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REVIEW ARTICLE

Microneedling and Adapalene-Thread Therapy for Striae Distensae: Review Article

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ABSTRACT

Striae distensae (SD), commonly known as stretch marks, are a prevalent dermatological concern affecting 40–70% of the population, particularly in women. SD manifests as two forms: striae rubrae (acute, erythematous, slightly raised lesions) and striae albae (chronic, atrophic, hypopigmented lesions). These marks commonly appear on the abdomen, breasts, thighs, and buttocks due to skin stretching, often associated with pregnancy, puberty, rapid weight changes, and certain medical conditions like Cushing's syndrome or chronic steroid use. SD results from dermal damage characterized by fibroblast dysfunction, collagen and elastin rupture, and inflammation. Hormonal factors, including elevated estrogen, glucocorticoids, and genetic predisposition, also play a role in SD development. Although SD is not a medical emergency, it can cause psychological distress due to its visible appearance. Treatments for SD are diverse, including topical agents like tretinoin and hyaluronic acid, procedural therapies such as laser therapy (fractional CO2, erbium, pulsed dye, and excimer lasers), microneedling, radiofrequency, and platelet-rich plasma (PRP). Microneedling, a collagen induction technique, creates controlled skin injuries to stimulate dermal remodeling, improving SD appearance. Adapalene, a third-generation retinoid, is also used due to its anti-inflammatory and collagen-promoting effects.

Keywords: Striae distensae, Microneedling, Topical retinoids, Adapalene.

INTRODUCTION

Striae distensae (SD), or stretch marks, afflict 80% of people. Striae rubra are erythematous, stretched lesions, while striae alba are atrophic, wrinkled, and hypopigmented (1, 2). SD, a common dermatological disorder, which challenges doctors with esthetic and psychological issues. These markings emerge opposite skin tension lines on the belly in areas of highest strain (3). They may expand to the breasts, lower back, buttocks, thighs, upper arms, axillae, and inguinal areas with pruritus (4).

Collagen, elastin, and vascular disorders make SD seem like scars, making therapy difficult. Their widespread non-facial involvement and variance between early erythematous striae rubra and later hypopigmented striae alba complicate matters (5). Despite several treatments, no cure has been found. Fibroblast activity, collagen and fibronectin production, anti-inflammatory effects, skin elasticity, and dermal thickness are improved by current treatments to improve hydration and blood perfusion (6-8). Topical medicines, laser irradiation, and other energy-based therapies

have been tested (5). Orentreich and Orentreich created microneedling in 1995 for scar therapy. Camirand's tattoo gun approach in 1997, followed by Liebl in 2000 and Fernandes in 2006, who advanced the approach for percutaneous collagen induction.

Third-generation retinoid adapalene (ADP) may also help. Vitamin A was discovered in 1909, and retinoids comprise natural and synthetic chemicals (9). Microdermabrasion, laser and light therapy, needling, RF devices, PRP injections, and adapalene have varied results (10). Clinical efficacy and patient satisfaction rankings for these medicines are unclear. Furthermore, recent cosmetic medicine advances require updated comparative research for better clinical decision-making.

Striae Distensae (SD)

Striae distensae (SD), or stretch marks, are a common dermatological concern. While not a medical emergency, they can cause psychological distress and impact quality of life. SD results from excessive skin stretching, leading to dermal damage and epidermal thinning, manifesting as linear atrophic scars (11).

a. Classification

SD occurs in two forms:

i. Striae rubrae (acute phase): Erythematous, red, slightly raised, sometimes symptomatic lesions.

ii. Striae albae (chronic phase): Atrophic, wrinkled, hypopigmented lesions (12).

b. Epidemiology

SD affects 40-70% of the population, predominantly female adolescents. Pregnant women have the highest incidence (up to 90% with striae gravidarum). Severity varies by ethnicity, with Black African women more affected than Caucasians (13).

c. Etiology

SD is linked to:

i. Pregnancy, puberty, rapid weight changes, obesity.

ii. Cushing’s syndrome, chronic steroid use, Marfan & Ehlers-Danlos syndromes (3).

iii. Anorexia nervosa, tuberculosis, typhoid, chronic liver disease (8).

iv. Corticosteroids and antiretroviral drugs (14).

d. Pathogenesis

SD formation is multifactorial

i. Dermal and Connective Tissue Changes: Over-stretching causes dermal tears, inflammation, fibroblast dysfunction, and collagen/elastin rupture (15).

ii. Genetic Factors: Secretoglobulins and keratin downregulation weaken skin integrity (16).

iii. Hormonal Factors: Elevated estrogen, androgens, and glucocorticoids suppress fibroblasts, reducing collagen/elastin synthesis. Relaxin further contributes to pregnancy-related SD (17).

iv. Vitamin Deficiency: Low vitamin D levels impair fibroblast activity (Fig 1) (18).

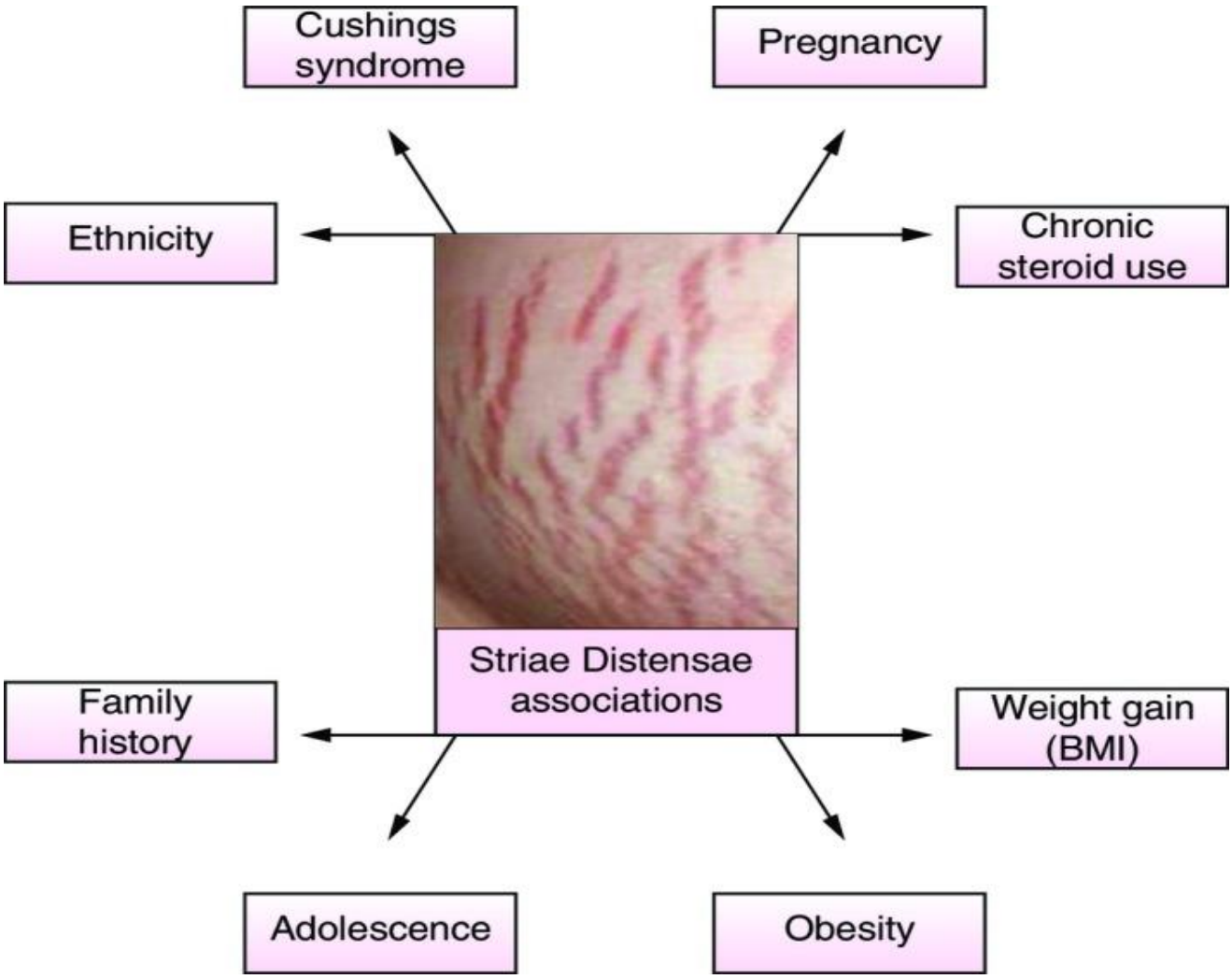

Fig (1): Factors associated with striae distensae (16).

e. Clinical Presentation

i. Striae rubrae: Flattened, pink, pruritic lesions that enlarge into reddish-purple streaks.

ii. Striae albae: Depressed, hypopigmented, irregularly shaped lesions (19).
Lesions can reach 25 cm in length and 1-10 mm in width, commonly affecting the abdomen, breasts, buttocks, and thighs (Fig 2) (20).

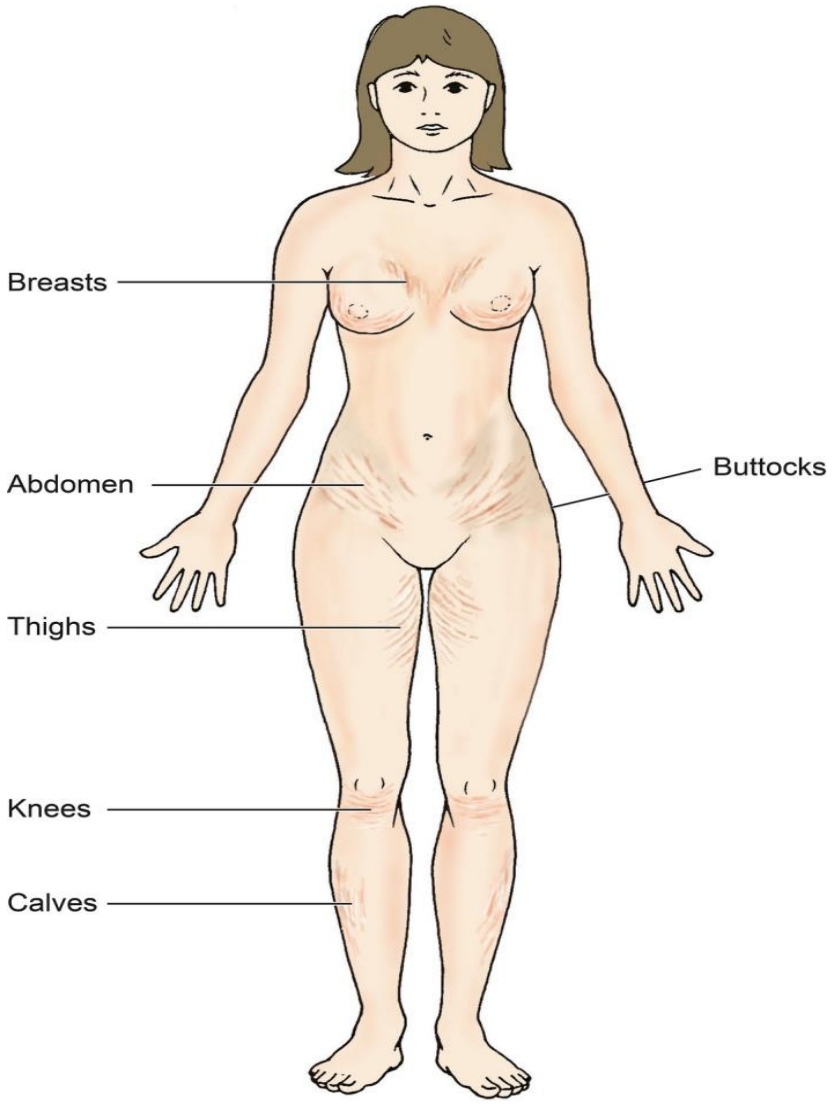

Fig (2): Illustration demonstrating the common anatomical locations affected by striae distensae (16).

f. Differential Diagnosis

i. Child abuse (misdiagnosed SD).
ii. Linear focal elastosis (asymptomatic, yellow, striae-like lines) (21).

g. Histologic Findings

SD features mid-dermis elastolysis, increased glycosaminoglycans, inflammatory infiltration, vascular changes, and epidermal atrophy. Striae rubrae show dense collagen fibers with reduced elasticity; striae albae resemble mature scars (22).

h. Treatment

i. Prevention: Avoiding rapid weight fluctuations, though effectiveness is debated (12).
ii. Topical Treatments:
   - Tretinoin (0.1%) boosts fibroblast activity but may cause irritation.
   - Hyaluronic acid enhances hydration and tensile strength.
   - Centella Asiatica counteracts glucocorticoids (useful for SG).
   - Glycolic/trichloroacetic acid peels stimulate fibroblasts (risk of hyperpigmentation in darker skin).
   - Pirfenidone promotes collagenase activity (needs further study) (23, 24).
iii. Procedural Therapies:
   - **Laser Therapy:**

- Fractional CO2 lasers improve mature SD.
- Erbium lasers stimulate collagen synthesis.
- Pulsed dye lasers reduce erythema in striae rubrae.
- Excimer lasers induce melanocyte activity (temporary repigmentation) (20, 25).

- **Microneedling & PRP:** Stimulate collagen/elastin, enhanced by fractional lasers (26, 27).
- **Radiofrequency (RF) Therapy:** Microneedle RF remodels collagen with minimal side effects.
- **Carboxy therapy:** CO2 injection boosts blood flow & collagen, though may cause bruising.

Despite various treatments, no single approach guarantees complete SD resolution. Ongoing research aims to establish standardized protocols and optimize patient satisfaction.

Topical Retinoids:

a. Overview and Classification

Topical retinoids have been widely used in dermatology for decades, treating conditions such as acne vulgaris, psoriasis, photoaging, cutaneous T-cell lymphoma, and Kaposi's sarcoma. Off-label uses include keratosis pilaris and hyperpigmentation (28). Retinoids are categorized into four generations based on molecular structure and receptor selectivity. Six topical retinoid classes exist: tretinoin (all-trans retinoic acid), adapalene, tazarotene, trifarotene, alitretinoin, and bexarotene. The last two are rarely used and often require compounding (29).

b. Mechanism of Action

Retinoids bind to and activate retinoic acid receptors, leading to gene transcription and influencing cell proliferation and differentiation. They regulate skin cell turnover, normalize abnormal desquamation, and prevent clogged pores (30). Additionally, retinoids inhibit collagen breakdown and promote collagen synthesis, improving skin texture and reducing signs of aging (31).

c. Clinical Applications and Generational Classification

The choice of a topical retinoid depends on the skin condition, patient characteristics, and clinician preference (32).

i. *First-Generation Retinoids:* Tretinoin is used for acne vulgaris and photoaging. Off-label uses include keratosis pilaris, actinic keratosis, and hyperpigmentation. Available in cream and gel formulations, tretinoin is cost-effective but irritating and photolabile. Microsphere technology (Retin-A Micro 0.04%, 0.1%) improves photostability and reduces irritation. It is also combined with clindamycin for acne treatment (33).

ii. *Second-Generation Retinoids:* No topical formulations exist (33).

iii. *Third-Generation Retinoids:* Tazarotene (0.05%, 0.1% cream/gel) treats acne and plaque psoriasis. A lotion combining tazarotene with halobetasol is also available (34). Adapalene (0.1% and 0.3% formulations) is used for acne, hyperpigmentation, and actinic keratosis. It is the least irritating and photodegradable, allowing daytime application. It is available over-the-counter (OTC) in the U.S. and is combined with benzoyl peroxide for acne (35).

iv. *Fourth-Generation Retinoids:* Trifarotene, a selective RAR agonist, treats acne on the face and trunk. It has minimal systemic absorption, as studies in patients aged ≥18 years and pediatric patients (9–17 years) showed unquantifiable systemic levels (36).

Adapalene:

a. Pharmacology and Applications

FDA-approved in 1996 for acne, adapalene is used off-label for conditions such as melasma, alopecia areata, and photoaging. Studies suggest potential antimicrobial, anticancer, and neuroprotective effects (37). Adapalene is

a stable third-generation retinoid with a 20-fold greater affinity for RAR-gamma than earlier retinoids (Kassir et al. 2020). It reduces microcomedone formation and inflammation by modulating immune responses and inhibiting Propionibacterium acnes (38). Meta-analyses suggest adapalene 0.1% gel has similar efficacy but better tolerability than tretinoin 0.025% gel. A 12-week study comparing adapalene 0.3% gel with tazarotene 0.1% gel showed a 61% reduction in acne lesions with adapalene versus 57% with tazarotene, with less irritation in the adapalene group (39). Combination therapy with benzoyl peroxide enhances efficacy (40).

b. Mechanism of Action and Pharmacokinetics

Adapalene binds RAR-beta and RAR-gamma, regulating keratinocyte differentiation and decreasing microcomedone formation. It suppresses inflammation by inhibiting toll-like receptor II and neutrophil chemotaxis (41). Systemic absorption is minimal; adapalene is metabolized primarily in the liver, with an elimination half-life of 7–51 hours (42).

c. Administration and Considerations

Adapalene is applied once daily to clean, dry skin. Moisturizers and sunscreen are recommended to mitigate irritation and photosensitivity. Initial irritation peaks within two weeks, often leading to discontinuation. Strategies to reduce irritation include every-other-day application, short-contact therapy, or alternative drug vehicles (43). Studies show no difference in efficacy among various application regimens (40).

d. Use in Specific Populations

No specific data exists for adapalene use in renal or hepatic impairment. Adapalene is classified as pregnancy category C due to potential teratogenicity. Although systemic absorption is minimal, retinoids should be avoided during pregnancy due to risks associated with vitamin A excess (44).

Microneedling:

a. Overview

Microneedling, also known as collagen induction therapy, is a minimally invasive dermatological procedure that uses instruments equipped with fine needles to create controlled micro-injuries in the dermis. These micro-injuries stimulate the natural wound healing process, leading to increased collagen and elastin production, which enhances skin remodeling (45). Initially developed for skin rejuvenation, microneedling is now widely used for treating scars, alopecia, drug delivery, hyperhidrosis, and stretch marks. When combined with radiofrequency energy, it can further enhance dermal remodeling. This procedure is considered safe, cost-effective, and well-tolerated with minimal downtime. However, despite its popularity, strong clinical evidence supporting its efficacy remains limited (46).

b. Mechanism of Action

Microneedling induces controlled skin injury without significantly damaging the epidermis. The process triggers a wound healing cascade, releasing platelet-derived growth factor, transforming growth factor α and β, fibroblast growth factor, and connective tissue growth factors (47). When used for scar treatment, microneedling disrupts scar tissue, promotes revascularization, and stimulates fibroblast migration and proliferation. A fibronectin matrix forms within five days, guiding collagen deposition, which results in skin tightening that lasts for 5 to 7 years. Histological analysis after four microneedling sessions spaced one month apart reveals a 400% increase in collagen and elastin deposition at six months, with epidermal thickening observed at one year post-treatment (48). Additionally, microneedling enhances transdermal drug delivery by bypassing the stratum corneum and delivering substances directly into the vascularized dermis. It also widens the follicular infundibulum by 47%, further facilitating medication penetration (49).

c. Indications and Contraindications

Microneedling is used for various dermatological conditions, including pigmentary disorders, acne scars, burn scars, traumatic scars, hypertrophic scars, striae distensae, androgenetic alopecia (with 5% minoxidil), alopecia areata (with topical steroids), melasma, periorbital hypermelanosis (with skin-bleaching agents), and primary axillary hyperhidrosis (with fractional radiofrequency microneedling) (5). Despite its general safety, microneedling is contraindicated in patients with active acne, herpes labialis, localized infections, severe chronic skin conditions like eczema or psoriasis, keloidal tendencies, immunosuppression, or those undergoing chemotherapy. Additionally, caution is advised near botulinum toxin injection sites to prevent unwanted diffusion (5).

d. Equipment and Technique

Microneedling devices include fixed needle rollers and electronically powered pens with disposable tips. Needle sizes vary based on treatment areas, with 1.5 to 2.0 mm needles preferred for scars and 0.5 to 1.0 mm for aging skin and fine wrinkles (50). Specialized devices include fractional radiofrequency microneedling, home-care rollers (0.1 mm needles for anti-aging agents), vacuum-assisted infusion microneedling, and LED microneedling rollers (51). Before treatment, topical anesthesia with lidocaine and prilocaine (EMLA) is applied for 15 to 45 minutes, followed by antiseptic cleansing (52). During the procedure, the skin is stretched while the microneedling device is moved in horizontal, vertical, and oblique directions 15 to 20 times until pinpoint bleeding is achieved. The process takes approximately 15 to 20 minutes, and immediate application of serums containing vitamins A and C can enhance regenerative effects (53).

e. Postoperative Care and Complications

Post-procedure erythema, mild edema, and exfoliative scaling typically resolve within 2 to 3 days. Patients should avoid sun exposure and harsh chemicals for at least one week and apply sunscreen regularly. While early serous drainage may occur, results become noticeable after 3 to 6 months due to ongoing collagen synthesis. Treatment can be repeated every three weeks (54). Common adverse effects include transient pain, erythema, irritation, and mild edema. Less frequent complications include hyperpigmentation (lower risk than with laser treatments), herpes simplex reactivation, superficial infections, allergic granulomatous reactions, and blood exposure risks (Fig 3, 4) (55).

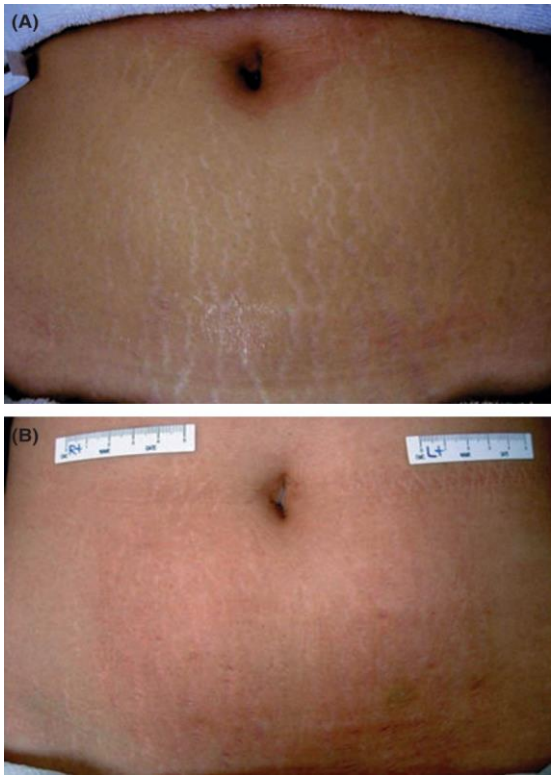

Fig (3): Photographs of a representative patient's abdomen with striae alba (A) before treatment and (B) three months after the final treatment with the microneedling system, showing marked improvement (56).

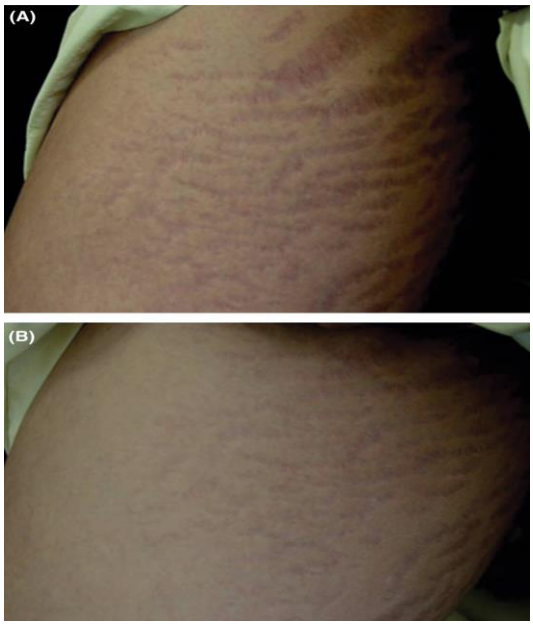

Fig (4): Photographs of a representative patient's thigh with striae rubra (A) before treatment and (B) three months after the final treatment with the microneedling system, indicating significant changes in texture and appearance **[56].**

f. Clinical Efficacy and Comparison with Laser Therapy

Microneedling is particularly effective for treating striae distensae due to its non-thermal approach and minimal risk of post-inflammatory hyperpigmentation, making it suitable for darker skin types (20). In a study involving 25 patients (skin types I-V), 1 to 3 microneedling sessions led to >50% improvement in stretch marks, with 28% of patients reporting >75% improvement [57]. study focusing on striae rubra confirmed increased collagen deposition and fibroblast proliferation, with minimal PIH cases (58). Compared to a 1340 nm non-ablative fractional laser, microneedling demonstrated similar efficacy for treating striae alba. However, pain levels were higher when needle depth exceeded 3 mm, necessitating local anesthesia (59).

Combination Therapies

Combining microneedling with platelet-rich plasma (PRP) has shown enhanced results, as PRP's growth factors further stimulate collagen production and accelerate healing. Patients receiving microneedling with PRP reported superior outcomes compared to microneedling alone (60).

Advantages and Limitations

Microneedling offers a shorter healing time, lower cost, and reduced risk of post-inflammatory hyperpigmentation compared to ablative treatments like CO2 laser resurfacing or deep chemical peels. It is also easy to perform and well-tolerated. However, the lack of large-scale, evidence-based studies and head-to-head comparisons with other treatments limit its definitive clinical recommendations (61).

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Consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interest.

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