

# STRIA ATROPHICA

Authored by  
**Mohammed looti**

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## Introduction and Definition of Stria Atrophica

Stria atrophica, commonly known as **stretch marks** or **striae distensae** (SD), represents a pervasive dermatological condition characterized by linear, scar-like lesions that result from the rapid stretching and subsequent tearing of the dermal layers. These lesions initially present as erythematous or violaceous streaks, often referred to as **striae rubrae** (red striae), which reflect underlying inflammation and vascular proliferation. Over time, as the lesions mature and the inflammatory response subsides, they transition into pale, hypopigmented, and atrophic bands known as **striae albae** (white striae). The primary areas of affliction are typically regions subjected to significant mechanical stress or rapid volumetric changes, including the abdomen, thighs, buttocks, breasts, and arms. While striae atrophicae are benign and do not pose a direct health risk, their widespread prevalence, particularly among adolescents and pregnant individuals, makes them a significant cosmetic concern, often leading to considerable psychological distress and reduced quality of life for affected individuals. The nomenclature itself, derived from the Latin term 'stria' meaning furrow or streak, accurately describes the appearance of these distinct linear lesions traversing the skin surface, indicating a fundamental disruption in the structural integrity of the dermis, primarily involving the collagen and elastin fibers crucial for skin elasticity and resilience.

The core mechanism underlying the formation of striae atrophicae involves the failure of the dermis to adapt to rapid physical expansion. The skin is composed of three primary layers--the epidermis, the dermis, and the hypodermis. The dermis, situated beneath the epidermis, is responsible for providing strength and elasticity, largely due to its extracellular matrix (ECM) components, chiefly **collagen** (Type I and Type III) and **elastin**. When the skin surface is stretched too quickly, mechanical forces exceed the tensile strength of the dermal matrix. This rapid stretching causes microtrauma, specifically fracturing the elastic fibers and disrupting the collagen bundles. The initial inflammatory response triggered by this dermal injury leads to the characteristic purple or red appearance of striae rubrae. This phase involves vasodilation, edema, and the infiltration of inflammatory cells, alongside attempts at dermal repair which often prove inadequate or disorganized. Understanding this initial phase is critical, as therapeutic interventions are generally more effective during the active, rubra stage before the lesions become fully atrophic and hypopigmented.

Striae atrophicae are not solely dictated by mechanical tension; they are almost universally associated with concurrent hormonal shifts or changes in systemic factors. The primary endogenous factors implicated include elevated levels of glucocorticoids, either naturally occurring during periods of rapid growth or pregnancy, or exogenously administered through therapeutic use. Glucocorticoids are known to inhibit fibroblast activity, reducing the production of necessary collagen and elastin, while simultaneously promoting the degradation of existing connective tissue components. This dual action--impaired synthesis and enhanced degradation--renders the skin

structurally weak and highly susceptible to tearing under stress. Therefore, while mechanical stretching acts as the immediate trigger, the underlying hormonal environment sets the stage for dermal vulnerability. This interplay between physical forces and biochemical factors highlights why striae frequently occur during distinct physiological periods such as **puberty**, **pregnancy** (striae gravidarum), and conditions involving significant weight fluctuation, rather than simply being a result of general stretching.

## Etiology and Primary Risk Factors

The etiology of striae atrophicae is complex and multifactorial, generally categorized into mechanical stressors, hormonal influences, genetic predisposition, and underlying systemic diseases. Mechanical stress, stemming from the rapid expansion of body volume, is the most visible trigger. This expansion can result from rapid weight gain or loss, intense muscle building, or the physiological changes of pregnancy. During adolescence, the rapid linear growth spurt, particularly in height and muscle mass development, frequently leads to the formation of striae. For instance, young males often develop striae on the shoulders and back due to rapid muscle gain, while females frequently develop them on the breasts, thighs, and hips during pubertal development. The degree of mechanical stretching required to induce striae is highly variable among individuals, suggesting that factors other than physical tension alone are essential determinants of susceptibility.

Hormonal factors play a paramount role in predisposition. The correlation between striae formation and elevated levels of corticosteroids is well established. Cortisol, the primary endogenous glucocorticoid, acts by binding to receptors on fibroblasts, leading to decreased synthesis of collagen and elastin fibers, and simultaneously activating matrix metalloproteinases (MMPs), which facilitate the breakdown of the existing dermal matrix. This catabolic effect significantly compromises the structural integrity of the dermis. High levels of circulating hormones during **pregnancy** (especially late gestation) combine mechanical distension of the abdomen and breasts with the influence of estrogen, relaxin, and corticosteroids, culminating in a high incidence of striae gravidarum. Furthermore, certain underlying endocrine disorders, such as **Cushing's syndrome**, which is characterized by hypercortisolism, almost universally result in extensive, wide, and often brightly colored striae, frequently appearing in atypical locations like the flanks and axillae, underscoring the critical role of glucocorticoid excess in pathogenesis.

Genetic predisposition is also a critical, though less precisely defined, risk factor. Epidemiological studies suggest that if a parent or sibling has severe striae, the individual's risk of developing them is substantially elevated. This suggests the involvement of inherited factors controlling the structural integrity of connective tissue or the responsiveness of fibroblasts to hormonal stimuli. Specific genetic markers related to collagen synthesis, elasticity, and dermal repair mechanisms are hypothesized to influence individual susceptibility. Risk factors can be systematically

summarized to include:

**Pregnancy** (affecting up to 90% of women).

**Adolescent growth spurts** (puberty).

Rapid weight fluctuations (obesity or rapid weight loss/gain).

Endocrine disorders (e.g., Cushing's syndrome, Marfan syndrome).

Chronic use of systemic or high-potency topical **corticosteroids**.

High Body Mass Index (BMI).

Family history of striae.

## **Pathophysiology: The Mechanisms of Dermal Damage**

The pathophysiology of stria atrophica fundamentally involves the molecular breakdown of the dermal extracellular matrix (ECM). When rapid stretching occurs, fibroblasts--the cells responsible for synthesizing collagen and elastin--are subjected to tensile stress. While some stress can stimulate repair, excessive, rapid stress leads to damage and dysfunctional repair mechanisms. Histological analysis of early striae rubrae reveals a pronounced inflammatory infiltrate, characterized by mast cells and lymphocytes, suggesting an acute response to dermal injury. Crucially, the elastic fiber network shows significant fragmentation and disorganization. Instead of the normal dense, wavy network, the elastic fibers appear reduced in number, broken, and clumped, losing their ability to recoil, which is essential for skin elasticity.

During the transition from striae rubrae to striae albae, the inflammatory process subsides, and the lesions enter an atrophic phase. In striae albae, microscopy reveals a flattening of the epidermis and a significant reduction in dermal thickness. The key characteristic of mature striae is the organization of collagen fibers. Unlike the basket-weave pattern found in normal skin, the collagen bundles in striae albae are thin, dense, and oriented parallel to the skin surface and perpendicular to the direction of the stretch. This unnatural parallel alignment contributes to the clinical appearance of atrophy and furrowing. Furthermore, the synthesis of crucial ECM components, particularly Type I and Type III collagen, is markedly reduced compared to adjacent healthy skin, leading to a permanent structural deficit.

The influence of glucocorticoids serves as a major pathogenic factor by modulating fibroblast function. Glucocorticoids inhibit the synthesis of tropoelastin and procollagen, thereby suppressing the production of new elastic and collagen fibers. Simultaneously, these hormones upregulate the activity of matrix metalloproteinases (MMPs), particularly MMP-1 (collagenase) and MMP-3 (stromelysin), which are enzymes designed to degrade the ECM. This combination of decreased synthesis and increased degradation creates a severe imbalance, rapidly weakening the dermal scaffolding. The result is a mechanically compromised dermis that easily yields to even minor tensile forces, explaining why striae associated with hypercortisolism tend to be extensive, deep,

and rapidly formed. Research also suggests a role for altered vascularity; early rubra lesions show increased vascularity, which diminishes drastically in the alba stage, contributing to the pale, hypopigmented appearance due to loss of melanin and reduced blood supply.

## Clinical Presentation and Progression Stages

Stria atrophica follows a predictable clinical progression, moving through two main stages: the initial inflammatory phase (*striae rubrae*) and the mature atrophic phase (*striae albae*). Recognition of these distinct phases is vital for guiding therapeutic strategies, as interventions are typically more successful in the earlier, active stage. The initial presentation, *striae rubrae*, is characterized by raised, linear bands that are bright red, pink, or purple (violaceous). The elevated nature of these lesions indicates underlying edema and inflammation. Patients may sometimes report mild pruritus (itching) or a burning sensation localized to the developing striae. These lesions are typically oriented perpendicular to the direction of maximum skin tension, reflecting the line of dermal tear. Common sites include the lateral aspects of the thighs, the buttocks, the abdomen, and the lower back.

Over a period ranging from several months to a year or more, the *striae rubrae* lesions gradually transition into the mature stage, *striae albae*. As the inflammatory process resolves and vascularity decreases, the lesions become flattened, depressed, and significantly paler than the surrounding skin, often appearing white, silvery, or hypopigmented. This hypopigmentation is due to the loss of functional melanocytes within the atrophic area, making the striae particularly noticeable on individuals with darker skin tones. Palpation of *striae albae* reveals a soft, wrinkled texture, indicative of the underlying dermal atrophy and loss of elasticity. Unlike the raised texture of *striae rubrae*, *striae albae* are characterized by dermal thinning, giving them the scar-like, depressed appearance that defines the condition.

The location and morphology of the striae can often provide clues regarding their etiology. Striae associated with pregnancy (*striae gravidarum*) are predominantly found on the abdomen and breasts, often radiating outwards from the umbilicus. Striae related to rapid adolescent growth often appear on the lower back, thighs, and hips. Crucially, wide, numerous, and particularly dark purple striae found in atypical locations (such as the upper back, axilla, or flank) should raise strong suspicion of underlying endogenous hypercortisolism, such as Cushing's syndrome, necessitating further endocrine evaluation. The persistence of *striae albae* signifies a permanent change in the dermal architecture, making their complete elimination challenging, emphasizing the importance of early intervention when the lesions are still in the rubra phase.

## Classification of Striae Distensae

While the primary classification involves the temporal distinction between *striae rubrae* (early,

inflammatory) and striae albae (late, atrophic), striae distensae are also classified based on their underlying cause or specific morphological characteristics. A practical etiological classification helps clinicians identify and address underlying systemic issues. The main classifications include:

**Striae Gravidarum:** Those occurring specifically during pregnancy, typically appearing in the third trimester due to maximum abdominal distension and hormonal shifts.

**Striae Pubertatis:** Lesions resulting from rapid physical changes during adolescence, common in both sexes due to growth spurts.

**Striae Atrophicae:** A general term often used to describe the mature, white, depressed lesions resulting from any cause.

**Striae Maceratae:** Striae that develop due to rapid weight gain and subsequent loss, often characterized by greater irregularity.

**Striae associated with disease:** Those linked directly to endocrine disorders (Cushing's syndrome) or connective tissue disorders (Marfan syndrome, Ehlers-Danlos syndrome), which are often more severe and widespread.

Furthermore, clinical assessment often utilizes grading systems to quantify severity, which is important for research and therapeutic comparisons. Severity is typically judged based on parameters such as length, width, number, location, degree of atrophy, and color. Wider, deeper, and more numerous striae are considered more severe. For instance, striae resulting from high-dose topical steroid misuse often present as thin, very pale, and highly atrophic lines confined to the application site, differing markedly from the broad, violaceous striae seen in systemic hypercortisolism. The consistent clinical observation of the purple or red colour followed by the eventual white colour remains the most fundamental distinction, reflecting the shift from acute inflammatory response to chronic dermal atrophy.

### Striae Associated with Endocrine Conditions

While most instances of striae atrophicae are physiological (e.g., pregnancy or puberty), the presence of unusual or extensive striae should prompt an investigation into underlying endocrine pathology, particularly conditions involving excessive glucocorticoid production. **Cushing's syndrome**, whether dependent on ACTH or independent, is classically associated with severe striae distensae. These striae are often wide (exceeding 1 cm in width), remarkably violaceous (deep purple), and frequently appear in areas not typically affected by physiological stretching, such as the axillae, flanks, and inner thighs. The profound catabolic effect of chronic, high-level hypercortisolism on dermal collagen and elastin results in a skin structure so fragile that minor stress leads to extensive tearing. The prominent purple hue is thought to be due to thin skin allowing underlying engorged vasculature to be highly visible.

Beyond hypercortisolism, other hormonal factors may influence striae formation. Conditions

affecting connective tissue integrity, such as Marfan syndrome or Ehlers-Danlos syndrome, which involve genetic defects in collagen and elastin synthesis, also increase susceptibility to striae, though these are primarily genetic connective tissue disorders rather than purely endocrine. However, the interplay of hormones and connective tissue strength is undeniable. During puberty, the rapid increase in growth hormone and sex hormones (estrogen and testosterone) accelerates growth, placing extreme mechanical tension on the skin, which when combined with potential genetic susceptibility, results in striae pubertatis. Managing the underlying endocrine cause, such as controlling cortisol levels in Cushing's syndrome, is paramount, though existing striae often require specialized dermatological intervention.

### Iatrogenic Causes and Corticosteroid Impact

One of the most significant preventable causes of striae atrophicae is the prolonged or inappropriate use of corticosteroids. This phenomenon is termed **iatrogenic striae**. Both systemic corticosteroids (taken orally or via injection) and high-potency topical corticosteroids applied over large surface areas or under occlusion can induce severe dermal atrophy and subsequent striae formation. The mechanism mirrors that of endogenous hypercortisolism: the potent anti-inflammatory and catabolic actions of synthetic corticosteroids severely inhibit fibroblast proliferation and reduce collagen synthesis. The resulting dermal thinning makes the skin extremely vulnerable to tearing.

Iatrogenic striae caused by topical steroids are typically confined to the site of application and are often observed in flexural areas (like the groin or axillae) where occlusion naturally enhances steroid absorption. These striae tend to be thin, numerous, and intensely white (atrophic). They frequently result from the misuse of potent topical steroids prescribed for conditions like eczema or psoriasis, especially when treatment extends beyond the recommended duration or strength. Physicians must exercise caution when prescribing these agents, especially for pediatric patients or for long-term use in areas prone to striae formation. Patient education regarding the potential for dermal side effects, including striae and telangiectasia, is crucial to prevent this avoidable complication.

The development of striae following corticosteroid exposure highlights the dose-dependent and cumulative impact of glucocorticoids on dermal integrity. While a short course of steroids may have minimal effect, chronic exposure, even at moderate doses, significantly compromises the skin's tensile strength. The reversibility of these striae is low once the atrophic phase is reached, making prevention the primary goal. When systemic steroid therapy is unavoidable, co-management with dermatological monitoring for early signs of skin thinning is recommended, although the systemic nature of the drug often leads to widespread striae formation similar to Cushing's syndrome, though usually less severe unless the dosage is extremely high and prolonged.

## Differential Diagnosis and Histopathology

Although striae atrophicae are usually easily recognizable clinically, it is important to differentiate them from other dermatoses that present with linear skin changes or atrophy. Key differential diagnoses include:

**Anetoderma:** Characterized by localized areas of slack, herniated skin due to loss of elastic fibers, often appearing as small, depressed pouches rather than linear streaks.

**Linear Morphea (Localized Scleroderma):** Presents as indurated, linear bands that are firm and bound down, typically preceding atrophy, unlike the primary atrophy seen in striae.

**Scarring:** Trauma or surgical scars typically follow a wound pattern, whereas striae follow lines of tension in previously intact skin.

**Atrophoderma of Pasini and Pierini:** Characterized by large, slightly depressed, hyperpigmented patches, usually lacking the initial inflammatory rubra phase of striae.

Histopathological examination provides definitive diagnostic confirmation and elucidates the structural changes underlying striae. Biopsies taken from striae rubrae show signs of inflammation: epidermal flattening, perivascular lymphocytic infiltrates, and mast cell proliferation in the upper dermis. The most striking finding is the fragmentation and disorganization of the elastic fiber network, often appearing curled and clumped. Fibroblasts may appear activated, attempting an abortive repair process.

In contrast, mature striae albae biopsies reveal severe dermal atrophy. The epidermis is significantly thinned (flattened rete ridges). The dermis itself is reduced in thickness, and the characteristic basket-weave pattern of collagen is replaced by thin, parallel collagen bundles oriented perpendicular to the skin surface. Elastic fibers are drastically reduced in number and often absent in the mid-dermis. Special stains, such as Verhoeff-Van Gieson stain for elastic tissue, clearly demonstrate the loss or fragmentation of the elastic fiber network, solidifying the diagnosis of stria atrophica as fundamentally a disorder of elastic tissue integrity resulting from mechanical and hormonal stress.

## Psychosocial Impact and Quality of Life

Despite being medically benign, striae atrophicae carry a significant psychosocial burden, particularly for young women and adolescents. The highly visible nature of the lesions, especially when located on areas frequently exposed (e.g., abdomen, thighs, breasts), can lead to considerable cosmetic distress, impacting self-esteem and body image. Studies have documented that individuals with prominent striae often report feelings of embarrassment, anxiety, and self-consciousness, which can lead to avoidance of activities such as swimming or wearing certain types of clothing.

The psychological impact is particularly pronounced in **striae gravidarum**, where the lesions persist long after childbirth. While society accepts striae as a common consequence of pregnancy, their presence can negatively influence the mother's perception of her post-partum body image. Similarly, adolescents developing striae during puberty, coinciding with a critical period of identity and body image formation, may experience heightened anxiety and depression. Therefore, dermatological management should not focus solely on physical resolution but must also incorporate sensitivity toward the patient's emotional well-being and psychological distress. Clinicians must validate the patient's concerns and manage expectations regarding treatment outcomes, emphasizing improvement rather than complete eradication.

## Management and Treatment Options

The management of striae atrophicae is often challenging due to the permanent structural changes present in the mature lesions (striae albae). Treatment goals focus on minimizing the appearance of the lesions, ideally through early intervention during the striae rubrae phase when inflammation and cellular activity are still present, theoretically allowing for better dermal remodeling. Treatment modalities fall into topical agents, light/laser therapies, and mechanical procedures.

### Topical Treatments

Topical agents are most effective for striae rubrae. **Tretinoin** (retinoic acid) is the most widely studied and proven topical treatment. Tretinoin, a retinoid, works by stimulating fibroblast activity, leading to increased collagen and elastin synthesis, and promoting epidermal turnover. Studies show significant improvement in the length, width, and severity of striae rubrae when applied consistently. However, tretinoin is contraindicated in pregnancy and lactation, limiting its use for striae gravidarum. Other topical agents used include Vitamin C, glycolic acid, and specialized striae creams containing centella asiatica or hyaluronic acid, though evidence supporting these is generally weaker than for tretinoin.

### Light and Laser Therapies

Laser and light therapies are cornerstone treatments, selected based on the stage of the striae.

**Pulsed Dye Laser (PDL):** Highly effective for striae rubrae. PDL targets the hemoglobin in the blood vessels, reducing the redness and inflammation associated with the rubra stage. This vascular damage stimulates the dermal remodeling process, potentially preventing progression to severe atrophy.

**Fractional Photothermolysis (Non-ablative and Ablative Lasers):** Used for striae albae. These lasers create microscopic zones of injury in the dermis, forcing the body to initiate a vigorous wound healing response. This remodeling replaces disorganized, atrophic collagen with new,

healthier connective tissue, significantly improving texture and depth.

**Excimer Laser:** Used specifically for the hypopigmentation of striae albae, stimulating melanocyte activity to restore color, making the lesions less noticeable, particularly in patients with darker skin.

### **Mechanical and Energy-Based Procedures**

Various mechanical procedures aim to induce controlled dermal trauma to stimulate repair:

**Microdermabrasion:** Removes the superficial layer of the epidermis, promoting cell turnover, but results are often subtle.

**Microneedling (Collagen Induction Therapy):** Creates tiny punctures in the skin, physically stimulating fibroblasts to produce collagen and elastin, effective for improving the texture and depth of striae albae.

**Radiofrequency (RF) Devices:** Deliver heat deep into the dermis, causing controlled thermal injury that tightens existing collagen and stimulates neocollagenesis. Combined with microneedling (RF microneedling), this offers one of the most promising treatments for mature striae.

Given the complexity of striae formation, combination therapy--such as using PDL for rubra followed by fractional laser for the residual alba components--often yields the best clinical outcomes. Prevention, particularly through moisturizing and maintaining stable weight during high-risk periods, remains the most effective strategy.