Striae distensae

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Striae distensae or stretch marks were described as a clinical entity hundreds of years ago, and the first histologic descriptions appeared in the medical literature in 1889.^[1] They are a common disfiguring cutaneous condition, characterized by linear smooth bands of atrophic appearing skin^[2] which occurs in areas of dermal damage produced by stretching.^[3]

ETIOPATHOGENESIS

The exact etiology still remains controversial and this is partly due to the variability in the clinical situations in which striae arise.^[1] They are the end result of various physiologic states, including pregnancy, adrenocortical excess and changes in body habitus, as seen in rapid weight change. A genetic predisposition is presumed;^[4] striae distensae have been reported in monozygotic twins.^[5] There is decreased expression of collagen and fibronectin genes in affected tissue.^[6] The role of genetic factors is further emphasized by the fact that they are common in inherited defects of connective tissue,^[2] as in Marfan's syndrome.^[3]

The occurrence of striae correlates closely with obesity.^[3] They are highly prevalent in obese adults and children,^[3] but the development of striae in adolescents is not related to obesity and rather coincides with the markers of adolescence such as breast development, pubic hair growth and menarche.^[4] In a study on skin diseases in children with organ transplants, steroid induced striae distensae were found only in adolescents

and not among younger children.^[7] Striae are seen in 90% of pregnant women,^[8] due to a combination of hormonal factors (e.g. adrenocortical hormones, estrogen, and relaxin) along with increased lateral stress on connective tissue.^[8] Young male weightlifters develop striae on their shoulders.^[3] Striae also occur frequently in patients with hypercortisolism as in Cushing's syndrome and in those using topical steroids.^[3] Edematous striae distensae are uncommon, but can develop due to the combined effects of systemic glucocorticoids and generalized edema.^[9]

It has been noted that striae are prevalent in cachetic states, such as tuberculosis, typhoid and after intense slimming diets.^[10] They may also be seen in anorexia nervosa.^[11] Striae have been reported to occur rarely in human immunodeficiency virus positive patients receiving the protease inhibitor indinavir.^[3] A case of idiopathic striae was also reported.^[12] Men and women with chronic liver disease may also have striae.^[13]

The pathogenesis of striae is unknown but probably relates to changes in the components of extracellular matrix, including fibrillin, elastin and collagen.^[14] Arem and Kisher have proposed that striae are a form of dermal scarring in which the dermal collagen ruptures.^[11] It has been suggested that they develop more easily in skin which has a critical proportion of rigid cross linked collagen as occurs in early adult life.^[3] In a study on early striae distensae, Sheu et al found that sequential changes of elastolysis accompanied by mast cell

How to cite this article: Singh G, Kumar LP. Striae distensae. Indian J Dermatol Venereol Leprol 2005;71:370-2. Received: April, 2005. Accepted: July, 2005. Source of Support: Nil. degeneration occur in the very early stage of striae distensae.^[15] Elastic fibre is the primary target of the pathological process and the abnormalities extend as far as 3 cm beyond the lesion into the normal skin.^[15]

PATHOLOGY

Inflammatory changes are conspicuous in the early stage, with dermal edema and perivascular lymphocytic cuffing.^[3] In later stages, there is epidermal atrophy and loss of rete ridges.^[14] In addition, hair follicles and other appendages are absent.^[11] The area of striae is sharply demarcated from the surrounding skin by a densely packed area of thin, eosinophilic, collagen bundles, horizontal to the surface in a parallel fashion.^[11] There is an increase in the glycosaminoglycan content in striae and furthermore, the number of vertical fibrillin fibres adjacent to the dermal-epidermal junction and the elastin fibres on the papillary dermis are significantly reduced in striae compared to normal skin.^[14] On ultrastructural analysis, the dermal matrix of striae was found to be looser and more floccular.^[14]

CLINICAL FEATURES

The natural evolution of striae is for the red to purple, raised wavy lesions (striae rubra) to fade and leave white atrophic lesions with a wrinkly surface (striae alba).^[1] Striae are predominantly located on the arms, thighs, abdomen and lumbosacral area, but may involve other regions, including the face, and flexures in case of striae induced by Cushing's syndrome or steroid therapy.^[3] In pregnancy, they occur most commonly on abdomen and breasts.^[10] In obese patients striae are lighter, with less atrophy, and narrower than those with Cushing's syndrome.^[1] Very rarely, severe, extensive striae may ulcerate or tear.^[10] Striae distensae like lesions can present as scarring alopecia among children.^[16]

DIAGNOSIS

There are no known laboratory abnormalities unless the patient has Cushing's syndrome, in which case the serum and urinary steroid levels are increased.^[10] Striae have been mistakenly reported as child abuse.^[4] Linear focal elastosis can be considered in the differential diagnosis of striae, but here the lesions are yellow and palpable unlike striae.[17]

TREATMENT

Effective treatment of striae must be instituted during the active stage, well before the scarring process is complete.^[1] Various modalities of treatment have been tried. Weight loss by diet alone or a combination of diet and exercise do not change the degree of striae distensae.^[18] Topical tretinoin (0.1%) ameliorates striae and the improvement may persist for almost a year after discontinuation of therapy.^{[1],[19]} A study comparing topical 20% glycolic acid and 0.05% tretinoin versus 20% glycolic acid and 10% L-ascorbic acid, found that both regimens improved the appearance of striae alba.^[20]

Lasers of various types are used in treating striae and seem to be a promising mode of treatment. The 585nm pulsed dye laser has a moderate beneficial effect in the treatment of striae rubra^[21] and the 308-nm excimer laser is effective in treating striae alba.^[22] Intense pulsed light is also useful, and has minimal side effects.^[23] Short pulsed carbon dioxide laser has been tried as well.^[24] Lasers and light sources emitting UVB radiation have been shown to repigment striae distensae.^[25]

There is no widely accepted surgical procedure for improving the appearance of striae.^[1] Patients should be reassured that striae do fade with time.^[3]

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