid and hydroxyurea.

Endogenous factors playing a role in epidermal barrier dysfunction and xerosis include advancing age. Epidermal and dermal changes both contribute to cutaneous aging and dehydration. Passage of keratinocytes from the deeper epidermal layers to superficial stratum corneum is slowed with age. This reduced keratinocyte proliferation rate negatively impacts corneocyte development and alters the EPB in this way. Water, lipid and NMF values become derranged with age.

An increase in the keratinocyte proliferation rate also influences EPB integrity by inhibiting uptake of nutrients (such as fatty acids), synthesis of proteins and lipids, and enzymatic activity.

Glycosaminoglycans (GAG), such as hyaluronic acid, reversibly bind water in the dermis and transport water to the epidermis. Reduced GAG levels in the dermis due to ageing result in reduced water content in the epidermis. Sebaceous gland activity also slows with age lowering sebum levels within the skin.

Co-existing medical conditions, such as hyper and hypothryoidism and malnutrition, may contribute to cutaneous dryness. Any cause of systemic dehydration, including vomitting, diarrhoea and profuse perspiration, can cause cutaneous dehydration.

Inflammatory skin conditions, such as atopic dermatitis (AD) and psoriasis, cause localised areas of xerosis. Patients with AD have inherent reduced lipid content in the stratum corneum, causing epidermal water loss and dry skin. Additionally, low NMF levels have been shown in AD, while in psoriatic skin and ichthyosis, NMF is essentially absent. This may be due to filaggrin gene mutations and resulting filaggrin deficiency. Hereditary disorders may cause severe, chronic xerosis. Genetic enzyme deficiencies can contribute to an ineffective or delayed desquamation process where flaky sheets of skin develop. The shed skin cells cannot detach from one another and are, therefore, more visible.

FACTORS INFLUENCING DRY SKIN:

ENDOGENOUS FACTORS	EXOGENOUS FACTORS
Age	Climate
Genetics	Clothing
Medical comorbidities	Excessive hand washing
Inflammatory skin disorders	Harsh detergents
	Medications: Diuretics, retinoids

PREVENTION

Xerosis causes mild to severe flaking of the skin as well as fissures and cracks. Itch is a frustrating symptom associated with cutaneous dryness, which may cause fissures to worsen and bleed. Secondary infection may ensue as any break in the skins epithelium predisposes to entry of microorganisms.

Both prevention and corrective intervention are necessary for management of xerotic conditions. Preventative measures involve eliminating aggravating factors.

Patients should:

- Avoid excessive hand washing with harsh soaps
- Replace standard soap with a soap-free cleanser, water-miscible emollient or bath oil
- Use rubber gloves to wash dishes
- Reduce shower duration
- Use a humidifier to increase air humidity
- Wear cotton or silk rather than clothing made of wool.

Moisturisers are one of the most important topical treatments for xerosis. Three categories of moisturisers exist: Emollients, humectants and occlusives.

Emollients soften and smooth the skin. Glycerol is an emollient, which allows degradation of desmosomes, thereby facilitating appropriate desquamation. It also plays a role in ceramide synthesis.

Humectants bind and hold water in the stratum corneum. Urea, lactate and Pyrrolidone Carboxylic Acid are all well-known humectants. They are widely used to replenish NMF content in the skin, alleviating the symptoms of dry skin, via external application of NFM-containing moisturisers.

Lactate improves and prevents the reappearance of symptoms of dry skin by stimulating the synthesis of ceramides in the stratum corneum. The strong water-binding potential of hyaluronic acid, allows hyaluronic acid based topical products to significantly improve skin hydration.

Occlusives provide a barrier that sits on the surface of the skin and prevents transepidermal water loss.

Moisturisers should be liberally applied. Emollients generally work best if applied to damp skin, for example, shortly after bathing. This simple topical therapy aids in reducing itch, improving barrier function and reducing transepidermal water loss.

Essential fatty acids in the diet of xerosis sufferers should be increased. These are found in salmon, herring, mackerel, nuts, avocados and flaxseed oil. Topical steroids are appropriate for inflammatory dermatoses or itch rather than dry skin in general. Topical calcineurin inhibitors can also be used. In inflammatory skin conditions, liberal use of moisturisers may improve the skin condition significantly such that the use of topical corticosteroids or calcineurin inhibitors could be minimised. This can aid, indirectly, in reducing potential adverse effects of topical steroids. MC

References available on request.