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# TREATMENT OF KERATOSIS PILARIS: A NARRATIVE LITERATURE REVIEW OF TOPICAL, PROCEDURAL, AND VARIANT-SPECIFIC THERAPIES

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**ABSTRACT**

Keratosis pilaris (KP) is a common disorder of follicular keratinization characterized by keratotic perifollicular papules with variable erythema and, in some patients, dyspigmentation. Although benign, KP may generate substantial cosmetic concern and psychosocial burden. Therapeutic management remains challenging because available interventions often produce incomplete and temporary improvement, and the evidence base is limited by small sample sizes, heterogeneous study designs, short follow-up, and inconsistent outcome measures. This narrative literature review synthesizes current evidence on treatment of KP, emphasizing topical keratolytics, retinoids, anti-inflammatory agents, laser- and light-based therapies, and emerging approaches. A separate section addresses keratosis pilaris atrophicans (KPA) and keratosis pilaris rubra faciei/rubra (KPRF/KPR), which warrant a distinct therapeutic approach because erythema, inflammation, and scarring risk may predominate over texture alone. Overall, treatment is difficult and rarely curative; however, a careful, phenotype-directed, stepwise approach can yield clinically meaningful improvement in roughness, erythema, and pigmentary change.

**Aim:** The purpose of this review is to present a comprehensive overview of the therapeutic approaches used in the treatment of keratosis pilaris. It outlines the roles of topical agents, procedural therapies, and emerging interventions, and reviews both commonly used treatments with broader clinical experience and newer or less well-established modalities supported by limited evidence. It also addresses variant-specific management of keratosis pilaris rubra/rubra faciei and keratosis pilaris atrophicans, in which erythema, inflammation, and scarring risk may influence treatment selection.

**Material and methods:** A literature search was conducted in PubMed and Google Scholar using the keywords “keratosis pilaris,” “keratosis pilaris rubra faciei,” and “treatment.” Additional relevant articles were identified through reference screening. Original studies, review articles, and other clinically relevant sources on therapeutic approaches and treatment outcomes were considered for this narrative review.

**Conclusions:** Keratosis pilaris and its erythematous variants can be challenging to treat, currently available therapies may provide meaningful clinical improvement, particularly when selected according to the predominant phenotype and patient-specific concerns. Further high-quality studies are needed to refine treatment strategies and strengthen the evidence base for individualized management.

**KEYWORDS**

Keratosis Pilaris, Keratosis Pilaris Rubra Faciei, Keratosis Pilaris Atrophicans, Follicular Keratosis, Topical Therapy, Keratolytics, Retinoids, Laser Therapy, Light-Based Therapy, Nd:YAG Laser, Pulsed Dye Laser, Intense Pulsed Light

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## 1. Introduction

Keratosis pilaris is a highly prevalent, chronic, usually benign disorder of keratinization presenting with rough folliculocentric papules, classically on the extensor upper arms, thighs, buttocks, and sometimes the cheeks [1-4]. Clinical variants include KP alba, KP rubra, erythromelanosis follicularis faciei et colli (EFFC), and the keratosis pilaris atrophicans spectrum, which includes ulerythema ophryogenes/keratosis pilaris atrophicans faciei (KPAF), atrophoderma vermiculatum, and keratosis follicularis spinulosa decalvans [2,5-7]. Despite its benign nature, visible involvement of exposed areas and persistent roughness or redness frequently motivate treatment [1,3,8].

The therapeutic literature has expanded in recent years, but no single intervention has emerged as uniformly effective or durable. Most patients require long-term maintenance, and recurrence after treatment cessation is common [2,8-10]. The present review summarizes the contemporary evidence base and proposes a practical, academically grounded synthesis of treatment options.

## 2. Research materials and methods

A comprehensive literature search was conducted using PubMed and Google Scholar to identify publications on the treatment of keratosis pilaris and keratosis pilaris rubra faciei. The search strategy included the keywords “keratosis pilaris,” “keratosis pilaris rubra faciei,” and “treatment.” Additional relevant studies were identified by screening the reference lists of included articles. Original studies, review articles, and other clinically relevant sources addressing therapeutic approaches, treatment outcomes, and clinical management were considered for inclusion.

After removal of duplicates and clearly irrelevant records, the evidence base for this narrative review comprised 60 cited sources, including reviews, randomized and nonrandomized trials, cohort studies, case series, case reports, and expert-reference summaries. Priority was given to studies with concrete treatment outcome data, subtype-specific information, and clinically interpretable endpoints.

## 3. Pathophysiology

KP is generally understood as a disorder of follicular keratinization in which abnormal cornification leads to keratin plugging of the follicular infundibulum, perifollicular rough papules, and variable perifollicular inflammation [1-3,11,21]. Histopathologically, lesions demonstrate orthokeratotic plugging of dilated follicular ostia, often with trapped or coiled hair shafts, hyperkeratosis, and a mild perifollicular lymphocytic inflammatory infiltrate [1,2,5,21]. This mechanistic framework underlies the rationale for keratolytic and retinoid therapy.

Genetic predisposition appears important. Associations with filaggrin-related barrier dysfunction have been repeatedly described, and epidermal barrier abnormalities, sebaceous gland changes, and hair shaft anomalies may contribute to disease expression [2,11,22]. Gruber et al. provided histologic and molecular evidence that KP may involve sebaceous gland and epidermal barrier abnormalities in addition to follicular plugging alone [11]. More recent reviews also emphasize that KP is not merely “cosmetic roughness,” but likely reflects interplay between aberrant keratinization, inflammation, hair shaft morphology, and barrier impairment [2,8,21].

This pathophysiology has therapeutic implications. Keratolytics aim to reduce infundibular plugging; emollients and urea-based preparations attempt to restore hydration and barrier function; retinoids normalize keratinocyte differentiation; and vascular or follicular laser therapies may better address erythema, pigment, or follicular prominence than topicals alone [1-3,8,10].

## 4. General Principles of Management

Treatment goals in KP are realistic rather than curative: reduction of roughness, erythema, pruritus, or dyspigmentation, with long-term control rather than eradication [1,3,5,8]. Patients should be counseled that spontaneous improvement with age may occur, yet persistence into adulthood is common, and relapse after discontinuation of therapy is frequent [1,5,23]. General skin care remains foundational, especially in xerotic or atopic patients: mild cleansers, avoidance of harsh friction, reduced exposure to hot water, and routine moisturization are standard adjunctive measures [1,3,5].

Because KP phenotypes differ, treatment selection should be phenotype-directed. Texture-dominant disease may respond best to emollients, keratolytics, and retinoids; erythematous disease may require a greater emphasis on anti-inflammatory care or vascular/light-based modalities; pigmentary variants may benefit from chemical exfoliation or selected lasers; and scarring/atrophic variants require more cautious, specialized management [2,5,8,10].

### 4.1. Emollients, Barrier Repair, and Urea

Emollients and barrier-supportive skin care are widely regarded as first-line therapy, particularly for patients with mild disease or prominent xerosis [1,3,5,8]. Although emollients alone rarely eliminate papules, they reduce scaling, soften roughness, improve tolerability of active treatment, and may be especially useful in patients with concurrent atopic dermatitis or ichthyosis vulgaris [1,5,11].

Urea occupies a particularly important position because of its combined humectant, barrier-supportive, and keratolytic properties [24]. Earlier expert sources and reviews recommended urea-containing preparations empirically [1,3,5], but newer evidence has strengthened this practice. In a 2024 open-label clinical study, a 20% urea cream improved skin texture and appearance over 4 weeks and was generally well tolerated [25]. Staubach et al. also reported improvement in texture, erythema, dryness, and overall appearance with an exfoliating cleanser plus urea-containing cream, though this was published as an abstract and should be interpreted cautiously [8,26].

The main limitation of emollient-centered therapy is that benefit tends to be modest and maintenance-dependent. Urea and moisturizers may improve roughness more consistently than erythema, and monotherapy is often insufficient in moderate or cosmetically troubling disease [1,5,8].

### 4.2. Keratolytic Acids

Topical keratolytics remain the cornerstone of pharmacologic treatment for classic KP. The principal agents studied include lactic acid, salicylic acid, glycolic acid, azelaic acid, and, less robustly, combination exfoliating regimens [3,8,10].

### 4.3. Lactic acid and salicylic acid

Kootiratrakarn et al. conducted a randomized, rater-blinded pilot study comparing lactic acid 10% with salicylic acid 5% cream applied twice daily for 12 weeks [12]. Both regimens significantly improved KP lesions, supporting the common clinical practice of using alpha- or beta-hydroxy acid formulations for papular roughness [12]. The study also emphasized the relevance of epidermal permeability barrier dysfunction, suggesting that successful therapy may require both exfoliation and barrier support [12].

Salicylic acid is mechanistically attractive because of its keratolytic and anti-inflammatory properties, but direct comparative evidence remains sparse [8,10]. In practice, it appears most useful in roughness-dominant disease, usually as part of a moisturizer-based regimen rather than as a standalone therapy [1,3,5].

### 4.4. Glycolic acid

Among keratolytics, glycolic acid has some of the more detailed prospective data. Tian et al. studied 25 patients treated with high-concentration glycolic acid in escalating regimens (50% and 70% for 5-7 minutes) at 20-day intervals for four sessions [13]. Significant reductions in papule counts were seen by day 20 and progressed through day 80, while pigmentation and erythema indices improved later during treatment [13]. The treatment was well tolerated overall, with only one patient experiencing transient burning/itching during

70% glycolic acid application [13]. However, long-term durability was poor: in the subset followed for five years, there was no significant difference from pretreatment values [13].

These data are clinically useful because they illustrate a recurring theme in KP management: keratolytic procedures can produce meaningful short-term cosmetic gains, but maintenance strategies are necessary, and durable remission is uncommon [8,13]. Lower-strength glycolic acid in leave-on products is often used in routine practice, though strong trial-level evidence remains limited [3,10].

### 4.5. Azelaic acid

Evidence for azelaic acid is modest. A right-left comparative study found that 20% azelaic acid improved hyperkeratosis and erythema, but the magnitude of benefit was comparable to moisturizer control in some outcomes [8,27]. Thus, azelaic acid may be considered in patients seeking a relatively gentle topical option, especially where erythema or postinflammatory dyspigmentation is also a concern, but it cannot currently be regarded as clearly superior to simpler moisturization strategies.

### 4.6. Topical Retinoids and Systemic Retinoids

Retinoids aim to normalize abnormal follicular keratinization and have long been used in KP, although the evidence base remains inconsistent [1,3,8,28].

**Topical retinoids**

Tazarotene has the best direct evidence among topical retinoids. In a randomized, placebo-controlled study, tazarotene 0.05% cream improved itching, roughness, and erythema compared with vehicle [29]. In an earlier open-label study, tazarotene 0.01% emulsion led to improvement or resolution within weeks in some patients [30]. Topical tretinoin has also been reported to improve hyperkeratotic and pigmentary features in isolated cases [31], but irritation and exacerbation of erythema may limit its use, especially in erythematous variants [8,28].

Clinically, topical retinoids are best viewed as second-line agents for patients who do not respond adequately to emollients and keratolytics [1,5]. Their major drawbacks are irritation, reduced tolerability in sensitive skin, and the need for prolonged use. In facial or rubra-predominant disease, cautious introduction is advisable because irritation can worsen redness.

**Oral isotretinoin**

Systemic retinoids have been reported primarily in severe or atypical disease, especially within the KPA spectrum. Baden and Byers found limited or inconsistent benefit from isotretinoin in patients with KPA [14]. By contrast, Kirchner and Hoyer described successful treatment of severe KP with a structured isotretinoin regimen, with benefit persisting after treatment cessation in a single case [32]. Historical reports also describe responses of atrophoderma vermiculatum or keratosis follicularis spinulosa decalvans to isotretinoin or etretinate [5,33,34].

Taken together, systemic retinoids may be considered in exceptional, severe, refractory cases under specialist supervision, but they cannot be recommended for routine KP because evidence is limited, relapse is common, and the risk-benefit ratio is unfavorable for a benign condition.

### 4.7. Anti-inflammatory Topicals: Corticosteroids, Tacrolimus, and Sirolimus

Inflammatory components of KP, especially in rubra phenotypes, have prompted the use of corticosteroids and calcineurin/mTOR-directed agents.

**Topical corticosteroids**

Topical corticosteroids have traditionally been used short term when inflammation is prominent [1,3,5]. However, direct evidence is weak. Baden and Byers reported that tretinoin combined with topical steroids could be more tolerable than tretinoin alone in KPA, where retinoid monotherapy worsened redness [14]. In contrast, hydrocortisone alone produced unsatisfactory results in a case of ulerythema ophryogenes/KPAF [35]. Thus, corticosteroids may have an adjunctive role for short courses in inflamed lesions, but they are not disease-modifying and are not appropriate long-term monotherapy.

**Tacrolimus**

Tacrolimus 0.1% ointment has been investigated in several studies. Conley et al. and Breithaupt et al. found that tacrolimus produced improvement, but outcomes were similar to emollient comparators such as Aquaphor [36,37]. A later prospective interventional study also reported benefit, again with only limited separation from simple occlusive therapy [38]. This suggests that part of the apparent benefit may derive from moisturization and barrier repair rather than immunomodulation per se.

**Sirolimus**

Interest in sirolimus reflects the possibility that vasoinflammatory mechanisms may contribute to erythematous KP variants. The most notable evidence is a 2022 case report by Eckburg et al., in which topical sirolimus 1% cream led to successful treatment of keratosis pilaris rubra in an adolescent patient [20]. Although intriguing, this remains anecdotal evidence and should currently be interpreted as hypothesis-generating rather than practice-changing.

### 4.8. Laser- and Light-Based Therapies

Procedural therapy has attracted growing interest because topical treatments frequently provide incomplete benefit. Across the contemporary literature, laser and light modalities have the strongest evidence for improving KP, especially when erythema and pigmentary change are substantial [8,10,18,19].

**Nd:YAG laser**

Among available devices, 1064-nm Nd:YAG laser has the most consistently favorable data. Park et al. reported meaningful improvement after ten biweekly Q-switched Nd:YAG sessions [39]. Saelim et al. subsequently demonstrated significant benefit in a randomized, evaluator-blind study using long-pulsed 1064-nm Nd:YAG laser [15]. Maitriwong et al. confirmed efficacy in a double-blind, sham-controlled trial, strengthening confidence that the effect exceeds placebo [16]. Hassan et al. found long-pulsed 1064-nm Nd:YAG laser superior or at least highly competitive relative to 20% trichloroacetic acid peel, with favorable clinical and dermoscopic improvement [17]. Comparative work by Bayazit et al. also supported efficacy versus fractional Er:YAG [18].

The likely advantages of Nd:YAG include deeper penetration, relative safety in darker skin phototypes, and the ability to improve roughness, perifollicular erythema, and dyspigmentation in the same patient [8,10]. Adverse effects are usually transient and include erythema, stinging, petechiae, or purpura [8,15-18,39].

**Pulsed dye laser**

Pulsed dye laser (PDL) has been used primarily for erythematous variants. Outcomes are mixed. Clark et al. documented erythema reduction in KPA but less consistent improvement in roughness [40]. Alcántara González et al. reported benefit in 10 cases of KPR and KPAF [19], while Schoch et al. described successful treatment of pediatric KPR [9]. Kaune et al. also reported marked improvement in severe KPR with 595-nm PDL [41]. However, recurrence of erythema and adverse effects such as purpura, swelling, and post-treatment discomfort limit enthusiasm [8-10,19,40,41].

Thus, PDL appears particularly useful when erythema is the dominant problem, but it is less convincing for texture improvement than Nd:YAG.

**Intense pulsed light**

Intense pulsed light (IPL) has demonstrated efficacy in several small studies. Eyler et al. reported improvement in KP severity after serial IPL sessions [42]. Maitriwong et al. later showed significant superiority of IPL over sham irradiation in a randomized, double-blind study, with improvement in roughness, erythema, hyperpigmentation, and global appearance [43]. However, adverse events including stinging, erythema, and in some cases substantial burning/desquamation have been reported [8,42].

In subtype-specific contexts, IPL has also shown value in KPA and EFFC [8,43,44]. It may therefore be a reasonable option when erythema and pigment coexist, though its adverse-effect profile may be less favorable than Nd:YAG in some patients.

**Fractional CO2, alexandrite, diode, and other devices**

Fractional CO2 laser has shown efficacy in randomized and comparative studies, but the balance between benefit and downtime is less favorable than for Nd:YAG [45,46]. Ismail and Omar found fractional CO2 comparable to topical urea in some clinical endpoints [46], while Sobhi et al. reported that Nd:YAG alone or in combination outperformed fractional CO2 monotherapy [47]. Vachiramon et al. demonstrated improvement in a randomized comparative study, but pain, erythema, burning, and transient dyspigmentation were common [45].

Long-pulsed 755-nm alexandrite laser has also shown benefit. Li et al. reported significant improvements in follicular plugs, erythema, and hyperpigmentation in a split-body randomized clinical trial [48]. Combination regimens using PDL, alexandrite laser, and microdermabrasion improved outcomes in retrospective series, though it is difficult to isolate the contribution of individual components [49,50].

Diode laser has randomized clinical trial support as well. Ibrahim et al. showed benefit with 810-nm diode laser, though postinflammatory hyperpigmentation occurred in some patients [51]. Additional devices

such as KTP laser, pro-yellow laser, radiofrequency, and photopneumatic therapy remain supported mainly by isolated reports or preliminary data [8,52,53].

Overall, procedural evidence suggests that laser and light therapy currently offers the most robust route to improvement in moderate-to-severe KP, especially where erythema and dyspigmentation are prominent. Nd:YAG presently appears to have the strongest overall balance of efficacy and tolerability [8,10].

### 4.9. Other and Emerging Therapies

Several unconventional strategies have been reported, including microdermabrasion, sonic brushes, chlorine dioxide wash, polymer coatings, fractional prickle coral calcium peels, and microbiome-restorative interventions [8,49,54-57]. Most of these studies are small, uncontrolled, or confounded by concurrent keratolytics, making interpretation difficult.

Microdermabrasion is often used as an adjunct rather than a standalone intervention and may help texture irregularity, but rigorous KP-specific data are limited [5,49]. A randomized trial of dermal microflora restoration using ammonia-oxidizing bacteria suggested potential symptomatic benefit, but this remains investigational [57]. At present, these approaches should be considered experimental or adjunctive rather than standard of care.

## 5. Keratosis Pilaris Atrophicans and Keratosis Pilaris Rubra Faciei/Rubra

Although these entities overlap with classic KP, they warrant separate consideration because treatment goals differ. In KPR/KPRF, erythema and cosmetic redness may dominate; in KPA, inflammation may progress to fibrosis, atrophy, alopecia, and permanent eyebrow loss, making early control conceptually important even when evidence is weak [5-7,14].

### 5.1. Keratosis pilaris rubra / rubra faciei

KPR is underrecognized and tends to persist beyond puberty more often than KP alba [6]. Because erythema is central, conventional keratolytics alone are often insufficient. PDL has the most specific subtype-oriented support: Alcántara González et al. described improvement in KPR/KPAF [19], Schoch et al. reported successful pediatric KPR treatment [9], and Kaune et al. documented a favorable response in severe KPR [41]. More recently, Temiz et al. described benefit with 577-nm pro-yellow laser [53]. IPL has also been used for KPR and related erythematous phenotypes [8,43].

Topical sirolimus is a promising but still anecdotal option. In the case reported by Eckburg et al., prolonged use of topical sirolimus 1% cream was associated with marked improvement in KPR [20]. This is of interest because it suggests that some cases may respond to antiangiogenic and anti-inflammatory approaches, but broader evidence is lacking.

Practically, KPR/KPRF should be approached as an erythema-predominant disorder. Gentle barrier repair and low-irritancy keratolytics may help roughness, but vascular laser/light modalities are generally the most rational interventions when facial redness is the major complaint [8,10,20].

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### 5.2. Keratosis pilaris atrophicans

KPA includes ulerythema ophryogenes/KPAF, atrophoderma vermiculatum, and keratosis follicularis spinulosa decalvans [5,7]. Pathogenetically, follicular plugging is followed by chronic perifollicular inflammation, fibrosis, follicular atrophy, and alopecia [5]. Consequently, treatment is less about smoothing papules and more about limiting inflammatory activity, reducing erythema, and managing permanent sequelae.

The prognosis is less favorable than for classic KP. UpToDate states that no consistently effective therapy exists, and topical corticosteroids, keratolytics, and retinoids are of limited value [5]. Baden and Byers similarly described generally unsatisfactory results across multiple interventions [14]. Nevertheless, selected patients may improve. Oral isotretinoin has been reported to benefit atrophoderma vermiculatum and inflammatory KFSD in isolated cases [5,33,34]. PDL may reduce erythema in KPAF [19,40], and IPL improved KPA in small series [44,58]. Once disease has become dormant, eyebrow reconstruction by hair transplantation has also been described [59].

These observations support a separate treatment philosophy for KPA: early, gentle attempts to suppress inflammation and erythema; realistic counseling about scarring permanence; and consideration of reconstructive approaches once inflammatory activity has subsided. Aggressive irritating topical regimens are often poorly tolerated and may be counterproductive in already inflamed facial skin [5,14,35].

## 6. Limitations of the Evidence

The KP treatment literature remains methodologically limited. Many studies are single-center, include small numbers of participants, employ nonvalidated or heterogeneous endpoints, and have short follow-up [8,10,13]. Subtype mixing is another problem: studies often combine classic KP, KPR, KPA, and EFFC, despite meaningful biological and therapeutic differences. Direct head-to-head comparisons are scarce, and maintenance strategies are poorly studied. Even when short-term benefit is convincing, long-term durability is usually not established, as illustrated by the five-year relapse data after glycolic acid treatment [13].

A further limitation is the absence, until recently, of a standardized KP-specific severity instrument. This makes cross-study synthesis difficult and likely contributes to the apparent inconsistency of results [8,60]. Future research should prioritize validated scoring systems, phenotype-specific enrollment, and pragmatic comparative trials.

## 7. Conclusions

Current evidence supports a stepwise, phenotype-directed approach to KP. Emollients and keratolytics remain the practical first-line foundation for classic texture-dominant disease, while topical retinoids serve as second-line options in selected patients. Laser and light therapies, particularly 1064-nm Nd:YAG and, for erythematous disease, PDL or IPL, presently offer the strongest evidence for clinically meaningful improvement, especially when redness and dyspigmentation are prominent. By contrast, KPA and KPR/KPRF require a more tailored strategy that places greater emphasis on erythema control, inflammation minimization, and scarring prevention or camouflage.

Treatment of KP remains difficult, often incomplete, and maintenance-dependent. Nonetheless, a careful, methodical, phenotype-specific approach can yield worthwhile improvement in symptoms and appearance, even if permanent remission remains uncommon.

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Conceptualization: TM, PB
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Writing-review and editing: TM, JM
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## REFERENCES


1. Landis, M. N., Fowler, J., & Corona, R. (2026, February 10). *Keratosis pilaris*. UpToDate. https://www.uptodate.com/contents/keratosis-pilaris
2. Wang, J. F., & Orlow, S. J. (2018). Keratosis pilaris and its subtypes: Associations, new molecular and pharmacologic etiologies, and therapeutic options. *American Journal of Clinical Dermatology, 19*, 733–757. https://doi.org/10.1007/s40257-018-0368-3
3. Kodali, N., Patel, V. M., & Schwartz, R. A. (2023). Keratosis pilaris: An update and approach to management. *Italian Journal of Dermatology and Venereology, 158*, 217–223. https://doi.org/10.23736/S2784-8671.23.07594-1
4. Hwang, S., & Schwartz, R. A. (2008). Keratosis pilaris: A common follicular hyperkeratosis. *Cutis, 82*, 177–180. https://cdn.mdedge.com/files/s3fs-public/Document/September-2017/082030177.pdf
5. Landis, M. N., Fowler, J., & Corona, R. (2024, June 4). *Keratosis pilaris atrophicans*. UpToDate. https://www.uptodate.com/contents/keratosis-pilaris-atrophicans
6. Marqueling, A. L., Gilliam, A. E., Prendiville, J., et al. (2006). Keratosis pilaris rubra: A common but underrecognized condition. *Archives of Dermatology, 142*, 1611–1616. https://doi.org/10.1001/archderm.142.12.1611
7. Arnold, A. W., & Buechner, S. A. (2006). Keratosis pilaris and keratosis pilaris atrophicans faciei. *Journal der Deutschen Dermatologischen Gesellschaft, 4*, 319–323. https://doi.org/10.1111/j.1610-0387.2006.05933.x
8. Wong, P. C., Wang, M. A., Ng, T. J., et al. (2024). Keratosis pilaris treatment paradigms: Assessing effectiveness across modalities. *Clinical and Experimental Dermatology, 49*, 1105–1117. https://doi.org/10.1093/ced/llae066
9. Schoch, J. J., Tollefson, M. M., Witman, P. M., & Davis, D. M. R. (2016). Successful treatment of keratosis pilaris rubra with pulsed dye laser. *Pediatric Dermatology, 33*, 443–446. https://doi.org/10.1111/pde.12892
10. Maghfour, J., Ly, S., Haidari, W., et al. (2022). Treatment of keratosis pilaris and its variants: A systematic review. *Journal of Dermatological Treatment, 33*, 123–142. https://doi.org/10.1080/09546634.2020.1818678
11. Gruber, R., Sugarman, J. L., Crumrine, D., et al. (2015). Sebaceous gland, hair shaft, and epidermal barrier abnormalities in keratosis pilaris with and without filaggrin deficiency. *American Journal of Pathology, 185*, 1012–1021. https://doi.org/10.1016/j.ajpath.2014.12.012
12. Kootiratrakarn, T., Kampirapap, K., & Chunhasewee, C. (2015). Epidermal permeability barrier in the treatment of keratosis pilaris. *Dermatology Research and Practice, 2015*, Article 205012. https://doi.org/10.1155/2015/205012
13. Tian, Y., Li, X. X., Zhang, J. J., et al. (2021). Clinical outcomes and 5-year follow-up results of keratosis pilaris treated by a high concentration of glycolic acid. *World Journal of Clinical Cases, 9*, 4681–4689. https://doi.org/10.12998/wjcc.v9.i18.4681
14. Baden, H. P., & Byers, H. R. (1994). Clinical findings, cutaneous pathology, and response to therapy in 21 patients with keratosis pilaris atrophicans. *Archives of Dermatology, 130*, 469–475. https://doi.org/10.1001/archderm.1994.01690040073011
15. Saelim, P., Pongprutthipan, M., Pootongkam, S., et al. (2013). Long-pulsed 1064-nm Nd:YAG laser significantly improves keratosis pilaris: A randomized, evaluator-blind study. *Journal of Dermatological Treatment, 24*, 318–322. https://doi.org/10.3109/09546634.2012.660518
16. Maitriwong, P., Tangkijngamvong, N., & Asawanonda, P. (2020). Innovative 1064-nm Nd:YAG laser significantly improves keratosis pilaris: A randomized, double-blind, sham-irradiation-controlled trial. *Lasers in Surgery and Medicine, 52*, 509–514. https://doi.org/10.1002/lsm.23184
17. Hassan, A. S., Abdel Aziz, M., & Saadi, D. G. (2022). Clinical and dermoscopic evaluation of trichloroacetic acid 20% versus long-pulsed 1064-nm Nd:YAG laser in the treatment of keratosis pilaris. *Dermatologic Surgery, 48*, 838–842. https://doi.org/10.1097/dss.0000000000003488
18. Bayazit, S., Akın, Ö., & Kutlubay, Z. (2022). Comparative study of the efficacy of fractional Er:YAG 2940 nm laser and Q-switched Nd:YAG 1064 nm laser in keratosis pilaris. *Journal of Cosmetic Dermatology, 21*, 3809–3813. https://doi.org/10.1111/jocd.15193
19. Alcántara González, J., Boixeda, P., Truchuelo Díez, M. T., & Fleta Asín, B. (2011). Keratosis pilaris rubra and keratosis pilaris atrophicans faciei treated with pulsed dye laser: Report of 10 cases. *Journal of the European Academy of Dermatology and Venereology, 25*, 710–714. https://doi.org/10.1111/j.1468-3083.2010.03772.x
20. Eckburg, A., Kazemi, T., & Maguiness, S. (2022). Keratosis pilaris rubra successfully treated with topical sirolimus: Report of a case and review of the literature. *Pediatric Dermatology, 39*, 429–431. https://doi.org/10.1111/pde.14963
21. Thomas, M., & Khopkar, U. S. (2012). Keratosis pilaris revisited: Is it more than just a follicular keratosis? *International Journal of Trichology, 4*, 255–258. https://doi.org/10.4103/0974-7753.111215
22. Landeck, L., Visser, M., Kezic, S., & John, S. M. (2013). Genotype-phenotype associations in filaggrin loss-of-function mutation carriers. *Contact Dermatitis, 68*, 149–155. https://doi.org/10.1111/j.1600-0536.2012.02171.x
23. Poskitt, L., & Wilkinson, J. D. (1994). Natural history of keratosis pilaris. *British Journal of Dermatology, 130*, 711–713. https://doi.org/10.1111/j.1365-2133.1994.tb03406.x

24. Piquero-Casals, J., Morgado-Carrasco, D., Granger, C., et al. (2021). Urea in dermatology: A review of its emollient, moisturizing, keratolytic, skin barrier enhancing and antimicrobial properties. *Dermatology and Therapy, 11*, 1905–1915. https://doi.org/10.1007/s13555-021-00611-y
25. McCormick, E., Nussbaum, D., Friedman, A., et al. (2024). Evaluation of a moisturizing cream with 20% urea for keratosis pilaris. *Journal of Drugs in Dermatology, 23*, 1274–1277. https://doi.org/10.36849/jdd.7806
26. Staubach, P., Baabaki, N., Yatskayer, M., & Colon, G. (2020). Evaluation of the efficacy of an exfoliating cleanser and urea-containing cream on keratosis pilaris using clinical and 3D image-based analysis. *Journal of the American Academy of Dermatology, 83*(Suppl.), AB90. https://doi.org/10.1016/j.jaad.2020.06.449
27. Yang, G., Bordeaux, J., & Ou, J. C. (2012). Prospective right-left comparison of azelaic acid and Cetaphil for treatment of keratosis pilaris. *Journal of the American Academy of Dermatology, 66*(4 Suppl. 1), AB167. https://doi.org/10.1016/j.jaad.2011.11.702
28. Motamedi, M., Chehade, A., Sanghera, R., & Grewal, P. (2022). A clinician's guide to topical retinoids. *Journal of Cutaneous Medicine and Surgery, 26*, 71–78. https://doi.org/10.1177/12034754211035091
29. Bogle, M. A., Ali, A., & Bartel, H. (2004). Tazarotene 0.05% cream for the treatment of keratosis pilaris. *Journal of the American Academy of Dermatology, 50*, P39. https://doi.org/10.1016/j.jaad.2003.10.158
30. Gerbig, A. W. (2002). Treating keratosis pilaris. *Journal of the American Academy of Dermatology, 47*, 457. https://doi.org/10.1067/mjd.2002.122733
31. Lee, Y., Son, S. J., Han, T. Y., & Lee, J. H. (2018). A case of atrophoderma vermiculatum showing a good response to topical tretinoin. *Annals of Dermatology, 30*, 116–118. https://doi.org/10.5021/ad.2018.30.1.116
32. Kirchner, A., & Hoyer, S. (2022). A detailed regimen of isotretinoin for the successful treatment of severe keratosis pilaris. *Clinical and Experimental Dermatology, 47*, 619–621. https://doi.org/10.1111/ced.15020
33. Weightman, W. (1998). A case of atrophoderma vermiculatum responding to isotretinoin. *Clinical and Experimental Dermatology, 23*, 89. https://doi.org/10.1046/j.1365-2230.1998.00326.x
34. Richard, G., & Harth, W. (1993). Keratosis follicularis spinulosa decalvans: Therapy with isotretinoin and etretinate in the inflammatory stage. *Hautarzt, 44*, 529–534. https://pubmed.ncbi.nlm.nih.gov/8376108/
35. Edelstein, J. E. (2007). Ulerythema ophryogenes successfully treated with hydrocortisone 2.5% ointment. *Cutis, 79*, 357–359. https://doi.org/10.1001/archderm.1955.01540280026004
36. Saka, S., Tatavarthi, R., Kolalapudi, S. A., Prasad Arumilli, K. P., Gandikota, R., & Arumilli, P. C. (2022). Efficacy of tacrolimus 0.1% ointment in keratosis pilaris: A prospective hospital based interventional study. *Journal of Medical Sciences, 42*(1), 22–25. https://doi.org/10.4103/jmedsci.jmedsci_356_20
37. Breithaupt, A. D., Alio Saenz, A. B., Weaver, A. L., & Davis, D. M. R. (2011). Randomized controlled trial of tacrolimus ointment 0.1% for keratosis pilaris. *Pediatric Dermatology, 28*, 459–460. https://doi.org/10.1111/j.1525-1470.2011.01497.x
38. Tatavarthi, R., Kolalapudi, S. A., Arumilli, K. P. P., et al. (2022). Efficacy of tacrolimus 0.1% ointment in keratosis pilaris: A prospective hospital-based interventional study. *Journal of Medical Sciences, 42*, 225–229. https://doi.org/10.4103/jmedsci.jmedsci_356_20
39. Park, J., Kim, B. J., Kim, M. N., & Lee, C. K. (2011). A pilot study of Q-switched 1064-nm Nd:YAG laser treatment in keratosis pilaris. *Annals of Dermatology, 23*, 293–298. https://doi.org/10.5021/ad.2011.23.3.293
40. Clark, S. M., Mills, C. M., & Lanigan, S. W. (2000). Treatment of keratosis pilaris atrophicans with the pulsed tunable dye laser. *Journal of Cutaneous Laser Therapy, 2*, 151–156. https://doi.org/10.1080/14628830050516416
41. Kaune, K. M., Haas, E., Emmert, S., Schön, M. P., & Zutt, M. (2009). Successful treatment of severe keratosis pilaris rubra with a 595-nm pulsed dye laser. *Dermatologic Surgery, 35*, 1592–1595. https://doi.org/10.1111/j.1524-4725.2009.01282.x
42. Teerawatanapong, K., Chaichalothornkul, S., & Saiwichai, T. (2025). Efficacy of home use intense pulsed light (IPL) for the improvement of keratosis pilaris. *Procedia Multidisciplinary Research, 3*(3), Article 41. https://so09.tci-thaijo.org/index.php/PMR/article/view/6159
43. Maitriwong, P., Asawanonda, P., & Tangkijngamvong, N. (2021). Intense pulsed light significantly improves keratosis pilaris: A randomized, double-blind, sham-irradiation-controlled trial. *Lasers in Medical Science, 36*, 241–247. https://pmc.ncbi.nlm.nih.gov/articles/PMC6937144/
44. Rodríguez-Lojo, R., Pozo, J. D., Barja, J. M., Piñeyro, F., & Pérez-Varela, L. (2010). Keratosis pilaris atrophicans: Treatment with intense pulsed light in four patients. *Journal of Cosmetic and Laser Therapy, 12*, 188–190. https://doi.org/10.3109/14764172.2010.502456
45. Vachiramon, V., Anusaksathien, P., Kanokrungsee, S., & Chanprapaph, K. (2016). Fractional carbon dioxide laser for keratosis pilaris: A single-blind, randomized, comparative study. *BioMed Research International, 2016*, Article 1928540. https://doi.org/10.1155/2016/1928540
46. Ismail, S., & Omar, S. S. (2020). Clinical and dermoscopic evaluation of fractional carbon dioxide laser in management of keratosis pilaris in Egyptian-type skin. *Journal of Cosmetic Dermatology, 19*, 1110–1120. https://doi.org/10.1111/jocd.13140

47. Sobhi, R. M., Adawy, N. A. H., & Zaky, I. S. (2022). Comparative study between the efficacy of fractional CO2 laser, Q-switched Nd:YAG laser (1064 nm), and both types in treatment of keratosis pilaris. *Lasers in Medical Science, 37*, 1685–1693. https://doi.org/10.1007/s10103-020-02956-w
48. Li, M., Bai, Y., Duan, Z., et al. (2022). Efficacy and safety of long-pulsed 755-nm alexandrite laser for keratosis pilaris: A split-body randomized clinical trial. *Dermatology and Therapy, 12*, 1897–1906. https://doi.org/10.1007/s13555-022-00771-5
49. Lee, S. J., Choi, M. J., Zheng, Z., et al. (2013). Combination of 595-nm pulsed dye laser, long-pulsed 755-nm alexandrite laser, and microdermabrasion treatment for keratosis pilaris: Retrospective analysis of 26 Korean patients. *Journal of Cosmetic and Laser Therapy, 15*, 150–154. https://doi.org/10.3109/14764172.2013.769276
50. Lee, S. J., Chung, W. S., Kim, J., & Cho, S. B. (2012). Combination of 595-nm pulsed dye laser, long-pulsed 755-nm alexandrite laser and microdermabrasion treatment for keratosis pilaris. *Journal of Dermatology, 39*, 479–480. https://doi.org/10.1111/j.1346-8138.2011.01374.x
51. Ibrahim, O., Khan, M., Bolotin, D., et al. (2015). Treatment of keratosis pilaris with 810-nm diode laser: A randomized clinical trial. *JAMA Dermatology, 151*, 187–191. https://doi.org/10.1001/jamadermatol.2014.2211
52. Dawn, G., Urcelay, M., Patel, M., & Strong, A. (2002). Keratosis pilaris rubra responding to potassium titanyl phosphate laser. *British Journal of Dermatology, 147*, 822–824. https://doi.org/10.1046/j.1365-2133.2002.493010.x
53. Temiz, S. A., Dursun, R., Özer, İ., et al. (2023). Keratosis pilaris rubra treated successfully with 577-nm pro-yellow laser. *Journal of Cosmetic Dermatology, 22*, 1487–1490. https://doi.org/10.1111/jocd.15088
54. Park, K. Y., Jang, W. S., Kim, I. S., et al. (2017). Combination peel with fractional prickle coral calcium for keratosis pilaris: A single-blind randomized study. *Journal of Dermatological Treatment, 28*, 153–157. https://doi.org/10.3109/09546634.2012.671914
55. Kearney, M., Henes, E., Peterson, G., et al. (2013). Efficacy and safety of a sonic skin care brush on keratosis pilaris. *Journal of the American Academy of Dermatology, 68*(4 Suppl. 1), AB49. https://doi.org/10.1016/j.jaad.2012.12.205
56. Zirwas, M. J., & Fichtel, J. (2018). Chlorine dioxide complex cleanser: A new agent with rapid efficacy for keratosis pilaris. *Journal of Clinical and Aesthetic Dermatology, 11*, 28–30. https://pubmed.ncbi.nlm.nih.gov/29742188/
57. Lee, N. Y., Ibrahim, O., Khetarpal, S., et al. (2018). Dermal microflora restoration with ammonia-oxidizing bacteria *Nitrosomonas eutropha* in the treatment of keratosis pilaris: A randomized clinical trial. *Journal of Drugs in Dermatology, 17*, 285–288. https://pubmed.ncbi.nlm.nih.gov/29537446/
58. Lodi, G., Cannarozzo, G., Fusco, I., et al. (2025). Successful treatment of ulerythema ophryogenes with pulsed dye laser: A case report. *Clinical Case Reports, 13*, Article e70956. https://pmc.ncbi.nlm.nih.gov/articles/PMC12319604/
59. Miranda, B. H., Farjo, N., & Farjo, B. (2011). Eyebrow reconstruction in dormant keratosis pilaris atrophicans. *Journal of Plastic, Reconstructive & Aesthetic Surgery, 64*, e303–e305. https://doi.org/10.1016/j.bjps.2011.06.009
60. Wang, M. A., Wilson, A., & Murrell, D. F. (2023). A review of the scoring and assessment of keratosis pilaris. *Skin Appendage Disorders, 9*, 241–251. https://doi.org/10.1159/000529487