Adolescent Striae

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Striae distensae, commonly referred to as stretch marks, have been recognized as sequelae of various physiologic states. These include, pregnancy (striae gravidarum), Cushing's syndrome, and changes in body habitus that accompany weightlifting and sudden weight gain or loss.[1-3] Acute infections, certain nutritional deficiency states, and toxic substances have also been implicated as possible etiologies.[2-4] The occurrence of stretch marks in a significant segment of the population as a result of puberty and adolescent growth spurts, however, is not as widely recognized. Although not physiologically significant in this one group, adolescent striae can have a pronounced impact on a teenager's body image and self-esteem. Parents also are at risk of social consequence, as their children's striae can often resemble whip or belt markings and have been known to prompt lengthy, traumatizing, and unwarranted investigations of possible child abuse.[5,6] These cases usually involve patients with hemorrhagic, lengthy, elevated stria rubra in the lumbosacral and gluteal regions. Adolescent striae warrant recognition and further investigation into their causes and possible treatments.

Clinical Manifestations

Adolescent striae are most commonly found over the thighs, gluteal region, and breasts in adolescent girls and over the lumbosacral region in adolescent boys.[1,5] The incidence has been estimated to be up to 70% in females and 40% in males. Most studies, however, report an overall incidence of approximately 25 to 35%.[6,7] Clinically, striae undergo a well recognized progression. Initially, they appear as violaceous or reddish pink, slightly raised, parallel lines that course in a wavy pattern. Termed stria rubra, they are generally 2 to 5 mm in width, with their length varying considerably based upon their location and tendency to coalesce as they progress.[1] During the earliest stages, patients may describe minor pruritus or irritation.[3] As striae progress, they gradually fade in color and lose their erythematous, hemorrhagic appearance. The end result is a white, atrophic, depressed lesion, often with a wrinkled surface. These striae alba, which evolve over months to years, are much less pronounced than their predecessors, fade with time, and are often subtle enough to be overlooked.

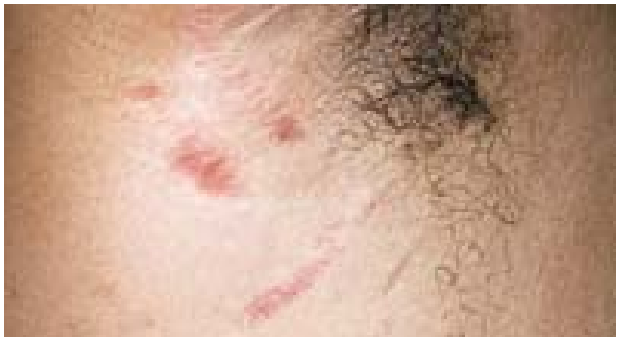

FIGURE 1. Axillary striae in an 18-year-old male.

Pathophysiology

Two main physiologic mechanisms are most likely to produce the unwelcome appearance of striae distensae. First, striae can appear as a result of mechanical shearing and stretching of the skin, as seen in rapid weight gain or loss, avid weightlifting, and pregnancy. [7] These lines appear in planes perpendicular to the direction of tension on the skin secondary to the rapid deposition of adipose tissue or muscular hypertrophy occurring beneath the dermal layer.[5,6,8] However, striae do not seem to develop in the clinical context of ascites and rapidly growing abdominal tumors.[4] Therefore, mechanical stretching is a contributing factor in striae formation, but may not be the primary insult.

The second group of conditions that can precede the appearance of striae share adrenocortical hyperac-

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tivity and/or cortisol excess as their underlying pathophysiologic mechanism. This subset includes acute infections, Cushing's syndrome, normal physiologic changes during puberty and pregnancy, exogenous administration of cortisol, nutritional disturbances, pituitary hyperactivity, and others.[1,6] Hypercortisolism results in excessive protein catabolism that compromises the integrity of the collagen and elastin fibers composing the extracellular matrix.[1]

Adolescent striae may in fact result from a combination of both of these basic mechanisms, as puberty is characterized by both rapid growth and stimulation of the hypothalamic-pituitary-adrenal axis. A genetic predisposition has been implicated in the development of all types of striae, including the atypical but possibly related hypertrophic striae of linear focal elastosis, an entity of excessive elastin deposition that is clinically very similar to the classic striae discussed here.[9] The role of mast cell degranulation and the activation of proteases in the breakdown of the extracellular matrix protein also have been considered.[3] This model suggests an inflammatory response to an unknown stimulus, which results in macrophage stimulation and mast cell degranulation. These inflammatory mediators incite the process of elastolysis that culminates in the appearance of clinical striae.

Histopathology

Histologically, striae 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 extracellular matrix in the dermis.[7] Fibroblast depletion has been implicated as a causative factor in these findings. Finally, the histologic picture shows a dilatation of blood vessels in the dermis in the regions of collagen bundle separation.

Therapeutic Modalities

Striae distensae are often of primarily cosmetic concern to patients. As these stretch marks often tend to regress spontaneously with time, in most instances treatment is not recommended. However, numerous methods have succeeded in improving the appearance of striae or at least in halting their clinical progression. Topical tretinoin cream (0.1%) has been shown to decrease the length and width of striae if applied during the early stages of striae evolution. This beneficial result is due possibly to the agent's antagonistic effect on extracellular matrix degrading enzymes such as collagenase and gelatinase.[10] Other topical therapeutic regimens, including 0.05%tretinoin/20%glycolic acid and 10%L-ascorbic acid/20%glycolic acid, may also be effective.[11] In one study, posttreatment groups showed subjective improvement as determined by both patients and physicians, as well as histologic analysis.[10] The latter included an increase in dermal elastic content, an increase in epidermal thickness, and papillary dermal thickening to levels approaching those of normal skin. These changes were seen in areas of mature stretch marks as well as in early lesions.

The 585 nm flashlamp pulsed dye laser has been reported in some studies to show minimal improvement over topical regimens.[12] The cost and associated risk of pigmentary alterations in patients of darker skin, however, have limited the use of lasers in the treatment of striae. Future advances in laser technology may yield more favorable outcomes and make lasers a more useful alternative treatment modality for insistent patients. Current approaches should focus on observation rather than aggressive treatment of these often temporary markings.

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