Keratosis pilaris (KP) is a common skin disorder characterized by rough follicular papules, giving it the unfortunate nickname “chicken skin.” It is most often seen in children and adolescents and is estimated to affect between 50 and 80 percent of all adolescents. It predominantly occurs on the extensor surfaces of the arms and legs but can also affect the face, buttocks, and trunk. There are several different KP variants, including less common variants such as KP atrophicans (KPA) and KP rubra (KPR). There have also been reports of drug-induced KP-like eruptions, with the most frequently documented contributing drugs being anticancer treatments such as vemurafenib and nilotinib. The pathophysiology of KP is still somewhat obscure but is currently believed to be due to three possible mechanisms: 1.) pathologic keratinization of the follicular epithelium; 2.) pathologic hair shaft development; and/or 3.) hypoplasia or aplasia of sebaceous glands. Despite its benign nature, KP can be a frustrating disorder for the patient, families, and clinicians, due to the cosmetic impact and lack of a clear solution. However, several topical therapies have been shown to reduce the severity of KP. Additionally, it is widely agreed that excellent moisturization, avoidance of overlong baths, and optimizing home humidity are reasonable baseline recommendations for most patients.

Keratolytic agents

Keratolytic agents such as lactic acid, salicylic acid, and urea cream have shown success in reducing the overall bumpy appearance of KP. (See Table)

In a 12-week randomized controlled trial, Kootiratrakarn et al. showed that both lactic acid (LA) 10% and salicylic acid (SA) 5% cream improved skin moisturization and the appearance of KP at weeks 4, 8, and 12. Improvement in KP was maintained until the follow-up period, four weeks after completion. Patients reported localized side effects such as skin irritation and malodor significantly more in the LA group than the SA group. In their final analysis, Kootiratrakarn et al. concluded that LA was somewhat more effective than SA at decreasing the follicular papules of KP. In another prospective cohort study combining LA with propylene glycol showed partial benefit in treating KP. Novick outlined a unique protocol to manage KP that incorporates a combination of keratolytic agents. He recommended gently massaging into skin a combination of salicylic acid 2% in urea cream 20% using a polyester sponge for five seconds for the first week of treatment. The duration of application can be gradually increased to build skin tolerance to the physical exfoliation of the sponge and to prevent excessive dryness. Once the KP is improved, Novick recommends 20% urea cream alone in place of the combination cream as an affordable over-the-counter long-term maintenance treatment.

Other topical treatments

Retinoids. Topical retinoids can improve KP through their ability to alter keratinocyte differentiation and via antiproliferative effects. In an open label study of 20 consecutive patients, Gerbig showed amelioration in KP using an oil-in-water emulsion containing 0.01% tazarotene nightly for four to eight weeks. Bogle at al. evaluated tazarotene 0.05% in a randomized, placebo-controlled, double blind prospective study of 33 patients using split application approach. At the last visit (week 12) tazarotene treated skin showed improved pruritus, erythema, and roughness compared to vehicle-treated skin. Another case series showed that tretinoin 0.025% showed slight effectiveness in treating drug-induced KP.

Corticosteroids. Topical steroids were not found to be useful in treating KP in two prospective cohort studies, however they may have beneficial effects in more inflammatory presentations and in KPA.

Tacrolimus. A double-blind study compared tacrolimus 0.1% ointment, a topical calcineurin inhibitor, with a petroleum-based moisturizer and found that both were beneficial in treating KP.

There are promising interventions for this challenging presentation.

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likely to lead to marked improvement by end of week 4, although statistical significance was not found, perhaps due to the small sample size of the study.\(^\text{18}\)

**ALTERNATIVE HYPOTHESIS OF PATHOGENESIS AND FUTURE THERAPEUTIC CONCEPTS**

The accumulation of keratin debris within the follicular orifices appears to cause the rough and bumpy texture of KP-involved skin. What is less clear is whether these findings are a primary or a secondary feature. In a study by Gruber et al., skin biopsies were taken from ten subjects with KP of lesional and non-lesional skin.\(^\text{19}\) There was a striking absence of sebaceous glands in biopsies from lesional skin compared to non-lesional skin histology of the same individual in all cases. Without sebaceous gland-derived lipids (i.e., sebum) corneocytes are unlikely to mature and shed properly, and hair shafts are unlikely to properly form, leading to hyperkeratinization of the acroinfundibula and subsequent chronic inflammation. Perhaps it is this sebaceous gland aplasia that is at the crux of keratosis pilaris pathophysiology, at least for some KP subtypes.

**KERATOSIS PILARIS AND OTHER SKIN DISORDERS**

When considering treatment for KP, it may be helpful to view KP as a disorder of hyperkeratosis and examine other effective treatments for this more general category of disease. There are many other skin disorders that fall under the umbrella of hyperkeratosis including actinic keratoses. Actinic keratoses can be treated with Diclofenac topical gel,\(^\text{20,21}\) an anti-inflammatory agent, and this may be a potential treatment option for KP, although studies are needed to investigate this hypothesis.
KP also has a strong link with atopy, ichthyosis vulgaris and xerosis. Therefore, oral supplements that have been shown to improve atopic dermatitis and skin dryness could be useful for KP. One such oral supplement is L-histidine, which plays an important role in forming the skin barrier protein filaggrin. Subsequent filaggrin proteolysis releases L-histidine as an important component of natural moisturizing factor (NMF), therefore L-histidine can potentially improve skin hydration by improving barrier function. A randomized, double-blind, placebo-controlled nutritional supplementation pilot study showed that L-histidine significantly increased both filaggrin formation and skin barrier function.

Hempseed oil has also been shown to improve the clinical symptoms of atopic dermatitis such as skin dryness and itchiness, which are symptoms seen in KP as well. A randomized, single-blind crossover study showed that oral administration of hempseed oil for 20 weeks resulted in significant changes in plasma fatty acid profiles and improvement in skin dryness and itchiness, along with decrease in use of topical medications. This may be due to the fact that hempseed oil is an abundant source of omega-6 and omega-3 polyunsaturated fatty acids (PUFAs).

In contrast, evening primrose and borage oils also contain omega-6 and high levels of gamma-linolenic acid (GLA) but lack omega-3 PUFAs, which may explain their—at best—substantial improvement in skin dryness and itchiness, along with decrease in use of topical medications. This may be due to the fact that hempseed oil is an abundant source of omega-6 and omega-3 polyunsaturated fatty acids (PUFAs).

Another oral supplement that could have potential benefit is flaxseed oil, which contains high amounts of lipids similar to those in the skin. In a study conducted on an atopic-dermatitis model in mice, topical application of flaxseed oil significantly alleviated symptoms such as redness, swelling, and itchiness.

**CONCLUSION**

KP is an extremely common, benign skin disorder that can be distressing for the patient due to its appearance on visible areas of the body, dryness, and rough texture. Although there is no clear consensus on the solution for KP, many different treatments are available to reduce its symptoms. The overall goal is to reduce abnormal folliculocentric thickening and dryness so treatments for related skin disorders such as atopic dermatitis should be considered when treating the symptoms of KP. Oral supplements that improve skin hydration or barrier may emerge as potential therapies for KP, although further large-scale studies are needed. Lastly, consideration of the sebaceous gland aplasia concept as a possible root cause of KP should be considered as a potential pathway to alternate therapeutic venues.