Original Article

Brief Overview About Striae Distensae

Hadeer Aladdin Ali El Nagdy *[1], Enayat Mohamed Atwa [1], Hala Mohamed Morsi [1], Kamal Ahmed El Qishishi [2]

1 Dermatology, Venereology & Andrology Department, Faculty of Medicine, Zagazig University, Zagazig, Egypt
2 Pathology Department, Faculty of Medicine, Zagazig University, Zagazig, Egypt
Corresponding author: Hadeer Aladdin Ali El Nagdy
Email : Hadeera30@gmail.com
DOI: 10.47750/pnr.2023.14.02.163

Abstract

Background: Striae distensae (also known as stretch marks) appear as linear or fusiform lesions having variable length and width. Striae are asymptomatic but may cause itching and burning sensation. Striae distensae causes frequent dermatological consultations. Although SD is considered an aesthetic complaint, it can have important psychosocial consequences. Four distinct types were identified by High-resolution epiluminescence colorimetric assessment of SD: striae albae (white), striae rubrae (red), striae caeruleae (blue) and striae nigrae (black) Melanocyte mechanobiology seems to have a prominent effect on the various colors of striae in a direct and indirect way**.** Striae histopathologically, are characterized by a thin, flattened epidermis with blunted rete ridges. The dermis also is thinned, with a disturbance of its extracellular matrix. Collagen bundles are frayed or ruptured. Elastic fibers may be completely absent or if present are also damaged or ruptured. A universal approach to evaluate the severity of SD does not exist. Visual scoring and imaging modalities have been utilized in the literature. Different treatment modalities have been used for the treatment of SD, but non of them is a simple and definitive treatment. Striae distensa are frequently distressing to those affected and can cause significant cosmetic problem. It was suggested that time is the only treatment for SD and that it returns to normal over years, which is not true.

Keywords: Striae Distensae

INTRODUCTION

Striae distensae causes frequent dermatological consultations. Although SD is considered an aesthetic complaint, it can have important psychosocial consequences. Four distinct types were identified by High-resolution epiluminescence colorimetric assessment of SD: striae albae (white), striae rubrae (red), striae caeruleae (blue) and striae nigrae (black). Melanocyte mechanobiology seems to have a prominent effect on the various colors of striae in a direct and indirect way **(1).**

Striae distensae is 2.5 times as frequent in women and can occur in adolescence, pregnancy and obesity.The prevalence of SD is diverse in these groups and ranges from 43% to 88% and 6% to 86%, in pregnant women and adolescents, respectively. Among obese individuals of body mass index (BMI) 27-51, the prevalence is reported to be 43%. Other patient groups such as non-pregnant women and adult males also report varied prevalences **(2).**

Geographically distribution of SD demonstrates similar micro- and macroscopic appearances. However, inter-racial differences in the severity of SD have been observed in a study carried out by **Elbuluk *et al.*, (3)** of 48 women evaluated, African-American women were more severely affected than white women within the same geographical region. The authors did also note that there was a difference in BMI and smoking status between the two groups, which may indicate that other factors aside from race are involved**.**

Prevalence and anatomical regions affected vary depending upon sex and age in adolescents, approximately 40% of male and 70% of female subjects are affected. In adolescent males the lower back and knees are usually affected whilst in female subjects the thighs and calves are more often involved. During pregnancy, the abdomen and breasts are common sites for SD **(4).**

Risk factors and etiology:

The main etiology remains unclear due to the variability in the clinical conditions in which striae appears. They are common in different physiological conditions, including pregnancy, body changes, as seen in rapid weight change and adrenocortical excess**.** The prevalence of SD among adolescents is 40-70% and 90% after pregnancy**.** Also Hypercortisolism, as in Cushing's disease and local or systemic steroid therapy has a catabolic effect on fibroblasts activity and reduces collagen deposition in the dermal matrix. One more of risk factors is a positive family history in both of these groups **(4)**. Striae distensae are also arise in monozygotic twins, and in Marfan syndrome (connective tissue disorder), which indicate an important genetic predisposition. **(4).** Also, many medical

conditions, surgical interventions, medications and others have have been associated with striae distensae **(4).**

Table (1): Associations with striae distensae **(4).**

Medical conditions	Marfan syndrome, Cushing syndrome, anorexia nervosa, typhoid fever, rheumatic fever, chronic liver disease, obesity
Surgery	Augmentation mammoplasty, tissue expanded skin, tension-requiring skin sutures, organ transplantation, cardiac surgery
Medications	Systemic and topical corticosteroids, HIV therapy, chemotherapy, tuberculosis therapy, contraceptives, neuroleptics
Other	Pregnancy, puberty, stretch and weigh gaining, idiopathic

Connective tissue disorders

Connective tissue disorders such as Marfan syndrome (MFS) is inherited as an autosomal dominant trait and which displays major manifestations in the ocular, skeletal, and cardiovascular systems. MFS was believed to be resulted from impaired elastogenesis by mutant fibrillin-1 molecules acting in a dominant-negative manner on microfibril assembly **(5).**

Obesity

Obesity affects skin physiology, such as skin barrier function, sebaceous glands and sebum production, sweat glands, lymphatics, collagen structure and function, wound healing, microcirculation and macrocirculation, and subcutaneous fat .Striae appear as along cleavage lines perpendicular to the direction of greatest tension in areas with the most adipose tissue **(6).**

Pregnancy

In pregnancy, Striae distensae or "stretch marks," called striae gravidarum (SG). They have a considerable cosmetic concern to many patients. Clinically, they are linear bands that are initially erythematous to violaceous and gradually fade to become skin colored or hypopigmented atrophic lines (thin or wide). Abdomen, breasts, buttocks, hips, and thighs are common sites. Striae gravidarum are usually develop after the 24th week of gestation, the prevalence of SG in pregnant women ranges from 50% to 90%. Hormones, like estrogen, relaxin, and adrenocortical hormones, decrease the adhesiveness between collagen fibers and increase ground substance, which results in the formation of striae in areas of stretching**.** Striae also form due to change in connective tissue structural that include realignment and reduced elastin and fibrillin in the dermis **(7)**.

Endocrine disorder

Cushing syndrome caused due to excessive secretion of corticotropin (ACTH) that leads to hypersecretion of cortisol and other steroids by the zonae fasciculata and reticularis of the adrenal cortex, which have potent effects on cutaneous structure and function. Hypercortisolism is characrized by wide and purple skin striae, which contrasts with the narrow and pale or pink striae of rapid weight gain. Increase in protein catabolism, which compromises the integrity of collagen and elastin in the extracellular matrix is thought to be the main cause **(8).** Both Topical and systemic glucocorticoid (GC) therapy may induce different cutaneous side effects.Their occurrence and severity dependes onThe potency and in particular the duration of therapy. Atrophy of the epidermis and the dermis is the most important side effects, or even the subcutis, which may result in the irreversible striae rubrae, and disturbed wound healing. A less unimportant, side effect is the hypertrichosis (enhanced hair growth), which facially could be particularly problematic for women. However, it is usually reversible and disappears after cessation of therapy **(8).**

Adolescent growth spurt

In adolescence, striae commonly present over the thighs, gluteal region, and breasts in girls and over the lumbosacral region in boys. The incidence has been estimated to be up to 70% in females and 40% in males and overall incidence of approximately 25 to 35% **(8).**

Clinically, they appear as violaceous or reddish pink, slightly raised, parallel lines that course in a wavy pattern (stria rubrae). They are usually 2 to 5 mm in width, and their length varies according to location. they tendency to coalesce as they progress. During the earliest stages, patients may complain from minor pruritus or irritation. By time striae gradually fade in color and lose their erythematous, hemorrhagic appearance. They end as a white, atrophic, depressed lesion, often with a wrinkled surface.Striae alba takes monthes to years to develop and gradually fade with time. The unwelcome appearance of SD of adolescent is most probably produced by two main physiological mechanisms either due to mechanical shearing and stretching of the skin or adrenocortical hyperactivity or even both, as puberty is characterized by both rapid growth and stimulation of the hypothalamic-pituitary-adrenal axis **(9).**

Pathogensis of striae distensae:

1 - Genetic predisposition

The onset of cutaneous striae is attributed to a familial tendency. **Chang et al., (10)** in a retrospective study found genetic factors, such as family history, personal background and ethnicity, as important predictors for the onset of SD.

Stretch marks also associated with a few monogenic connective tissue diseases, including Marfan syndrome and congenital contractural arachnodactyly. These syndromes are caused by mutations in genes that encode extracellular matrix proteins (fibrillin-

1 and fibrillin-2, respectively) that are part of elastic microfibrils present in skin and other tissues. An important genetic predisposition also present as striae distensae are also described in monozygotic twins, in a familial form and **Marfan syndrom**. This is supported by biopsy findings that report slower migration and proliferation rates in fibroblasts of patients with SD **(11).**

Watson et al., (12), attribute the pathogenesis of striae to changes in the components of extracellular matrix, including fibrillin, elastin, and collagen**.** It was reported that , extracted total RNA from five samples of skin with striae, studied procollagen gene expression for type I and III, elastin, fibronectin and beta-actin, and compared with normal skin. Reduction of genes encoding for collagen, elastin and fibronectin, and a marked change in the metabolism of fibroblasts was observe. However, until now, no genetic variants are known to be associated with isolated stretch marks that afflict the general population **(4).**

2 - Mechanical agents

As a heterogeneous tissue the skin can produce 3 responses in response to a stretching force:

1. Reversible elongation, i.e., an "elastic" stretch response;
2. Elongation failure, to the extreme, with cleavage, i.e., an "inelastic" rigid response;
3. Mixture of the two responses with limited stretch and rigidity.

The third response corresponds to cutaneous striae. It was suggested that striae are always initiated by stretching, no matter if the stimulus is excessive or minimum. The cross-linking of collagen seems to be more important than the quantity of collagen in striae response to stretching. An increase in cross links, such as older age, increases the resistance to the deformation by stretching, but this rigidity leads to skin cleavage and not to formation of striae **(13).**

On the other hand, the absence of cross-linking leads to excessive elasticity and stretchability, with possible rupture of the skin if stretching is beyond the limit of elasticity, but without formation of striae. This occurs only in the skin area where the connective tissue is partially mature with a critical amount of collagen cross linked and immature "elastic" collagen, which allows a limited degree of stretch and a partial intradermal rupture, or the striae. The limited balance of stretching and cleavage would be a continuous process and an adaptation to the needs of growth in adolescence, and to changes in body mass in early adulthood **(13).**

It was reported that striae result from disruption of elastic fibers due to the forces of tension. Histological changes found in this study, such as fragmented collagen, abundant essential substance, and globular and quiescent fibroblasts that lose all signs of fibrillar secretion suggest a fibroblastic dysfunction due to distension. However, **Zheng et al., (14)** believe that striae are the result of an inflammatory reaction that determines the initial destruction of elastic fibers and collagen. The process would be followed by regeneration of elastic fibers in the direction imposed by mechanical forces.

Moraes et al., (15) observe in their study on skin distensibility and elasticity that it is possible to predict the onset of atrophic scars and cutaneous striae through a clinical test of distensibility with deformation higher than 0.4 cm. There are also studies on the contractile properties of fibroblasts of skin with SD. They found no significant difference in the generation of forces between fibroblasts from old SD, compared to normal skin fibroblasts. Furthermore, it was observed that the contractile properties of SD fibroblasts vary according to the lesion stage. In early injury, fibroblasts have a more contractile phenotype, similar to myofibroblasts.

3 - Hormonal and biochemical factors in the genesis of striae

Few studies actually reported the involvement of hormonal factors in the physiopathogenesis of SD, in spite of being cited in many studies of striae, mainly in cases related to pregnancy, puberty, and the use of corticosteroids.

Cordeiro et al., (13) observed significant increase in the expression of androgen, glucocorticoid, and especially estrogen receptors in skin with early SD, compared to skin without cutaneous striae. From this study, it is assumed that changes in the expression of hormone receptors occur in a well-defined time period of SD formation; therefore, there would be differences in the skin hormonal action in different stages of evolution of striae lesions. Similarly, to the tissue repair that occurs in the process of skin healing, in order to form cutaneous striae, there should be a reorganization and restructuring of the extracellular matrix (ECM) – related to factors that initiate the process of degradation of ECM macromolecules, coordinated by hormonal stimulation**.**

Histo-Pathology:

Striae histopathologically, are characterized by a thin, flattened epidermis with blunted rete ridges. The dermis also is thinned, with a disturbance of its extracellular matrix. Collagen bundles are frayed or ruptured. Elastic fibers may be completely absent or if present are also damaged or ruptured. Decreased expression of collagen, elastin, and fibronectin mRNA results in a loss of extra cellular matrix in the dermis **(13).**

In the early stages, histopathological dermal alterations, including mast cell degranulation and macrophage activation leading to elastolysis of the mid-dermis.Mast cells releases enzymes, includes elastases, which is proposed as a key initiatory process in SD pathogenesis. Recent SD show a deep and superficial perivascular lymphocytic infiltrate around the venules. Striae rubrae differs from striae albae clinically and histopathologically. Besides the inflammatory process, there is alterations in collagen, elastin and fibrillin content. It is thought The reorganization of fibrillin and elastin play an important role in SD pathogenesis and those predisposed to developing SD may have an underlying deficiency of fibrillin **(16)**.

Treatment of striae distensae

Different treatment modalities have been used for the treatment of SD, but none of them is a simple and definitive treatment. Striae distensa are frequently distressing to those affected and can cause significant cosmetic problem. It was suggested that time is the only treatment for SD and that it returns to normal over years, which is not true **(4).**

It has always been suggested that effective treatment of SD be instituted during the active stage, well before the scarring process is

complete **(17).**

Topical oil massage and herbal topical remedies

Some unconventional therapies and Traditional reports recommend applying unproven oils and natural remedies to stretch marks. The underlying principle for this use would probably be keeping the skin well hydrated. Sweet almond oil, wheat germ oil, olive oil, avocado oil, and castor oil and applying seaweed wraps have these properties. Other remedies such as comfrey, hypericum, maritime pine, equisetum, slippery elm, wheat grass and eucalyptus tree oil and cocoa butter are all used in creams or oils, but no efficacy studies have been performed to support these practices **(18).**

Tretinoin

Using topical tretinoin has yielded variable results, and some of the studies had proven the inefficacy of the vitamin A derivative in the treatment of SD but most of the patients included in this early study presented with old lesions that had evolved into whitish atrophic scars **(Elson, 1990)**.IN another study, tretinoin has been shown to improve the clinical appearance of stretch marks during the active stage (striae rubrae), although with not much effect during the mature stage (striae albae). Tretinoin is thought to work through fibroblast stimulation and induction of collagen synthesis **(19).**

Hydrant creams:

Despite the general hydration is essential to maintain the integrity and barrier function of the skin, little in the literature is available on the use of hydrant creams in prevention of SD. It is difficult to conclude such creams are effective due to the lack of clarity on the studies and availability of the scientific data **(20).**

Hyaluronic acid:

Hyaluronic acid is an organic substance found in human skin. Some preparations for prevention and treatment of stretch marks contain hyaluronic acid. It acts by stimulating fibroblast activity and production of collagen to restore any inhibition and collagen loss induced by hormonal fluctuations or mechanical stress **(21).**

Chemical and mechanical depridement techniques

Acid peel treatments such as glycolic acid and trichloroacetic acid are thought to act by inducing collagen synthesis **(16).** The correct concentration should be used; as higher levels may cause irreversible scarring**.** Microdermabrasion is a skin resurfacing technique using aluminium oxide**.** It has been reported to increase type I collagen and have a greater response in striae alba **(16).**

Lasers and Light Devices

Lasers have become a popular therapeutic modality used to improve stretch marks. The 585-nm flash lamp- pumped pulsed-dye laser (PDL) is the most commonly reported laser used in treatment of SD. The use of ablative technologies such as the short pulse carbon dioxide and erbium-substituted yttrium aluminium garnet (YAG) enjoyed a brief popularity because of prolonged healing and pigmentary alterations, especially in darker skin tone **(22)**.

Pulsed-Dye Laser

The early stage of the stretch mark formation is dilated blood vessels which render the striae rubrae is a good candidate for PDL**.** a clinical improvement in immature SD is achieved after several courses of 585-nm flash lamp PDL therapy using dynamic cooling. This laser has been purported to increase the amount of collagen in the extracellular matrix. It was reported that effectiveness of the 585-nm flash lamp PDL in stretch marks of skin types I to IV and demonstrated that collagen changes precede any clinical significant change. In a study by **Shokeir et al., (23)** striae rubrae were noted to respond better than striae albae to PDL or Intense pulsed light as assessed clinically by the width, color, and texture of the striae; however, this difference was not borne out by histological studies! This reiterates the principle that the early scars respond best to most interventions and histology can prove to be the gold standard to validate results**.**

Excimer Laser

The 308-nm xenon chloride (XeCl) excimer laser allows treatment of focal areas with a wavelength close to that of traditional narrow-band ultraviolet B (UVB) light. The excimer laser has the advantage over standard phototherapy of having greater precision and the ability to deliver higher energy fluences to the target tissue in less time. It is also possible that UVB radiation delivered in the form of laser light has a different light–tissue interaction, which may cause greater efficacy. After the use of the excimer in many hypopigmentary conditions, it was used for striae albae. Atemporary repigmentation and improvement of leukoderma was showen in SD with excimer laser. Post-laser biopsies showed greater melanin content and hypertrophy of the melanocytes, although it failed to show any improvement in skin atrophy **(24)**.

Copper-Bromide

The copper-bromide laser is a 577-nm laser that is only mentioned once in the literature as being used for stretch marks. A study concluded that the copper-bromide laser was effective in decreasing the size of the SD, although further studies are needed to determine the ideal parameters and the number of sessions needed for an optimum response **(4).**

Fractional Photothermolysis

Fractional photothermolysis is a nonablative resurfacing laser technique. This 1,550-nm laser creates microzones or microthermal zones of ‘‘injury’’ onto the skin. Within these areas, localized epidermal necrosis occurs alongside collagen denaturation. Ultimately, the necrotic debris is expelled, and neocollagenesis occurs. Additionally, because this laser treatment is non ablative, the islands of normal skin serve to speed the healing process. Fractional photothermolysis has been FDA approved for dermatological procedures requiring the coagulation of soft tissue; treatment of periorbital wrinkles; treatment of acne scars and surgical scars; photocoagulation of pigmented lesions such as lentigos (age spots), solar lentigos (sun spots), melasma, and dyschromia; and skin-resurfacing procedures. Results show that this method may result in clinical and histopathologic improvement

of striae distensae. All striae that showed excellent improvement were white, suggesting that fractional photothermolysis is most effective for patients with late-stage white striae. It may show transient and limited erythema, edema, and pigmentation as a side effect **(25**).

Radiofrequency Devices

Using radiofrequency (RF) devices have been reported to be an effective and safe noninvasive technique to tighten the face and neck skin. Unlike lasers, which convert light to heat and target a specific chromophore through the selective photothermolysis, RF devices transfer higher-energy fluences to the skin through a coupling method. The electrical energy transmitted is converted to heat upon reacting with the skin's resistance**.** It is reported that collagen fibril contraction occurs immediately after RF treatments, which induces new collagen formation **(26)**.

Fractionated microneedle radiofrequency and fractional carbon dioxide laser

In one stydy, Combination of fractionated microneedle RF with fractional CO2 laser induced clinical improvement of SD without serious side effects. This is, the first study using combined therapy of microneedle fractional RF with fractional CO2 laser that showed statistically significant improvement in roughness and thinning of the skin. Subjects who received combined treatment showed clinical improvement in skin texture and width according to investigator assessment. This Combination treatment markedly increased the number and thickness of collagen fibers. Greater expression of TGF-β1 and stratifin is likely to be responsible for accumulation and thickening of collagen fibers in the dermis. Stratifin, which is a keratinocyte-derived extracellular matrix modulating factor for dermal fibroblasts, induces matrix metalloproteinase-1 expression and activity in dermal fibroblasts to cause dermal remodelling. Postinflammatory hyperpigmentation was the only notable problem, but it spontaneously resolved in 2 months without significant adverse effects **(27)**.

Carboxytherapy

Carbon dioxide therapy (CDT), or carboxytherapy, improves skin elasticity, circulation and the appearance of fine lines and wrinkles, aids collagen repair, and destroys localised fatty deposits. Using intradermal carboxytherapy for the treatment of ageing or damaged skin was developed from an animal-based blind, interventional, cross-sectional study, which injected carbon dioxide into the dermis of wistar rats The researchers in the study observed the compacting of collagen fibres following the infiltration of carbon dioxide, and especially when this took place intradermally. Furthermore, there was a notably improved appearance in the skin, as well as intense collagen turnover **(27)**.

Carboxytherapy is safe and effective in stretch mark reduction**.** it is better to perform the technique on old, white depressed marks rather than young, red stretch marks. Also, deep striae are better than thin striae. Treatment should combine intradermal and subcutaneous injections using warm gas and a 30 G needle. The flow should be up to 80–150 cc/min. More often than not, the patient will not necessarily experience any pain with this treatment as the stretch marks already represent ruptured tissue; this means that the gas can diffuse more easily and is therefore less painful. Results are usually seen between four and 10 sessions, and should be maintained with two to three sessions per year. After injection, patients may experience erythema and a warm sensation **(28).**

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