

Striae Distensae: physiopathology

ABSTRACT

Introduction: Cutaneous atrophic striae or striae distensae (SD) is a very common disease. Although SD is considered an aesthetic complaint, it can have important psychosocial consequences. Moreover, its occurrence may reflect changes in tissue, and express local and systemic pathological conditions. Considering the multiplicity of factors involved, the literature is divergent and inconclusive. **Objective:** To investigate striae physiopathological aspects, which have been studied, through systematic review of the literature. **Method:** We conducted searches using three databases: MEDLINE (1966–2009), Cochrane Library and LILACS, in Portuguese, English and Spanish, besides search for keywords in PubMed, review of bibliographic references of the articles found, and manual search of the main Dermatology journals. **Results:** We identified 113 articles, 101 in MEDLINE, 12 in Cochrane Library and none in LILACS, for striae distensae. Of these, 25 publications were considered for descriptive purposes, were analyzed 10 studies that were individually controlled (compared with samples of healthy skin) or comparative (recent and old striae). **Conclusions:** There are few researches of good quality on the physiopathology of cutaneous striae. The priority of most studies is on therapy, with little interest in understanding the physiopathology. The knowledge of SD physiopathology is not only important for the development of preventive methods and more effective treatment, but also for better understanding of local and systemic changes related to the connective tissue.

Keywords: esthetics, physiopathology, elastic tissue, collagen.

INTRODUCTION

Cutaneous atrophic striae or striae distensae (SD) is a very common disease, which causes frequent dermatological consultations. Although SD is considered an aesthetic complaint, it can have important psychosocial consequences.¹ Moreover, the occurrence of striae distensae may reflect changes in tissue, and express local and systemic pathological conditions.²

According to Rabello,³ SD are characterized by “skin atrophy with elongated form, sometimes wavy, elevated, flat or depressed, but always soft and depressive, which after some time become less apparent. The color is pale or bluish when recent, and pearly white towards the end. When touched, they seem soft with a relative emptiness, as if the skin was over a mobile and fleeting plan. The direction, roughly speaking, corresponds to cleavage lines or skin tension”.

Striae can be associated with puberty (Figure 1), pregnancy (Figure 2),^{4,5} Cushing’s syndrome,⁶ obesity,⁷ and topical⁸ and sistemic^{9,10} use of corticosteroids (Figure 3). Associations have also been described with Marfan’s syndrome,¹¹ infections such as tuberculosis,¹² load lifting and bodybuilding,¹³ rapid changes in weight, tissue expansion, sutures with tension¹⁴ and, more recently, related to breast enlargement surgery.¹⁵

Although striae are described by some authors as a condition of skin stretching or distention, with loss or disruption of elastic fibers in the affected region, others note that the striae do not arise frequently on the abdominal skin above tumors, ascites, extensive bleeding or large hernias.⁶ Despite innumerous studies, the cause of striae appearance remains obscure. Rosenthal¹⁶ proposed four etiological mechanisms of striae formation: insufficient development of tegument, including elastic properties deficiency; rapid stretching of the skin; endocrinal changes; and other causes, possibly toxic. Considering the multiplicity of factors involved, the literature is divergent and inconclusive.

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OBJECTIVE

The objective was to study through systematic review of literature the physiopathological aspects of striae.

METHOD

Search strategy and studies selection

In the period from March 5 to April 15, 2009, searches were performed using three databases: MEDLINE (1966–2009), Cochrane Library, and LILACS, in Portuguese, English and Spanish. We used the following keywords: striae distensae, stretch marks, estrias cutâneas; and cross searching with the words: fisiopatologia/physiopathology, genética/genetics, fatores mecânicos/mechanical factors, fatores hormonais/hormonal factors, estrógeno/estrogen, andrôgeno/androgen, glicocorticoide/glicocorticoid, fibras elásticas/elastic fibers and colágeno/collagen. Other strategies used were: search for keywords in PubMed, review of bibliographic references of the articles found, and manual search of the main Dermatology journals.

Study selection criteria

A total of 113 articles were identified, 101 in MEDLINE, 12 in Cochrane Library and none in LILACS, for striae distensae. Twenty five publications were considered for descriptive purposes, with joint analysis, since they mentioned some aspects of striae physiopathology. For the proposed objective, 10 studies that were either controlled (compared with samples of healthy skin) or comparative (recent and old striae) were individually analyzed. Included in these categories were also the randomized, blind, or open studies.

Methodological quality

The methodological quality of the studies was assessed through evaluation of to the following criteria: appropriate randomization, use of control group, inclusion and exclusion criteria clearly described; technique appropriately described, use of laboratory methods for evaluation, biopsy of skin, and anatomopathological examination with special staining, electron microscopy, immunohistochemistry, among other methods.

RESULTS

For didactic purposes, the physiopathological aspects were divided into genetic factors, mechanical factors, and hormonal factors. Nine publications were related to mechanical factors, 8 to hormonal factors, and 8 to genetic predisposition.

1 - Genetic predisposition

Some authors attributed the onset of cutaneous striae to a familial tendency.^{4,17} Chang *et al.*¹⁸ in a retrospective

study found genetic factors, such as family history, personal background and ethnicity, as important predictors for the onset of SD. Lerner *et al.*¹⁹ reported the onset of striae rubrae in monozygotic twins of 6 years old. The patients had no dimorphic features or musculoskeletal deformities. They had moderate hyperextension of the joints and ligaments, and present no hematological or endocrinal changes.

Genetic factors may be related with the presence of striae, and may be associated with syndromes such as Ehlers-Danlos,²⁰ Marfan,¹¹ ectodermal dysplasia, and autosomal dominant familial striae distensae.²¹ Watson *et al.*²² attribute the pathogenesis of striae to changes in the components of extracellular matrix, including fibrillin, elastin, and collagen. Lee *et al.*,²³ 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 no skin lesion. The authors observed reduction of genes encoding for collagen, elastin and fibronectin, a marked change in the metabolism of fibroblasts.

2 - Mechanical agents

According to Shuster,²⁴ the skin is a heterogeneous tissue and 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. The author suggests 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. 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.

Pieraggi *et al.*²⁵ suggest 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.*²⁶ 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.

Henry *et al.*²⁷ observed changes in mechanical properties of the skin throughout pregnancy, with increased extensibility and maintenance of elasticity, leading to the formation of striae, especially in the last 3 months.

Piérard *et al.*²⁸ reported differences between mechanical properties of the skin with striae *in vivo* and *ex vivo*. The mechanical properties of skin with SD were markedly different from the apparently normal skin. In the skin with SD, all rheological parameters of elasticity and extensibility had abnormal responses. Increase was observed in the extensibility of striae site. However, elasticity was reported to be decreased *ex vivo* and unchanged *in vivo*, probably due to inherent forces present *in situ*. It is assumed that in SD, the connective tissue shows weak resistance to tensile stress.

Moraes *et al.*,²⁹ in a study on skin distensibility and elasticity, observed 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 skin with SD fibroblasts^{30,31}. 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

Until very recently, despite the involvement of hormonal factors being cited in many studies of striae,^{4,5,6,8,9,10} mainly in cases related to pregnancy, puberty, and the use of corticosteroids, few studies actually found their involvement in the physiopathogenesis of SD.

Simkim and Arce³² studied the 24 hours urinary excretion of 17-cetosteroides and 17-ketogenic steroids in obese patients. Although the mean excretion of all obese patients (15.8 mg) was significantly higher, compared to nonobese patients, the excretion was higher in obese patients who had skin striae (20.4 mg). Approximately 78% of obese patients with striae showed increased 17-cetosteroides, but this result was not statistically significant.

Cordeiro, Zecchin and Moraes³³ 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 receptor hormones 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.

DISCUSSION

There are few researches of good quality on the physiopathology of cutaneous striae. The focus of most studies is on therapy, with little interest in understanding the physiopathology. Among the studies on striae physiopathology, the majority emphasizes only one of the factors associated with the onset of SD, with the mechanical factor been the most



Figura 1 – Striae associated with puberty.



Figura 2 – Striae associated with pregnancy.



Figura 3 – Striae associated with use of systemic corticosteroids.

studied. Perhaps the methodological difficulty of conducting further laboratory studies explains the lack of systematic studies on this subject. SD are basically an aesthetic dermatological change, disfiguring but harmless, so there are restrictions on obtaining material for skin biopsies in larger studies, especially for early striae, and comparative studies with healthy skin.

The knowledge of SD physiopathology is not only important for the development of prevention methods and more effective treatment, but also for a better understanding of local and systemic changes related to the connective tissue.

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