

1. INTRODUCTION

Striae distensae (SD) are a common aesthetic skin condition that causes distress to the affected patients. They are a common finding on the abdomen, hips, thighs, and breasts. Women are most commonly affected (*Hexsel, 2009*).

Causes of striae distensae are not clear, and a number of theories have been proposed. *Kogoj (1925)* anticipated that a striatoxin damages the tissues in a toxic way resulting in striations. Others have shown that mechanical stretching is the main cause leading to rupture of the connective tissue framework. Normal growth has been suggested as another cause, with these marks commonly developing during adolescence and associated with the rapid increase in size of particular regions of the body (*Osman et al., 2007*).

Earlier-stage or immature SD tend to appear pink or red in color (striae rubra) and over time and with atrophic changes attain a white color (striae alba). High-resolution epiluminescence colorimetric assessment of SD identified two distinct types, striae alba and striae rubra. The direct and indirect influences of melanocyte mechanobiology appear to have a prominent effect on the various colors of SD (*Hermans and Pierard, 2006*).

Several treatments have been proposed, yet no consistent modality is available. Some authors, have suggested that time is the only treatment for SD and that it returns to normal over years, which is not true (**Alaiti and Obagi, 2007**).

It has always been suggested that effective treatment of SD be instituted during the active stage, well before the scarring process is complete (**Garcia, 2002**).

The use of topical tretinoin in SD in the last decades yielded variable results, and some of the studies proved the inefficacy of the vitamin A derivative in the treatment of SD. Tretinoin has been shown to improve the clinical appearance of SD during the active stage (striae rubra), although with not much effect during the mature stage (striae alba) (**Kang et al., 1996**). It was demonstrated that SD are associated with loss of fibrillin, a fact that explains the counter replacement of fibrillin upon retinoic acid therapy (**Watson et al., 1998**).

The dilated blood vessels marked at the early stage of the SD formation render the striae rubra a good candidate for pulsed dye laser (**Karsai et al., 2007**). The optimal treatment fluence was 3 J/cm². This laser has been purported to increase the amount of collagen in the extracellular matrix. **Jiminez et al. (2003)** documented the effectiveness of 585-nm flashlamp pulsed dye laser in SD of skin types I to IV and demonstrated that collagen changes precede any clinical significant change.

Dermaroller (Microneedling) is a new treatment modality for the treatment of scars, especially acne scars, stretch marks, wrinkles, and for facial rejuvenation. It is a simple and relatively cheap modality that also can be used for transdermal drug delivery (**Doddaballapur, 2009**).

The medical dermaroller needles are 0.5-1.5 mm in length. During treatment, the needles pierce the stratum corneum and create microconduits (holes) without damaging the epidermis. It has been shown that rolling with a dermaroller (192 needles, 200 μm length and 70 μm diameter) over an area for 15 times will result in approximately 250 holes/ cm^2 (**Fernandes, 2006**).

Microneedling leads to the release of growth factors which stimulate the formation of new collagen (natural collagen) and elastin in the papillary dermis. In addition, new capillaries are formed. This neovascularisation and neocollagenesis following treatment leads to reduction of scars (**Aust et al., 2008**).

2. AIM OF THE WORK

The aim of this study is to compare the effect of 585nm flashlamp pumped pulsed dye laser, topical tretinoin and dermaroller in treatment of striae rubra both clinically and histopathologically.

3.1 STRIAE DISTENSAE

Striae Distensae (SD) or Stretch Marks, become one of the common skin changes encountered especially with the worldwide increased frequency of obesity and subsequent lines of management that results in excessive loss of weight and concomitantly lax skin (*Zedek et al., 2007*).

3.1.1. Definition:

Striae distensae, are linear atrophic lesions that occur in the body. They typically begin as violet or reddish-pink wavy lines, slightly raised (striae rubra), and over a period of months to years, fade to barely visible white atrophic lesions with a wrinkled surface (*Feldman and Smith, 2007*).

Multiple names have been given to identify this condition: (striae atrophicae, striae distensae, stretch marks and line atrophicae). Striae are a very common condition in all age groups. They form in areas of dermal damage produced by stretching of the skin (*Ammar et al., 2000*).

Striae distensae represent a significant source of distress to those affected. The pathogenesis of striae is poorly understood but probably related to changes in the components of the extracellular matrix, including fibrillin, elastin and collagen. Striae are a form of dermal scarring in which the dermal collagen ruptures (*Hidalgo, 2002*).

3.1.2. Epidemiology:

The highest incidence of striae is 20%-70% between 10 and 16 years of age among girls and 6%-40% between 14 and 20 years among boys (*Goldman and Rostan, 2008*). Approximately 90% of pregnant women, 70% of adolescent females, and 40% of adolescent males (many of whom participate in sports) have SD (*Perez et al., 2002*).

Striae are 2.5 times more often seen in women than in men and appear in many healthy women, originating either during puberty or at the time of the first pregnancy (*Arnold et al., 2000*).

Striae distensae affect persons of all races. However, women or men with greater amounts of melanin are less prone to these marks (*Arnold et al., 2000*).

Although SD represent a cosmetic problem if extensive, they may tear and ulcerate when an accident or excessive stretching occurs (*Alaiti, 2001*).

3.1.3. Aetiology:

The exact pathogenesis of striae has yet to be elucidated and a number of theories have been proposed but, mechanical, hormonal, and genetic factors may play a role (*Zedek et al., 2007*).

3.1.3.1. Genetic Factors:

Skin structure and skin properties are genetically determined and therefore individuals may be predisposed to the development of SD. An individual is likely to be more susceptible to SD if there is a family history (**Goldman and Rostan, 2008**). It is presumed that SD have been reported in monozygotic twins. There is decreased expression of collagen and fibronectin genes in affected tissue (**Dilernia et al., 2001**).

The role of genetic factors is further emphasized by the fact that they are common in inherited defect of connective tissue. The absence of striae in pregnancy in women with Ehlers-Danlos syndrome and their presence as one of the minor diagnostic criteria for Marfans syndrome emphasize the importance of genetic factors in determining susceptibility of connective tissue to this lesion (**Barrows and Lovell, 2004**).

3.1.3.2. Mechanical stress:

Striae distensae affect skin that is subjected to continuous and progressive stretching due to increased size of the various parts of the body, rapid deposition of adipose tissue, or muscular hypertrophy beneath the dermal layer (**Petrou, 2007**).

Normal growth has been suggested as another cause, with these marks commonly developing during adolescence and associated with the rapid increase in

size of particular regions of the body. Obesity and rapid increase or decrease in weight have been shown to be associated with the development of SD (*Elsaie et al., 2009*).

Many authors have denounced this theory, as they did not find any relationship between growth in abdominal girth in pregnant women and formation of SD (*Osman et al., 2007*).

3.1.3.3. Hormonal Factor:

Corderio et al. (2010) quantified the estrogen, androgen and glucocorticoid receptors in skin sample with striae and comparing them with normal skin. Skin sample from striae showed increased number of receptors for these hormones than normal healthy skin. Regions that undergo greater mechanical stretching of the skin may express greater hormonal receptors activity. This activity may influence the metabolism of the extracellular matrix, causing the formation of SD.

During stretching, hormonal receptors affect the epidermis by preventing the fibroblasts from forming collagen and elastin fibers, necessary to keep rapidly growing skin taut. This create a lack of supportive material as the skin is stretched and leads to dermal and epidermal tearing. If the epidermis and the dermis have been torn, treatment will be more difficult (*Petrou, 2007*).

Adrenocorticosteroids also may have a role, higher levels of urinary adrenocorticosteroids is found in obese

patients with striae as opposed to obese patients without striae (*Zedek et al., 2007*).

There are many factors that alter the hormonal effect on the skin and induce striae. These factors include puberty, pregnancy, oral contraceptive pills, and corticosteroids (*Burrows and Lovell, 2004*).

3.1.3.4. Other Factors:

Other rarely reported causes of SD include cachetic states, such as tuberculosis and typhoid and after intense slimming diet (*Sparker et al., 1996*). They may also be seen in anorexia nervosa (*Strumia et al., 2001*).

Striae distensae have been reported to occur rarely in patients positive for the human immunodeficiency virus receiving the protease inhibitor indinavir (*Burrows and Lovell, 2010*). Men and women with chronic liver disease may also have striae distensae (*Johnston and Graham, 2007*).

3.1.4. Pathogenesis:

The pathogenesis of striae is unknown but in the light of morphological and molecular data, striae suggest a correlation between loss of fibroblast synthesis and abnormalities in connective tissue, in addition to decreased collagen, elastin and fibrillin fibers when compared with normal skin (*Vinnet et al., 2005*).

The controversial etiology of SD has been explored through multiple factors such as genetic predisposition, normal growth, mechanical stretch, hormonal changes, and others (*Elsaie et al., 2009*).

It has been proposed that striae are a form of dermal scarring in which the dermal collagen ruptures (*Garcia, 2002*). It has been suggested that they develop more easily in skin which has a critical proportion of rigid cross linked collagen as occurring in early adult life (*Burrows and Lovell, 2004*).

Sheu et al. (1991) in a study on early striae distensae, found that sequential changes of elastolysis accompanied by mast cell degeneration occur in the very early stage of SD. Elastic fibers are the primary target of the pathological process and the abnormalities extend as far as 3cm beyond the lesion into the normal skin. This is evident in striae due to pregnancy, lactation, weight lifting, and other stressful activities. Increased adrenal cortical activity has been implicated in the formation of striae, as in the case of Cushing syndrome. Additionally, the cellular and extracellular matrix alterations that mediate the clinical phenotype of stretch marks remain poorly understood (*Arnold et al., 2000*).

Striae could be a feature of high serum levels of steroid hormones. They are a common feature of Cushing's disease and local or systemic steroid therapy may induce them. High steroid hormone levels have a catabolic effect

on the activity of fibroblasts and decrease the deposition of collagen in the substance of the dermal matrix (**Nieman and Ilias, 2005; Das et al., 2009**). Both systemic and topical steroid therapy can produce cutaneous atrophy by a dose-related pharmacological effect. The effect is more severe with the more potent steroids (as assessed by the vasoconstrictor assay test), but both fluorinated and non-fluorinated topical steroids can cause atrophy. The effect is most marked when potent steroids are applied topically under an occlusive dressing. The skin becomes thin, fragile and transparent, and striae may develop (**Burrows and Lovell, 2010**).

3.1.5. Clinical picture:

3.1.5.1. Sites:

In pregnancy, striae usually affect the abdomen and the breasts. The most common sites for striae in adolescents are the outer aspects of the thighs, lumbosacral regions in boys and thighs, buttocks and breasts in girls. Considerable variation occurs, and other sites are also affected including the inner aspects of the upper arms (**Salter and Kimball, 2006**).

Striae induced by prolonged systemic steroid use are usually larger and wider than other phenotypes of striae, and they involve widespread areas. Striae secondary to topical steroid use are usually related to enhanced potency of the steroids, especially when using occlusive plastic wraps. They usually affect the flexures and may become less visible if the offending treatment is withheld early enough (**McDaniel, 2002**).

3.1.5.2. Signs and Symptoms:

Early striae present as flattened, thinned skin with a pink hue that may occasionally be pruritic (**Jimenez et al., 2003**). They usually increase in length, width and acquire a darker reddish purple appearance (striae rubra). Mature striae assume white, depressed, irregularly shaped bands with their long axis parallel to the lines of skin tension (striae alba). They are generally several centimeters long and 1-10 mm width. Gradually, some striae may fade and become inconspicuous (**Burrows and Lovell, 2004**).

The Striae distensae were classified into 6 stages according to the clinical appearance as shown in table (3.1) (**Adatto and Deprez, 2004**).

Table (3.1): Classification of striae distensae based on the clinical appearance (**Adatto and Deorez, 2004**).

	Clinical Appearance
Stage (I)	Fresh, inflammatory usually livid striae.
Stage (IIa)	White, superficial striae without laddering and without palpable depression at the surface of the skin
Stage (IIb)	white, superficial striae without laddering but with palpable depression at the surface of the skin.
Stage (IIIa)	White, atrophic striae with laddering measuring less than 1 cm width, without deep pearliness.
Stage (IIIb)	White, atrophic striae with laddering measuring less than 1 cm width, with deep pearliness.
Stage (IV)	White, atrophic striae with laddering measuring more than 1 em width, with or without deep pearliness.

3.1.6. Histopathology:

The histology of SD is that of a scar, and the development has been linked to that of wound healing or scar formation. Histopathology findings vary according to the duration of lesions (*Atwal et al., 2006*).

In early lesions, there are superficial and deep perivascular infiltrate of lymphocytes and sometimes of eosinophils, as well as widely dilated venules and edema in the upper part of the dermis. Fully developed lesions show scant infiltrate of lymphocytes around venules. Bundles of collagen in the upper third of the reticular dermis are thinned and aligned parallel to the skin surface. Elastic fibers seem to be increased in number and packed together as a consequence of loss of collagen bundles (*Burrows and Lovell, 2010*).

In later stages of SD formation, there is a thinning of the epidermis due to flattening of the rete ridges. The dermal collagen is layered in thin eosinophilic bundles, orientated in straight lines parallel to the surface in the direction of the presumed stress. Scanning electron microscopy shows it as amorphous, sheet-like structures (*Zheng et al., 1985*).

There is a loss of elastic tissue which accompanies the loss of collagen. Elastic stains show breakage and retraction of the elastic fibers in the reticular dermis. The broken elastic fibers curl at the sides of the striae to form a distinctive pattern (*Arnold et al., 2000*). Loss of both collagen and

elastic tissue are a requisite for the development of striae (*Ackerman et al., 1997*).

At the end, there is a decrease in the thickness of the dermis (*Zheng et al., 1985*). In addition, hair follicles and other appendages are absent (*Garcia, 2002*). There is an increase in the glycosaminoglycan content in striae; furthermore, the number of vertical fibrillin adjacent to the dermo-epidermal junction and the elastin fibers on the papillary dermis are reduced (*Watson et al., 1998*).

3.2 TREATMENT MODALITIES OF STRIEA DISTENSÆ

A number of treatment modalities are available for treatment of SD, yet none of them is consistently effective, and no single therapy is considered to be pivotal for this problem. With a high incidence and unsatisfactory treatments, SD remain an important target of research for an optimum consensus of treatment (*Elsaie, 2009*).

Various treatment modalities are available for the purpose of improving the appearance of existing SD, including topical treatment (*Rangel et al., 2001*), Chemical peel (*Khunger, 2008*), Mesotherapy (*Rotunda et al., 2004*), Dermal roller (*Doddaballapur, 2009*), Intense pulsed light (*Perez et al., 2002*) and Laser treatment (*Suh et al., 2007*).

3.2.1. Topical treatment:

Improvement in the appearance of striae by topical agents has been aggressively sought for many years. In general, topical treatment has yielded disappointing results with only modest improvement reported in mature striae (*Ash et al., 1998*).

3.2.1.1. Topical natural remedies:

Some unconventional therapies and anecdotal reports recommend applying unproven oils and natural remedies to SD. The underlying principle for this use would probably