

Restoring Skin Quality in Acne Vulgaris Using Fractional Non-Ablative Laser Resurfacing



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Abstract

Acne vulgaris is a chronic inflammatory disease of the pilosebaceous unit and remains one of the most prevalent dermatological conditions worldwide. It can result in persistent erythema, post-inflammatory hyperpigmentation, uneven skin texture, and scarring, contributing to considerable cosmetic and psychosocial burden. A 27-year-old female presented with persistent acne predominantly involving the right cheek, associated with facial erythema, post-inflammatory hyperpigmentation, mild atrophic scarring, and significant cosmetic concern. The patient reported recurrent inflammatory lesions despite conventional topical therapy. Clinical examination revealed multiple inflammatory papules and pustules on an erythematous background, accompanied by diffuse hyperpigmented macules and uneven skin texture. The patient underwent three sessions of 1565 nm fractional non-ablative laser therapy, with parameters

individualized according to skin type and standard pre- and post-procedure care. Following treatment, marked clinical improvement was observed, including reduction in inflammatory acne lesions and background erythema, lightening of post-inflammatory hyperpigmentation, and improvement in overall skin texture and appearance. Treatment was well tolerated, with minimal discomfort and no observed post-procedure hyperpigmentation, prolonged erythema, or scarring. The patient expressed high satisfaction with the outcome, including improved confidence and quality of life. Fractional non-ablative laser resurfacing may provide a minimally invasive treatment option for selected patients with acne vulgaris and associated post-acne skin changes.

Keywords: Acne vulgaris; Fractional non-ablative laser; Post-inflammatory hyperpigmentation; Acne scarring; Laser resurfacing; Skin texture

Introduction

Acne vulgaris is a chronic inflammatory disease of the pilosebaceous unit and remains one of the most prevalent dermatological conditions worldwide. It affects individuals across all age groups, most commonly manifesting during adolescence and frequently persisting into adulthood, particularly among women. Owing to its high prevalence, relapsing nature, and potential to cause permanent scarring, acne represents a substantial clinical and psychosocial burden. Clinically, acne is characterized by a polymorphic eruption consisting of non-inflammatory lesions such as open and closed comedones, along with inflammatory papules, pustules, nodules, and cysts. These lesions predominantly involve sebaceous-rich areas, including the face, neck, chest, and upper back. Disease severity ranges from mild comedonal acne to severe nodulocystic or scarring forms. The etiopathogenesis of acne is complex and multifactorial, involving abnormal follicular keratinization, androgen-mediated sebaceous hypersecretion, colonization by *Cutibacterium acnes*, and subsequent immune-mediated inflammation. The process begins with microcomedo formation due to follicular obstruction by keratinocytes and sebum. Increased androgen activity during puberty stimulates sebaceous gland enlargement and sebum production, facilitating follicular rupture. *C. acnes* contributes to inflammation through lipase-mediated breakdown of sebum, release of pro-inflammatory

mediators, and activation of both innate and adaptive immune responses. Genetic predisposition, hormonal fluctuations, diet, stress, environmental factors, cosmetic use, and medications further modulate disease expression and severity. Clinically, acne exhibits diverse variants ranging from self-limiting neonatal forms to severe presentations such as acne conglobata and acne fulminans, as well as mechanically, chemically, and drug-induced acne.^{1,2}

One of the most significant and enduring sequelae of acne vulgaris is acne scarring, resulting from sustained inflammation and dysregulated wound healing. Scars are classified as atrophic or hypertrophic, with atrophic scars being most common. Atrophic scars include icepick scars, narrow, deep, V-shaped tracts; boxcar scars, round to oval depressions with vertical edges; and rolling scars, broad, shallow depressions from fibrous dermal tethering. Hypertrophic and keloidal scars, less frequent, result from excessive collagen deposition and are more common in predisposed individuals.³

The development of acne scars is closely linked to the inflammatory pathophysiology of acne vulgaris. Persistent perifollicular inflammation, driven by follicular hyperkeratinization, increased androgen-mediated sebum production, and *C. acnes* colonization, can compromise the follicular wall, causing rupture and dermal spread of inflammatory mediators and keratinous debris. This triggers the wound-healing cascade,

normally progressing through inflammation, granulation, and matrix remodeling. In acne, dysregulated inflammation disrupts repair, with fibroblasts initially producing type III collagen, later replaced by type I collagen. Balance between matrix metalloproteinases (MMPs) and tissue inhibitors (TIMPs) is critical; disruption leads to either excessive collagen breakdown, causing atrophic scars, or overproduction, resulting in hypertrophic or keloidal scars. Scar severity and morphology are determined by the depth, intensity, and duration of inflammation. Beyond structural effects, acne and scarring impose a significant psychosocial burden, including reduced self-esteem, impaired body image, social withdrawal, anxiety, and depression, especially when occurring during adolescence, with long-lasting psychological impact even after lesion resolution.^{2,3}

Collectively, the high prevalence, chronic inflammation, diverse clinical spectrum, and risk of permanent scarring emphasize the need for early recognition, timely treatment, and comprehensive management to control active acne, limit inflammation, and prevent long-term physical and psychological morbidity.

Case Report

A 27-year-old female presented with persistent acne predominantly involving the right cheek, associated with facial erythema, post-inflammatory hyperpigmentation, and mild atrophic scarring, resulting in significant cosmetic concern. The patient reported recurrent inflammatory lesions despite

conventional topical therapy. There was no history of systemic illness, hormonal disorder, or recent isotretinoin use. On clinical examination, the right cheek showed multiple inflammatory papules and pustules on an erythematous background, accompanied by diffuse hyperpigmented macules. The surrounding skin demonstrated uneven texture and tone, consistent with post-inflammatory changes secondary to acne. No nodulocystic lesions were noted at the time of presentation. The patient was treated with three sessions of 1565 nm fractional non-ablative laser therapy. Parameters were individualized to the patient's skin type, with standard pre- and post-procedure care, including strict photoprotection and gentle skincare. At follow-up evaluation after completion of the laser sessions, the patient demonstrated marked clinical improvement. There was a significant reduction in inflammatory acne lesions, with visible decrease in background erythema. Post-inflammatory hyperpigmentation showed notable lightening, resulting in a more even skin tone and improved overall facial appearance. The skin texture appeared smoother, with enhanced clarity and radiance. The treatment was well tolerated, and the patient reported minimal discomfort during procedures. No adverse effects such as post-procedure hyperpigmentation, prolonged erythema, or scarring were observed. The patient expressed high satisfaction with the outcome, reporting improved confidence and quality of life.



Before treatment



After treatment

Figure 1: Significant improvement in inflammatory acne, background erythema, post-inflammatory hyperpigmentation, and scarring following fractional non-ablative laser therapy, with smoother skin texture and a more uniform skin tone

Treatment

Fractional non-ablative laser resurfacing (NFSR) is a minimally invasive dermatologic technique that delivers focused thermal energy into the dermis to enhance surface texture, even out pigmentation, and reduce the appearance of scars while preserving the integrity of the superficial skin layers. It delivers energy in a fractionated microthermal pattern, creating controlled dermal injury while preserving the epidermis, thereby stimulating neocollagenesis and dermal remodeling. Compared with traditional ablative lasers, NFSR reduces the risk of erythema, dyspigmentation, infection, and scarring, offering a safer approach for gradual skin rejuvenation.^{3,12,13}

Patient Selection

Careful patient selection and preparation are essential to optimize outcomes and minimize complications. Indications include active acne, acne scars, facial and extra-facial rhytides, photodamage, surgical or traumatic scars, and melasma. Contraindications such as active or recurrent herpes simplex infection should be identified, as these may reactivate following fractional photothermolysis. Gender-related differences may influence recovery, with males typically experiencing shorter durations of erythema and edema due to thicker dermal and epidermal layers and larger sebaceous glands.^{3,12,13}

Pre-Treatment Preparation

A thorough consultation is essential to discuss patient expectations, outline an individualized treatment plan, and review potential side effects. Pre-treatment may

include topical tretinoin, started 4–6 weeks before therapy, discontinued 5–7 days prior, and resumed five days post-procedure. Pain management is typically achieved with topical anaesthetics, as local or systemic anaesthesia is rarely required.^{3,12,13}

Laser Parameters

NFSR targets tissue water to create microthermal zones (MTZs) of less than 400 microns, preserving surrounding tissue for rapid healing. Commonly used wavelengths for acne scar treatment include 1440 nm, 1540 nm, and 1550 nm. These wavelengths penetrate the dermis to stimulate neocollagenesis and dermal remodeling without ablating the stratum corneum.^{3,12,13}

Treatment Technique

The laser is applied systematically over the treatment area in a grid-like or stamped pattern. Only a fraction of the skin is treated during each session, allowing untreated tissue to aid rapid re-epithelialization. Multiple sessions are required to cover the entire scarred area, typically 4–6 sessions with an interval of 7–10 days between each session.^{3,12,13}

Safety and Efficacy

NFSR and adjunctive therapies demonstrate a favourable safety profile. Common short-term effects include erythema, edema, dryness, flaking, and transient bronzing. Serious complications are rare but may include isolated blistering, transient hyperpigmentation, or petechiae. Permanent scarring or pigmentary changes are uncommon; however, post-

inflammatory hyperpigmentation is more likely in darker skin types. Histologic studies show effective epidermal healing within 24 hours, with neocollagenesis and dermal remodeling evident several months post-treatment. Multiple clinical studies confirm consistent improvement in acne scars, photodamage, rhytides, and skin laxity.^{3,12,13}

Post-Procedure Care

Following NFSR, patients should maintain strict photoprotection and adhere to recommended skincare regimens to support healing and minimize complications. Topical tretinoin is typically resumed five days post-procedure. Gentle cleansing and regular moisturizing are advised. Patients should monitor for prolonged erythema, unusual pigmentation, or signs of infection, and seek prompt evaluation if these occur. Most patients can resume normal daily activities immediately, with minimal downtime.

Other therapies for acne vulgaris and post-acne sequelae include topical agents such as clindamycin with benzoyl peroxide, retinoids, niacinamide, salicylic acid, mandelic acid, azelaic acid, and dapsone to reduce *C. acnes*, inflammation, sebum, and hyperkeratinization; chemical peels for epidermal renewal and dermal remodeling; systemic treatments like isotretinoin and oral antibiotics for severe or refractory cases; hormonal therapy in females with oral contraceptives or anti-androgens; and supportive care with non-comedogenic moisturizers and sunscreen.¹

Treatments for acne scars use energy-based therapies

such as vascular lasers, IPL, and fractional ablative or non-ablative lasers, minimally invasive procedures like microneedling, radiofrequency-assisted microneedling, subcision, chemical reconstruction with TCA peels, surgical approaches including punch excision, punch elevation, dermabrasion, and soft-tissue augmentation with fillers or autologous fat.¹⁴ Combining these modalities addresses multiple skin layers, improves texture, contour, and overall appearance, and optimizes outcomes.

Discussion

Management of acne and its sequelae requires consideration of active inflammation as well as persistent erythema, post-inflammatory hyperpigmentation, textural irregularity, and scarring. The coexistence of these features may require individualized treatment approaches directed toward different clinical components.

Acne vulgaris and post-acne scarring can be assessed using standardized clinical and patient-reported instruments. Acne severity is quantified with the Global Acne Grading System (GAGS), which evaluates lesion type and anatomical distribution, and the Investigator Global Assessment (IGA), an ordinal clinician-reported scale based on lesion number, type, and intensity. Patient-reported outcomes such as the Dermatology Life Quality Index (DLQI) and Cardiff Acne Disability Index (CADi) measure psychosocial and functional impact. Scar severity is evaluated with validated systems including the Goodman

and Baron scales, Global Acne Scar Severity Score, and ECCA scale.^{4,5,6} Hormonal and laboratory investigations may be considered in severe, persistent, or atypical cases to identify underlying endocrine disorders such as polycystic ovary syndrome or adrenal hyperplasia.⁷

Adjunctive imaging enhances diagnostic precision in acne scarring. Dermoscopy allows detailed visualization of surface texture, pigmentation, and scar margins.⁸ Three-dimensional photography quantifies scar volume, depth, and topography for accurate assessment.⁹ Optical coherence tomography evaluates dermal architecture, scar thickness, and fibrotic bands in vivo.¹⁰ High-frequency ultrasonography provides real-time measurement of scar depth and tissue density.¹⁰ Reflectance confocal microscopy enables cellular-level assessment of collagen organization and fibrosis.¹¹ This structured multimodal approach provides an objective, reproducible, and holistic evaluation of acne vulgaris and post-acne scarring, supporting accurate diagnosis, documentation, and outcome monitoring.

Management of post-acne sequelae and scars is multimodal and individualized according to scar type, depth, dermal involvement, skin phototype, and patient-specific aesthetic goals. Vascular-targeted lasers and intense pulsed light may be used to decrease erythema and correct abnormal blood vessels, while ablative and non-ablative fractional lasers facilitate collagen remodeling, improve dermal structure, and enhance

epidermal regeneration. Minimally invasive procedures, including microneedling, radiofrequency-assisted microneedling, and subcision, promote collagen formation and release tethered fibrotic bands. Chemical reconstruction with high-concentration trichloroacetic acid peels may smooth surface irregularities, while surgical techniques such as punch excision, punch elevation, and dermabrasion may be used for sharply defined or deep scars. Soft-tissue augmentation with dermal fillers or autologous fat grafting may restore volume and improve skin contour. Strategic combination of these therapies may provide complementary effects across the dermis and epidermis.¹

Fractional non-ablative resurfacing provides controlled dermal heating while preserving the epidermis and surrounding tissue. This stimulates collagen synthesis and dermal remodeling while allowing treatment with relatively minimal downtime. NFSR may be combined with adjunctive technologies such as Nd:YAG 1,064 nm lasers, diode 1,450 nm lasers, bipolar radiofrequency, and intense pulsed light to address pigmentation, vascular changes, skin laxity, and other components of post-acne skin changes. Emerging regenerative approaches, including platelet-rich plasma and microneedling, may further support collagen production, angiogenesis, and tissue regeneration.³

Fractional non-ablative laser treatment was selected for active inflammatory acne accompanied by persistent erythema, post-inflammatory

hyperpigmentation, and mild atrophic scarring. The controlled delivery of thermal energy while preserving surrounding epidermal tissue makes the modality a minimally invasive option when improvement in multiple post-acne skin changes is desired.^{3,12,13}

The clinical improvement observed following three treatment sessions supports the potential utility of fractional non-ablative resurfacing in appropriately selected patients. Improvement was observed in inflammatory lesions, background erythema, post-inflammatory hyperpigmentation, and overall skin texture. However, the outcome of a single case cannot establish comparative efficacy or superiority over other therapeutic modalities.

Prevention of acne scarring remains an important component of management and depends on timely control of active acne, treatment adherence, appropriate skincare, avoidance of comedogenic products, and photoprotection. The psychological impact of acne and its sequelae should also be considered when establishing treatment goals.

Conclusion

Fractional non-ablative laser resurfacing provided favorable clinical improvement in this case of acne vulgaris with associated erythema, post-inflammatory hyperpigmentation, and mild atrophic scarring. The modality may be considered as part of an individualized treatment approach when improvement across multiple post-acne skin

parameters is desired with minimal downtime. Appropriate patient selection, treatment planning, photoprotection, and consideration of complementary modalities remain important for achieving optimal outcomes.

Declarations

Patient Consent

Written informed consent was

obtained from the patient for publication of the case details and accompanying clinical images.

Conflict of Interest

The author declares no conflict of interest.

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Ethics Approval

Where applicable.

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