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Review Article

Tranexamic acid versus kojic acid in facial post-inflammatory hyperpigmentation following acne: a narrative review

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ABSTRACT

Facial post-inflammatory hyperpigmentation (PIH) following acne is a frequent and distressing sequela, especially in darker Fitzpatrick skin phototypes. Tranexamic acid (TXA) and kojic acid are increasingly used as non-hydroquinone depigmenting agents, but their comparative role in acne-induced PIH remains insufficiently defined. Objective of the study was to compare the mechanistic rationale, clinical evidence, safety profile, and practical role of TXA versus kojic acid in facial PIH after acne. A focused narrative review was performed using DOI-traceable studies and reviews on acne-induced PIH, TXA, kojic acid, and facial hyperpigmentation. Evidence from acne-specific PIH studies was prioritized, while melasma studies were used only as indirect supportive evidence because melasma and PIH differ in pathogenesis. Acne-induced PIH is driven by inflammation-mediated melanocyte stimulation, excess epidermal melanin, and, in some cases, dermal melanophages. TXA acts upstream by inhibiting plasminogen activation and reducing inflammatory and vascular mediators involved in melanogenesis. Clinical evidence for TXA in PIH includes systematic review data and a randomized trial showing topical 5% TXA to be comparable to 20% azelaic acid for acne-related PIH, with favourable early tolerability. Kojic acid acts mainly by copper chelation and tyrosinase inhibition. Its evidence in acne-PIH is more limited, with small preliminary data in post-acne hyperpigmentation and stronger indirect evidence from melasma studies, often in combination with hydroquinone, glycolic acid, or vitamin C. No robust direct head-to-head trial of TXA versus kojic acid for facial post-acne PIH was identified. Current evidence supports both agents as useful non-hydroquinone options. TXA may be preferable where inflammation, erythema, recurrence, or sensitive skin are prominent, while kojic acid may be useful for epidermal brown macular pigmentation in patients tolerating tyrosinase inhibitors. Direct comparative randomized trials are needed.

Keywords: Tranexamic acid, Kojic acid, Post-inflammatory hyperpigmentation, Acne vulgaris, Facial hyperpigmentation, Skin of colour

INTRODUCTION

Post-inflammatory hyperpigmentation (PIH) is among the most common sequelae of acne vulgaris and may persist long after inflammatory lesions have resolved. It is particularly frequent and clinically significant in patients with darker skin types, in whom even mild acne can leave cosmetically disturbing brown macules.[1-4] Acne-related PIH also has a psychosocial burden because patients may perceive the pigmentary marks as more persistent than active acne itself.[5-7]

The pathogenesis of acne-induced PIH is multifactorial. Inflammatory papules and pustules trigger cytokine release, prostaglandin activity, arachidonic acid metabolites, and melanocyte stimulation, leading to increased melanin synthesis and transfer to keratinocytes. Deeper inflammation or excoriation may cause pigmentary

incontinence with dermal melanophages, explaining why some lesions are more resistant to topical therapy.[6,8] Management must therefore address both acne control and pigmentation. Sunscreen, avoidance of picking, barrier repair, and early anti-acne therapy are fundamental.

Hydroquinone remains an established depigmenting agent but is limited by irritation, rebound pigmentation, exogenous ochronosis with misuse, and patient preference for non-hydroquinone alternatives. Tranexamic acid (TXA) and kojic acid are increasingly used in this context. TXA is a synthetic lysine analogue with anti-plasmin, anti-inflammatory, anti-angiogenic, and melanogenesis-modulating effects. Kojic acid is a fungal metabolite that inhibits tyrosinase mainly by chelating copper at the enzyme active site.[9-12] This review compares the two agents for facial PIH after acne.

METHODS

This is a narrative comparative review. Literature was selected from DOI-indexed articles addressing acne-induced PIH, TXA for PIH or hyperpigmentation, kojic acid for hyperpigmentation, and clinical consensus on acne-associated hyperpigmentation. Acne-specific PIH evidence was prioritized. Melasma studies were included only where they clarified mechanism, tolerability, or depigmenting efficacy, because melasma cannot be directly equated with post-acne PIH.

EVIDENCE FOR TRANEXAMIC ACID

TXA targets several pathways relevant to acne-induced PIH. By inhibiting plasminogen activation, TXA may reduce arachidonic acid release and downstream prostaglandin-mediated melanocyte stimulation. It may also reduce vascular and inflammatory signalling, which is relevant because acne PIH often follows active inflammation rather than purely ultraviolet-driven pigmentation.[10,13]

The most directly relevant randomized evidence is the single-blinded trial by Sobhan et al, comparing 20% azelaic acid cream with 5% TXA solution in acne-related PIH. Both treatments were effective, and TXA had a better early tolerability profile.[9] A systematic review by Alsharif et al. identified multiple routes of TXA use in PIH, including topical, oral, and intradermal approaches, but emphasized heterogeneity in study design, dose, indication, and co-interventions.[10] Case-level evidence also supports oral and topical TXA in long-standing PIH and TXA combined with low-fluence Q-switched Nd: YAG laser in resistant PIH after contact dermatitis.[11,12]

Indirect evidence from melasma trials supports the pigment-modulating potential of topical TXA. Topical 5% TXA has been compared with hydroquinone in melasma, with improvement in MASI scores and acceptable tolerability.[14,15] However, extrapolation to acne-induced PIH must be cautious because melasma has hormonal, ultraviolet, vascular, and genetic components that differ from acne inflammation.

Clinically, TXA may be attractive in patients with sensitive skin, inflammatory acne background, post-acne erythema overlap, recurrent pigmentation, or intolerance to stronger tyrosinase inhibitors. Topical TXA concentrations of 3–5% are commonly studied. Oral TXA should not be used casually for cosmetic pigmentation; it requires medical screening for thromboembolic risk, hormonal therapy use, pregnancy status, menstrual history, and personal or family clotting history.

EVIDENCE FOR KOJIC ACID

Kojic acid is a well-known tyrosinase inhibitor. By chelating copper, it reduces conversion steps in melanin synthesis and is therefore mechanistically suited to epidermal brown macular pigmentation.[17,20] It is usually used in concentrations around 1–2%, either alone or in combination with agents such as hydroquinone, glycolic acid, vitamin C, niacinamide, or retinoids.

Direct acne-PIH evidence for kojic acid is limited. A preliminary hyperspectral imaging study evaluating a kojic-acid-containing preparation included patients with post-acne skin and reported measurable reduction in hyperpigmentation parameters.[19] Older and stronger clinical data exist in melasma rather than acne PIH. Lim demonstrated that adding kojic acid to a gel containing hydroquinone and glycolic acid improved melasma outcomes.[17] Monteiro et al compared 4% hydroquinone with 0.75% kojic acid plus vitamin C in facial melasma, showing both regimens to be effective, although hydroquinone had faster onset.[18]

The main limitation of kojic acid is tolerability. Irritation, stinging, sensitization, and barrier disruption can worsen acne-related pigmentation if inflammation is provoked. This is especially relevant in patients already using retinoids, benzoyl peroxide, salicylic acid, or chemical peels. Kojic acid is therefore best introduced gradually, preferably at night, with moisturization and sunscreen.

COMPARATIVE INTERPRETATION

There is no sufficient direct evidence to declare either TXA or kojic acid superior for facial PIH after acne. Their roles are better understood as mechanistically complementary rather than mutually exclusive.

TXA acts more upstream on inflammation-linked melanogenesis and may be better suited for patients with ongoing inflammatory acne, post-acne erythema, sensitive skin, or recurrent pigmentation. Kojic acid acts more directly at the tyrosinase level and may be useful when acne is controlled and pigmentation is predominantly epidermal, brown, and macular.

For Indian and other darker skin phototypes, the practical priority is to avoid irritation. Any depigmenting agent that causes burning, peeling, or dermatitis may worsen PIH. Thus, a mild TXA-based regimen may be preferable initially in sensitive or inflamed skin, while kojic acid can be added later or used in stable lesions. Combination therapy is rational but needs controlled study, because many marketed formulations combine TXA, kojic acid, niacinamide, retinoids, or acids without factorial evidence proving the independent contribution of each ingredient.

SUGGESTED CLINICAL APPROACH

Initial management should include acne suppression, broad-spectrum sunscreen, avoidance of picking, and barrier repair. In mild-to-moderate facial PIH, topical TXA 3–5% or kojic acid 1–2% may be selected according to skin tolerance and lesion behaviour. TXA may be preferred where irritation risk is high. Kojic acid may be considered for stable epidermal macules but should be avoided or reduced if dermatitis occurs. Retinoids and azelaic acid remain important alternatives or adjuncts, especially when comedonal acne persists.

For resistant PIH, dermatologic procedures such as chemical peels, lasers, or microneedling should be used cautiously in darker skin because procedure-induced inflammation can worsen pigmentation. TXA has been explored as an adjunct in procedure-related PIH prevention and treatment, but protocols remain heterogeneous.[10,12]

FUTURE RESEARCH

A direct randomized split-face trial comparing topical 5% TXA with 2% kojic acid for 12–16 weeks in patients with facial post-acne PIH is needed. Both groups should receive identical acne control, moisturizer, and tinted broad-spectrum sunscreen. Outcomes should include melanin index, validated acne-PIH scores, investigator global assessment, patient satisfaction, time to visible improvement, relapse after discontinuation, and adverse events. Stratification by Fitzpatrick type, acne activity, and epidermal versus dermal pigmentation would improve clinical applicability.

CONCLUSION

TXA and kojic acid are both reasonable non-hydroquinone options for facial PIH after acne, but current evidence does not prove superiority of one over the other. TXA has stronger acne-PIH-specific supportive evidence and a broader anti-inflammatory rationale, while kojic acid has a long history as a tyrosinase inhibitor with mainly indirect evidence from melasma and small post-acne studies. The best current approach is individualized selection based on acne activity, pigment depth, skin sensitivity, and tolerance, with strict photoprotection and acne control as the foundation.

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