10.48047/jocaaa.2024.33.06.110

Topical Trichloroacetic Acid for Atrophic Post-Acne Scars

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ABSTRACT

Background: Acne scarring is a frequent sequel of inflammatory acne, driven by dysregulated wound healing and extracellular-matrix remodeling, with substantial psychosocial burden. Among procedural treatments, trichloroacetic acid peel particularly when delivered focally via the Chemical Reconstruction of Skin Scarring (CROSS) technique offer a cost-effective, predictable option for atrophic scars. TCA induces controlled protein coagulation from epidermis into the upper reticular dermis, promotes re- epithelialization and dermal regeneration. This review summarizes the pathogenesis and classification of acne scars, the biological and clinical effects of TCA, practical technique (including preparation, application to frosting endpoints, and post-comfort measures), advantages, and adverse effects, and highlights priorities for standardization and future research.

Conclusion

Trichloroacetic acid especially the CROSS technique, provides a practical, cost-effective option for managing atrophic acne scars with acceptable safety when applied precisely. Standardized protocols for concentration, layering, endpoints, and outcomes are now the key to improving outcomes and optimizing long-term results.

Keywords*:* acne scars, atrophic scars, TCA, chemical peel, CROSS technique, cytokines, growth factors, dyspigmentation

Introduction

Acne scars are a common dermatological concern that can persist long after active acne has been resolved. These scars develop as a result of inflammation within the dermis, often due to the body's attempt to repair damage caused by severe or prolonged acne. The severity of acne scarring typically correlates with the intensity and duration of acne lesions, particularly cystic and nodular forms [1].

The pathogenesis of acne scars involves an intricate interplay between inflammatory mediators, immune responses, and the skin's wound healing mechanisms. When acne lesions become inflamed, there is a breakdown of follicular walls and dermal structures. This leads to a disorganized healing response, which can culminate in either tissue loss or excess tissue formation [2]

Acne scars are generally categorized into two main types: atrophic and hypertrophic. Atrophic scars, which are more common, result from a net loss of collagen and include icepick, boxcar, and rolling scars. Each subtype has distinct clinical features based on the depth, width, and morphology of the scar [3]. Icepick scars are narrow, deep, and tapering punctate depressions that extend into the dermis. They are the result of deep follicular inflammation and are often considered the most resistant type of scar to improve naturally over time. Icepick scars account for a significant proportion of acne-related atrophic scarring [4].

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Boxcar scars, in contrast, are wider than icepick scars and have well-defined vertical edges. These scars are caused by inflammatory processes that destroy collagen, resulting in broad, crater-like depressions in the skin. They are commonly found on the cheeks and temples [5].

Rolling scars are characterized by shallow, wide depressions with sloping edges. These scars are caused by fibrous bands of tissue pulling on the epidermis, creating an undulating skin surface. Rolling scars typically develop as a result of long-standing inflammatory acne [6].

Hypertrophic scars and keloids, although less common than atrophic scars, represent an overproduction of collagen during the healing process. Hypertrophic scars remain within the boundaries of the original lesion, whereas keloids can extend beyond the original wound margins, often becoming raised and firm [7].

Several risk factors increase the likelihood of developing acne scars. These include the severity and duration of acne, genetic predisposition, delayed treatment, and manipulation or picking of acne lesions. Male gender and darker skin phototypes have also been associated with a higher risk of certain types of scarring [8].

Inflammation is the key driver of acne scar formation. The longer a lesion remains inflamed, the greater the disruption to skin architecture. This prolonged inflammation results in the degradation of extracellular matrix components, particularly collagen and elastin fibers, which are critical for skin integrity [9].The inflammatory process in acne not only involves neutrophils and lymphocytes but also an increase in cytokines such as interleukin-1 and tumor necrosis factor-alpha. These factors contribute to the recruitment of additional immune cells and the subsequent remodeling or destruction of the dermis, leading to scar formation [10].
Acne scars can have profound psychological and social implications. Individuals with visible scars often experience decreased self-esteem, social withdrawal, anxiety, and even depression. These effects are especially pronounced during adolescence and early adulthood, which are critical periods for self- image development [11].

The distribution of acne scars often corresponds with areas prone to severe acne outbreaks. Commonly affected sites include the cheeks, forehead, chin, and back. The visibility of facial scars makes them particularly distressing for many individuals [12].

Histologically, atrophic acne scars show epidermal thinning and loss of dermal appendages. There is often a reduction in dermal collagen and elastin, accompanied by fibroblast inactivity. These histopathologic features reflect the underlying structural damage that perpetuates the scarred appearance [13].The role of matrix metalloproteinases (MMPs) in scar formation is well established. MMPs are enzymes that degrade collagen and other components of the extracellular matrix. An imbalance between MMPs and their inhibitors during acne lesion resolution contributes significantly to dermal remodeling and scar formation [14].Fibroblasts play a dual role in wound healing and scarring. In acne scars, fibroblast function becomes dysregulated, leading to inadequate or excessive collagen deposition. The phenotype of fibroblasts in scar tissue differs from that of normal skin, influencing the final scar morphology [15].

Genetic and ethnic factors also influence scar formation. Certain populations, particularly individuals of African, Hispanic, or Asian descent, may have a higher tendency to develop keloid or hypertrophic scars due to genetic differences in wound healing responses and collagen synthesis [16].

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The location and depth of the original acne lesions determine the resultant scar type. Deep nodulocystic acne is more likely to lead to atrophic scars due to the intense and prolonged inflammatory response. Superficial acne lesions, such as comedones or papules, rarely result in permanent scarring [17].

Scarring is more prevalent in individuals who delay or avoid treatment for active acne. Early control of inflammation is crucial in preventing the destruction of dermal structures that leads to scar formation. However, many individuals are unaware of the long-term consequences of untreated acne [18].

Despite being a non-life-threatening condition, acne scarring can significantly impact quality of life. Many sufferers report embarrassment, low confidence, and difficulties in professional and social interactions. The psychosocial burden can be as debilitating as that associated with chronic physical conditions [19].

TCA as a Therapeutic Modality Chemistry

TCA is a chlorinated acetic acid derivative (three hydrogens on the methyl group replaced by chlorine) that produces **coagulative necrosis** of cellular and extracellular proteins on contact. Penetration depth depends on factors such as the number of application layers and whether it's combined with enhancers (eg, croton oil, Jessner's solution) or agents that limit penetration [20].

Mechanism of action.

Among chemical peels, TCA is notably versatile. It denatures epidermal and dermal proteins and, at higher strengths, can induce collagen necrosis reaching the upper reticular dermis [21]. Clinically, improvements result from increased dermal volume via stimulated production of collagen, glycosaminoglycans, and elastin [21]. Concentrations typically range from 10%–50%; higher concentrations cause more robust ablation and correspondingly greater rejuvenation effects [22].

Peels stronger than 30% extend into the dermis, classifying them as medium to deep. Historically, 40%–60% TCA was used for acne scarring, but these strengths were associated with higher risks—particularly scarring and post-inflammatory hyperpigmentation—so they are no longer recommended in practice [21].

Cellular signaling.

TCA exposure in epidermal cells triggers a surge in growth factors and cytokines—PDGF-B, TGF-α1, TGF-β1, VEGF, IL-1, and IL-10. PDGF rises promptly within keratinocyte cytoplasm after application but returns to baseline by ~24 hours, coinciding with complete epidermal necrosis [23]. The pre-necrosis release of these mediators is pivotal for re-epithelialization and dermal remodeling—the hallmark outcomes of TCA peels. In fibroblasts, the dominant response is upregulated synthesis of keratinocyte-derived growth factor (KGF), further supporting repair and regeneration [23].

CROSS technique.

Trichloroacetic acid (TCA) is currently the workhorse agent for atrophic acne scars treated by CROSS [24]. Clinical data show that higher TCA concentrations generally track with greater scar improvement; nevertheless, multiple independent studies report that a broad span of concentrations including comparatively lower strengths such as 50% can achieve meaningful cosmetic gains with fewer adverse effects [25].

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Key properties of TCA.

TCA does not require external neutralization because it causes irreversible protein coagulation that self-limits penetration and prevents systemic absorption. At strengths used for superficial and medium peels, TCA self-neutralizes rapidly, seen clinically as progressive "frosting." In superficial peels this appears as a pseudofrost; at >50%, neutralization slows, increasing the chance of deeper injury and complications [26].

How to use:

Before applying TCA, the skin is thoroughly cleansed and decreased with alcohol or acetone to ensure even penetration of the solution. Topical anesthesia is avoided, as it can cause vasoconstriction, which may interfere with the concentration and effectiveness of TCA. The application is performed using cotton buds or gauze swabs in a sequential manner until the desired level of frosting is achieved. To soothe the burning sensation, vaporized water or moisturizing cream can be used [27].

Advantages

TCA solutions are inexpensive, easy to prepare, chemically stable with long shelf-life, and lack systemic toxicity. These properties, combined with versatility across depth targets, make TCA a pragmatic moderate-to-deep peeling agent for scar rejuvenation [26].

Adverse Effects and Safety

- Edema: mild–moderate swelling is common for 2–3 days.
- Discomfort: usually minimal; simple analgesia if needed.
- Erythema: pink/red discoloration for 2–8 weeks (occasionally up to 6 months).
- Photosensitivity: common for ~2–8 weeks; counsel photoprotection.
- Pigmentary shifts**:** both hyper- and hypopigmentation may occur.
- "Hot spots"**:** localized deeper penetration with higher risk of hyperpigmentation; less frequent when lower concentrations are used [21]

Conclusion

TCA is a cornerstone intervention for **atrophic acne scars**, having both a clear mechanistic basis (controlled coagulation) and practical advantages (low cost, stability, self-neutralization, lack of systemic toxicity). Evidence supports **dose-responsive improvement** across a spectrum of strengths; careful selection of concentration, good skin preparation, **precise focal application to frosting endpoints**, and excellent post-procedure care collectively optimize outcomes while limiting complications. Further standardization across concentration, studies in different phototypes will refine protocols and enhance reproducibility. Until then, TCA CROSS remains a **high-value,**

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technique-dependent solution for elevating atrophic scar depressions and improving patient quality of life.

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