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An Overview on Striae Distensae

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Abstract:

Background: Striae distensae (SD), commonly known as stretch marks, are a frequent dermal disorder characterized by linear atrophic lesions resulting from disruption of the extracellular matrix. Although not medically harmful, SD can cause considerable cosmetic concerns and negatively affect quality of life. Their pathogenesis is multifactorial, involving genetic susceptibility, hormonal influences, mechanical stretching, inflammatory mediators, and alterations in collagen and elastin metabolism.

Objective: To provide a comprehensive overview of the current evidence regarding the definition, classification, epidemiology, histopathology, pathogenesis, prevention, and treatment of striae distensae.

Methods: A narrative review of the recent literature was conducted, emphasizing clinical, histopathological, and molecular aspects of SD, together with evidence supporting currently available preventive and therapeutic interventions.

Results: SD progress through an early inflammatory phase (striae rubrae) and a chronic atrophic phase (striae albae), each exhibiting distinct clinical and histopathological characteristics. Molecular studies demonstrate impaired extracellular matrix synthesis, increased matrix degradation, altered growth factor signaling, and dysregulated collagen and elastin organization. Management remains challenging because no treatment achieves complete resolution. Preventive strategies show limited but promising efficacy, particularly formulations containing *Centella asiatica* derivatives. Therapeutic modalities—including topical retinoids, lasers, radiofrequency, microneedling, platelet-rich plasma, carboxytherapy, and microdermabrasion—have demonstrated varying degrees of clinical and histological improvement, with combination therapies generally producing superior outcomes compared with monotherapy.

Conclusion: Striae distensae are a multifactorial dermatologic condition with complex pathogenesis and substantial cosmetic impact. Advances in understanding their molecular mechanisms have expanded available treatment options; however, current therapies provide only partial improvement. Future well-designed randomized controlled trials and standardized outcome measures are needed to establish optimal evidence-based management strategies and develop more effective therapeutic approaches.

Keywords: Striae distensae, Stretch marks, Striae rubrae, Striae albae, Laser therapy.

Introduction:

Striae distensae (SD) are a common cutaneous disfigurement that present as linear streaks varying in color from erythematous and violaceous to hypopigmented. The early inflammatory stage, known as striae rubrae (SR), manifests as erythematous to violaceous lesions, whereas the chronic stage, striae albae (SA), appears as hypopigmented, atrophic lines. When striae occur during pregnancy, they are termed striae gravidarum (SG) **(Oakley & Patel, 2023)**.

The pathogenesis of SD is complex and remains incompletely understood. Current evidence suggests that their development results from the interaction of mechanical stretching, genetic predisposition, hormonal influences, inflammatory pathways, and dysregulation of extracellular matrix remodeling. These mechanisms lead to alterations in collagen and elastin architecture, impaired fibroblast function, and progressive dermal atrophy.

Advances in molecular biology and histopathological studies have considerably improved our understanding of the biological processes underlying SD formation and progression (**Macgregor & Wesley, 2024).**

Despite their high prevalence, the management of SD remains challenging because no available therapy completely restores normal skin architecture. Numerous preventive and therapeutic modalities—including topical agents, laser technologies, radiofrequency, microneedling, platelet-rich plasma, and carboxytherapy—have been investigated with variable levels of evidence. Continuous advances in energy-based devices and regenerative therapies have expanded treatment options, yet consensus regarding the optimal therapeutic approach has not been established **(Oakley & Patel, 2023)**.

Types and Clinical Presentations

The progression of SD generally follows two distinct phases: an early inflammatory stage, and a later chronic atrophic stage.

Striae rubrae, the early stage of SD, present as erythematous linear lesions that may be pruritic and raised **(Cho et al., 2006)**. The erythematous appearance is thought to result from increased dermal vascularity, including greater branching and widening of vessels, as well as enhanced vascular neogenesis mediated by endothelial cells. Separated collagen bundles may also increase the visibility of deep dermal vessels **(Wang et al., 2018)**.

Striae albae, the chronic stage of SD, appear as hypopigmented and atrophic dermal striations **(Tong et al., 2013)**. The white discoloration of SA is likely due to reduced melanin content, resulting from the flattening of rete ridges and dermal papillae, which decreases melanocyte density and melanin deposition **(Stamatas et al., 2015)**.

Epidemiology

Striae distensae are among the most common dermatological conditions, affecting an estimated 30% to 80% of the general population **(Cho et al., 2006; Seirafianpour et al., 2021)**. In adolescents, SD occured in approximately 40% of males and 70% of females **(Al-Himdani et al., 2014)**. SG occurred in around 60% of pregnant women and were more prevalent in those with younger gestational age and higher body mass index **(Ersoy et al., 2016; Punj et al., 2022)**.

Histopathology

Striae Rubrae

Microscopic examination of SR revealed edema between melanocytes and keratinocytes accompanied by increased melanogenesis in the epidermis **(Al-Himdani et al., 2014)**. Within the dermis, elastic fibers appeared thin and disorganized, while collagen bundles were abnormally separated, with greater separation in the reticular dermis compared to the papillary dermis. Spaces between collagen bundles contained loosely packed, disorganized collagen fibrils, tropoelastin-rich fibrils, and branching dilated vessels **(Wang et al., 2018)**. Prominent vasodilation and angiogenesis were observed, together with deep and superficial perivascular lymphocytic infiltrates and occasional eosinophils **(Borrelli et al., 2021)**.

Striae Albae

Microscopic examination of SA demonstrated a markedly thinned, atrophic epidermis with loss of rete ridges **(Borrelli et al., 2021; Mitts et al., 2005)**. The dermis was hypocellular, containing scant lymphocytic infiltrates and reduced microvasculature. Elastic fibers appeared thickened, and collagen bundles were densely packed and arranged parallel to the epidermis **(Borrelli et al., 2021; Hague & Bayat, 2017)**. In the papillary dermis, collagen bundles were finer than those in the reticular dermis **(Rolfe et al., 2012)**. The degree of collagen alignment correlated significantly with the loss of dermal papillae **(Bertin et al., 2014)**.

Pathogenesis

- **Molecular and Cellular Mechanisms**

Gene expression analyses of dermal fibroblasts from SD lesions have demonstrated downregulation of multiple extracellular matrix (ECM) components, including reduced expression of collagen type I (COL1),

collagen type III (COL3), and transforming growth factor-β1 (TGF-β1), a key inducer of collagen synthesis. Vascular endothelial growth factor A (VEGF-A), important for proper collagen deposition, was also downregulated **(Perez-Aso et al., 2019)**.

Additionally, the decreased expression of biglycan and lumican impaired collagen fibril assembly. Lysyl oxidase and fibronectin, essential for cross-linking of collagen and elastin, were reduced. ECM degradation was further enhanced by elevated levels of matrix metallopeptidase-3 (MMP-3) in combination with decreased expression of tissue inhibitors of metalloproteinases 1 and 2 (TIMP-1, TIMP-2). Increased plasminogen activator inhibitor-1 (PAI-1) also contributed to ECM breakdown **(Perez-Aso et al., 2019)**.

SR samples showed upregulated expression of genes associated with local tissue angiogenesis, including collagen type I alpha 1 (COL1A1), 5-hydroxytryptamine receptor 5A (HTR5A), connective tissue growth factor (CTGF), and pleiotrophin (PTN). On the other hand, analysis of SA samples revealed alterations in fundamental cellular physiological processes, including adenosine triphosphate (ATP) biosynthesis, purine and nucleotide metabolism, as well as keratin and keratin-associated proteins, which may contribute to local tissue atrophy **(Schuck et al., 2020)**.

- **Genetic Factors**

The genetic contribution to SD remains controversial **(Borrelli et al., 2021)**. A positive family history and prior occurrence of striae on the breasts, thighs, or buttocks, as well as striae during puberty were found to increase susceptibility to SG **(Mallol et al., 1991; Punj et al., 2022)**. Fibroblasts isolated from clinically normal skin of patients with SD demonstrated slower growth, migration, and proliferation **(Mitts et al., 2005)**.

Moreover, SD has been linked to monogenic connective tissue disorders such as Marfan syndrome and congenital contractual arachnodactyly, which result from mutations in fibrillin-1 and fibrillin-2, respectively, the principal structural proteins of elastic microfibrils **(Ledoux et al., 2011; Tung et al., 2013)**. Monozygotic twins have also been reported to develop identical SR patterns at the age of six in the absence of identifiable risk factors such as weight change, systemic disease, or drug exposure **(Di Lernia et al., 2001)**.

Three single nucleotide polymorphisms (SNPs) were significantly associated with SD: variants in the elastin (ELN) gene, the sushi-repeat containing protein X-linked (SRPX) gene, and the transmembrane protein 18 (TMEM18) gene, the latter of which has also been linked to obesity **(Tung et al., 2013)**. Conversely, another study failed to identify significant associations between SD and polymorphisms in ELN or COL3A1 genes. **(Kasielska-Trojan et al., 2019)**.

- **Hormonal Influences**

Hormonal signaling appears to play a key role in SD formation. Skin samples from patients with SD demonstrated increased expression of estrogen receptors (ER), androgen receptors (AR), and glucocorticoid receptors (GR) **(Cordeiro et al., 2010)**. Another study found ER expression to be doubled in SD compared with healthy skin **(Farahnik et al., 2017)**.

Examination of SD skin samples revealed a significant increase in GR in both nulligravida and multigravida females, as well as a significant increase in AR in multigravida females. In contrast to other studies, a significant decrease in ER was observed in SD lesions compared with skin samples from the healthy control group **(Youssef et al., 2017)**.

A prominent increase in the expression of secretoglobin (SCGB) genes, whose products selectively interact with some steroid hormones, has been reported. This finding supports the proposed association between the appearance of striae and elevated serum steroid hormone levels, which exert a catabolic effect on fibroblasts, thereby reducing collagen deposition in ECM and decreasing skin elasticity **(Schuck et al., 2020)**. In line with this, a study reported a significant association between appearance of SD in gymnastic males and their serum cortisol levels **(Hatta et al., 2022)**.

Clinically, SG has been associated with gestational diabetes, a potential indicator of adrenocortical hyperfunction **(Punj et al., 2022)**. The prevalence of SG has also been reported to be significantly higher in

pregnant women carrying a male fetus, possibly due to differences in sex steroid levels between fetal genders. **(Ersoy et al., 2016)**.

- **Mechanical Stress**

Excessive skin stretching, such as during pregnancy, puberty-associated growth spurts, or rapid weight gain, is thought to contribute to SD pathogenesis **(Tung et al., 2013)**. Increased weight gain and larger abdominal circumference during pregnancy were associated with higher incidence of SG **(Punj et al., 2022)**. SD typically appear parallel to skin tension lines, supporting the hypothesis that mechanical stretching disrupts dermal elastic fibers **(Mitts et al., 2005)**.

- **Inflammatory Mediators**

Local inflammation may play a role in SD pathogenesis. Inflammatory processes can weaken and destroy dermal collagen and elastic fibers while stimulating deposition of new ECM components along natural tension lines, leading to the clinical presentation of SD **(Borrelli et al., 2021)**. Elevated expression of inflammatory mediators such as interleukin-8 (IL-8), tumor necrosis factor-α (TNF-α), hyaluronan synthase-1 (HAS1), and interleukin-6 (IL-6) has been recognized in SD lesions **(Perez-Aso et al., 2019)**.

Management

Prevention

Certain formulations have shown partial efficacy. For instance, a topical formulation containing hydroxyprolisilane-C, rosehip oil, Centella asiatica–derived triterpenes, and vitamin E significantly reduced the incidence of SG and decreased the severity of erupted lesions during pregnancy **(García Hernández et al., 2013)**.

Another preparation containing Centella asiatica extract effectively lowered SG incidence in women with prior striae during puberty; in cases where SG still developed, lesions were less severe than in the placebo group **(Mallol et al., 1991)**. Application of bitter almond oil combined with massage also significantly reduced SG incidence, whereas the oil alone did not yield significant effects **(Taşhan & Kafkasli, 2012)**.

Treatment

- **Topical Treatments**

Topical agents have demonstrated variable efficacy. A combination of Centella asiatica and onion extract improved the appearance, softness, color, and texture of SR **(Draelos et al., 2010)**. Daily massage with silicone gel increased collagen content and reduced pigmentation more effectively than placebo, although both treatments improved erythema **(Ud-Din et al., 2013)**.

Mixtures of herbal oils and vitamins applied twice daily for four months enhanced clinical appearance, increased epidermal thickness, and stimulated dermal neocollagenesis **(Cantelli et al., 2021)**. Another cream containing silicon, synthetic recombinant growth factors and Centella asiatica extract improved the appearance and texture of SA after one month of twice-daily application **(Kikuchi et al., 2020)**.

Daily application of 0.1% tretinoin for three months did not alter SA lesion dimensions, but histologically increased dermal collagen content **(Listiawan et al., 2021)**. Regarding SR, application of 0.1% tretinoin cream once daily for six months significantly improved clinical appearance and reduced lesion dimensions, though histological collagen scores were not significantly changed **(Kang et al., 1996)**. Applying 0.05% tretinoin cream daily for four months similarly improved clinical appearance and reduced width and length of SR **(Hexsel et al., 2014)**.

- **Lasers**

A study evaluating the 308-nm excimer laser in the treatment of SA reported a significant improvement in color after nine treatments. Pigment correction maintained for two months then gradually declined to the baseline level which suggest the need to maintain treatment every two to three months **(Alexiades-Armenakas et al.,**

2004). Non-ablative fractional 1340 nm neodymium: yttrium–aluminum–perovskite (Nd:YAP) laser enhanced collagen and elastin formation and improved SD texture and color **(Naspolini et al., 2019; Viviano et al., 2022)**.

Pulsed dye laser (PDL) and intense pulsed light (IPL) were effective to improve the color and texture of targeted SD with superior outcome in early SR over old SA, and both triggered neocollagenesis. Post inflammatory hyperpigmentation (PIH) was an adverse effect in patients with dark skin types **(Al-Himdani et al., 2014; Nehal et al., 1999; Nouri et al., 1999; Shokeir et al., 2014)**.

Short-pulsed 10600-nm carbon dioxide (CO2) laser effectively improved the clinical appearance of SA. Histologically, studies reported thickening in the epidermis and new formation of rete-ridges with significant increase in the composition of dermal collagen and elastin. With taking into consideration that post inflammatory hyperpigmentation is a common adverse effect specially in dark skinned patients **(Al-Muriesh et al., 2020; Lee et al., 2010; Naspolini et al., 2019; Saki et al., 2022; Soliman et al., 2019)**.

- **Radiofrequency**

Both tripolar radiofrequency device and fractional microneedling radiofrequency devices were reported to improve the overall appearance of SD regarding color, texture and width. Under microscope, the treatment increased epidermal thickness, collagen bundles density and elastic fibers content **(Sobhi et al., 2019; Ahmed & Mostafa, 2019; Afify et al., 2020; Al-Muriesh et al., 2020)**.

- **Carboxytherapy**

Injection of CO_2 gas into tissues decreases pH to achieve the Bohr effect (the lower the pH, the weaker the bond between hemoglobin and oxygen), which stimulates an acute inflammatory reaction and leads to the release of oxygen from hemoglobin and vasodilatation of microcirculation **(Elmorsy et al., 2021; Hodeib et al., 2018; Shamban et al., 2025)**. Injection of CO_2 gas into the skin, either subcutaneously or intradermally improved the clinical appearance of SD by reducing their width and enhancing skin texture. Histologically, it stimulated collagen and elastic fiber formation and increased epidermal thickness **(Ahmed & Mostafa, 2019; Elmorsy et al., 2021; Hodeib et al., 2018)**.

- **Microneedling**

Microneedling (MN) has emerged as an effective therapeutic modality for striae distensae (SD), demonstrating efficacy comparable to fractional CO_2, plasma jet, and non-ablative fractional neodymium: yttrium–aluminum–perovskite (Nd:YAP) lasers, while offering a more favorable safety profile and a lower risk of post-inflammatory hyperpigmentation. Clinically, MN improves the color, texture, and dimensions of SD. At the microscopic level, it increases epidermal thickness and enhances collagen and elastin fiber density in the dermis **(Mohamad et al., 2022; Naspolini et al., 2019; Park et al., 2012; Saki et al., 2022; Soliman et al., 2019)**.

- **Microdermabrasion**

Microdermabrasion is a simple procedure that exfoliates the skin by rapidly brushing aluminum oxide crystals across the surface, removing the stratum corneum and at higher intensity, it may reach the papillary dermis **(Andrews et al., 2011; Bhalla & Thami, 2006; Spencer, 2005)**.

Weekly dermabrasion for 16 weeks significantly reduced the width and length of SR. Histologically, it increased epidermal thickness and stimulated new collagen formation. Compared with daily topical tretinoin application, dermabrasion produced similar clinical outcomes with fewer adverse effects **(Hexsel et al., 2014)**.

Platelet-rich plasma (PRP) injection demonstrated superior clinical and histological outcomes compared with microdermabrasion **(Ibrahim et al., 2015)**. Similarly, MN treatment was reported to achieve better clinical and histological improvements than microdermabrasion **(Nassar et al., 2016)**.

- **Intradermal Platelet-rich plasma Injection**

Platelet-rich plasma is a cost affordable treatment with minor side effects like transient bruising or pain during injection. Three to six sessions of intradermal injection of PRP improved the clinical appearance of both SA and

SR. Histologically, PRP injections improved epidermal thickness with new rete ridges formation, increased dermal density, induced collagen and elastin formation and decreased perivascular inflammatory infiltrate **(Ahmed & Mostafa, 2019; Hodeib et al., 2018; Ibrahim et al., 2015; Sawetz et al., 2021)**.

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