Acta Biomed 2020; Vol. 91, N. 1: 176-181 DOI: 10.23750/abm.v91i1.9248

Review

Striae distensae in adolescents: a mini review

Heba Elsedfy
Pediatrics Department, Ain Shams University, Cairo, Egypt

Summary. Striae distensae or stretch marks are mainly a cosmetic concern. They commonly occur in adolescence and in pregnant women. Although, generally more common in females; physiological striae atrophicae of adolescence are more common in males. The pathophysiology is multifactorial with mechanical stretching of the skin being the most important. Despite of an abundance of treatment modalities none is 100% effective. (www.actabiomedica.it)

Key words: adolescents, striae distensae, stretch marks

Introduction

Striae distensae (SD) were first described in the medical literature by Troisier and Menetrier in 1829. In 1936, Nardelli named the lesions striae atrophicae (1).

SD or stretch marks result from dermal scarring and epidermal atrophy. The epidermis is thin with loss of dermal papillae and rete ridges and the dermis shows a decrease in extracellular matrix (ECM) components; collagen, fibronectin, fibrillin and elastin (2-4).

The dermis is composed of an interwoven matrix of collagen and elastin (3). In normal skin collagen fibrils are organized in densely packed bundles that provide support to the skin (5). Elastic fibres allow the skin to stretch and return to its original shape (6). With development of SD, the collagen bundles separate and collagen fibrils fail to form bundles. Elastic fibres are disrupted and tropoelastin (soluble elastin)-rich fibrils unable to organize into normal-appearing elastic fibres form (5).

Stretch marks are caused by excessive mechanical stretching of skin to the point of rupturing dermal elastic fibers with local fibroblasts unable to adequately repair or replace ECM components (4). Aberrant fibroblast function may be responsible for development of SD as fibroblasts from striae expressed significantly less fibronectin and both type I and type III procollagen (7).

Clinically, the condition passes through two stages: an initial raised erythematous, inflammatory stage (striae rubrae; SR) and a white, depressed, finely wrinkled second stage (striae albae; SA) (8).

Prevalence, etiology and risk factors

In the adolescent population reported prevalence ranges from 6% to 86%. In adolescent males the buttocks, lower back and knees are usually affected while in females the buttocks, thighs and calves are more often involved (9).

Three main theories underlying the development of SD are described: mechanical stretching of the skin, hormonal changes and an innate structural disturbance of the skin (9). SD are postulated to result from an initial inflammatory reaction that destroys collagen and elastic fibers, followed by the regeneration of collagen and elastic fibers in the direction imposed by mechanical forces (10).

Genetic factors may be operative as a familial form of striae was described by McKusick (11). Also striae in monozygotic twins and striae in syndromes as Ehlers-Danlos, Marfan and ectodermal dysplasia have

been reported (12). Results of a genome wide association analysis support the hypothesis that variations in the elastic fiber component of the skin extracellular matrix contribute to the development of stretch marks (13).

Physiological striae atrophicae of adolescence occurs mainly in healthy, nonobese individuals at around puberty in association with the adolescent growth spurt (14). It commonly occurs in the gluteal region, breasts, thighs, lower abdomen and back (15). SD associated with pubertal growth spurt becomes less conspicuous with time and has excellent prognosis as compared to other SD (16).

In adolescents, high BMI, obesity during childhood, and facial seborrhea correlate positively with development of SD. Striae have also been observed in conjunction with Cushing syndrome and exogenous steroid use (17). To investigate the role of hormones in the development of SD, the expression of estrogen receptor (ER), androgen receptor (AR) and glucocorticoid receptor (GR) in SD was studied. Cordeiro et al. (18) found 2.2-fold more ER, 1.8-fold more AR and 1.7-fold more GR in SD samples compared to normal skin (9). These results were supported by other studies, however, one study found reduced ER β expression in SD lesions and perilesional normal skin compared to a control group despite the presence of increased expression of both AR and GR.

Hormone receptor expression is increased under certain conditions suggesting that regions undergoing greater mechanical stretching of the skin may express more hormone receptor activity thus influencing the metabolism of the extracellular matrix, causing SD formation. This could be the link between hormonal and mechanical theories underlying the development of SD (19).

ACTH has a catabolic effect on fibroblasts with a resulting decrease of mucopolysaccharides in collagen tissue. Elevated serum levels of steroid hormones (or of their metabolites) have been found in people with striae (1). Striae formation resulting from the use of topical steroids seems to be related to the use of the more potent preparations. Adolescents and young adults seem to be particularly prone to this form of striae formation (20).

Evaluation of striae distensae

A numerical scoring system for the severity of striae was devised for the evaluation of striae gravidarum. The number of striae present at different sites (Figure 1) and the degree of erythema were evaluated. At each site striae were scored up to a maximum of six; 0-3 for number of striae present and also 0-3 for the degree of erythema. The number of striae was recorded as: no striae, 0; <5 striae, 1; 5-10 striae, 2; and >10 striae, 3. The degree of erythema was recorded as: no erythema, 0; mild erythema (light red or pink) (Figure 2), 1; marked erythema (dark red), 2 (Figure 3); and violaceous erythema (purple), 3 (Figure 4). The following sites are evaluated: abdomen, hips, breasts, thigh/buttocks with a maximum score of 24 (21).

Management of striae distensae

Many therapeutic modalities are available but none can completely eradicate SD: laser, light therapy, acid peel treatments, collagen injection, laser lipolysis, radiofrequency techniques and microdermabrasion (22). The majority of treatments aim to increase colla-

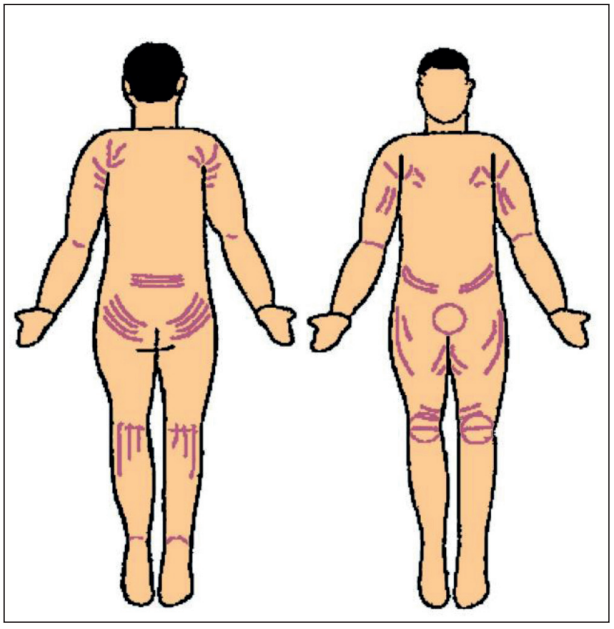

Figure 1. Typical distribution of striae distensae (from: Cho S et al. J Eur Acad Dermatol Venereol. 2006;20:1108-13; modified)

Figure 2. Striae distensae type 1

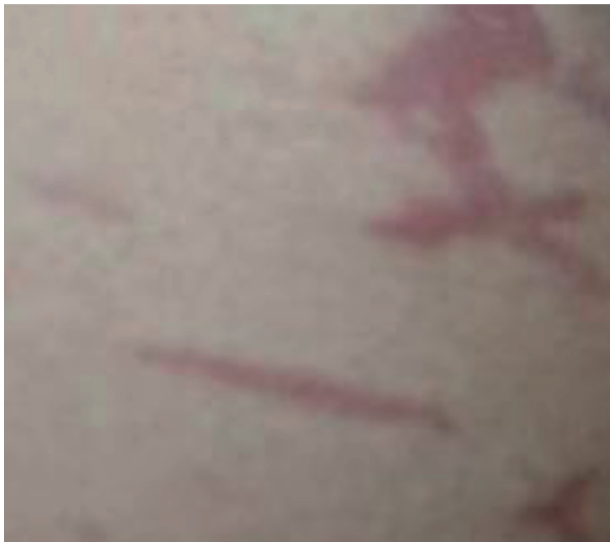

Figure 3. Striae distensae type 2

Figure 4. Striae distensae type 3

gen production, reduce erythema, or increase pigmentation (23).

1. **Enhanced collagen production**

- Tretinoin and retinoic acid are believed to act by stimulation of fibroblasts leading to increase tissue collagen I levels. Side effects include transient erythema, scaling and itching or a burning sensation (24, 25).
- Centella asiatica is a medicinal herb thought to increase the production of collagen and elastic fibers (26). No side effects were observed with its use, however, when combined with boswellic acid which has an anti-inflammatory action, pruritus was reported (23).
- Hyaluronic acid is also suggested to stimulate fibroblast activity and the production of collagen (22).
- Chemical peels are divided into superficial, medium-depth and deep subtypes based on the depth of their penetration (27). For striae distensae 20% glycolic acid and trichloroacetic acid (TCA) 10-35% are also reported to stimulate collagen production by fibroblasts (7). Superficial peels target the epidermis and the epidermal-dermal interface causing partial or complete necrosis. They exfoliate the skin from the stratum corneum down to the papillary dermis at a depth of 60 μm (27).
- Aluminum oxide microdermabrasion induces epidermal signal transduction pathways that are associated with remodeling of the dermal matrix. It produces epidermal and dermal changes through superficial wounding (28).
- Bipolar radiofrequency (RF) devices generate heat in response to poor electrical conductance according to Ohm's law (heat generation is di-

rectly correlated with tissue resistance). The heat generated is responsible for the partial denaturation of pre-existing elastic fibers and collagen bundles. Initial collagen denaturation causes immediate tissue contraction; subsequent neocollagenesis further tightens the dermal tissue and reduces striae. Autologous platelet-rich plasma (PRP) can be injected using the needle electrode of the intradermal RF device as the delivery route. At sites of tissue damage, platelets are the first cells to arrive and through the release of growth factors from their a-granules act on endothelial cells, erythrocytes, and collagen thus aiding in the healing of localized chronic inflammation believed to be a factor in the etiology of striae distensae (29).

- Fractional lasers (FL): produce small columns of thermal injury to the skin, which are known as microthermal zones (MTZs). Some cause nonablative (leave a functionally and histologically intact stratum corneum) dermal injuries only; whereas, others are associated with ablative changes in the skin, causing both epidermal and dermal injuries. MTZs also vary in their diameter and depth. Once injured, the skin begins a very rapid process of repair. The rapid healing process is made possible through the help of the surrounding normal or untreated skin (30). When compared with nonablative lasers, ablative lasers are less well-tolerated and produce inconsistent results (23). A similar technique is microneedling which is a minimally invasive procedure that uses fine needles to puncture the epidermis. The microwounds created stimulate the release of growth factors and induce collagen production. The epidermis remains relatively intact, therefore helping to limit adverse events (31).
- Galvanopuncture, a needling procedure that uses continuous direct microcurrent as an alternative to induce a local inflammatory process intended to repair the affected tissue. The microamperage galvanic current reaches intensities between 50 and 200 μA. It induces modification of the vasculature with dilation of blood vessels, tissue edema, and associated redness. The outcomes are angiogenesis, cell proliferation, as well as reorganization of collagen bundles. The best outcome was observed in subjects with darker skin, which is usually the most difficult skin type (32).

2. Reduction of vascularity

- Lasers with wavelengths of 585 to 595 nm are used, due to a high absorption by haemoglobin and decreased absorption by the competitive chromophore melanin, thereby reducing injury to the epidermis. Longer wavelength lasers (alexandrite laser 755 nm, Nd:YAG laser 1064 nm) have been developed to target oxy- and deoxyhaemoglobin which have the advantage of deeper tissue penetration (33). The treatment of erythematous striae using the 1064-nm long-pulsed Nd:YAG laser demonstrated clinical improvement of such lesions, probably due to the laser's affinity toward the vascular target present in the striae. The absorption of the laser by oxyhemoglobin, leads to an improvement in the redness. In addition, like other luminous sources, the long-pulsed Nd:YAG laser also induces the formation of new collagen (34).

3. Increase melanin production

- A targeted narrow band UVB/UVA1 therapy caused 51% improvement in SA pigmentation after weekly (maximum 10 weeks) phototherapy sessions. Transient hyperpigmentation of striae was seen in almost half the subjects as an adverse event. Skin biopsy failed to show any effect on collagen remodeling, thus limiting its efficacy only for repigmentation of SA (35).
- The 308 nm xenon chloride (XeCl) excimer laser has a wavelength close to that of traditional narrow band ultraviolet B (UVB) light. It causes temporary repigmentation and improvement of leukoderma in SD. Post laser biopsies showed greater melanin content and hypertrophy of the melanocytes, although it failed to show any improvement in skin atrophy (36).

Conclusion

SD or stretch marks are a relatively common skin condition that occurs frequently in association with the adolescent growth spurt and pregnancy. Their etiology is still not completely established. The most promising treatment modality is laser therapy.

Conflict of interest: Each author declares that he or she has no commercial associations (e.g. consultancies, stock ownership, equity interest, patent/licensing arrangement etc.) that might pose a conflict of interest in connection with the submitted article

References

1. Rogalski C, Haustein UF, Glander HJ, Paasch U. Extensive striae distensae as a result of topical corticosteroid therapy in psoriasis vulgaris. Acta Derm Venereol. 2003;83:54-55.
2. Stamatas GN, Lopes-DaCunha A, Nkengne A, Bertin C. Biophysical properties of Striae Distensae evaluated in vivo using non-invasive assays. Skin Res Technol. 2015;21:254-258.
3. Tong PL, Qin J, Cooper CL, et al. A quantitative approach to histopathological dissection of elastin-related disorders using multiphoton microscopy. Br J Dermatol. 2013;169:869-879.
4. Mitts TF, Jimenez F, Hinek A. Skin biopsy analysis reveals predisposition to stretch mark formation. Aesthet Surg J. 2005;25:593-600.
5. Wang F, Calderone K, Do TT, et al. Severe disruption and disorganization of dermal collagen fibrils in early striae gravidarum. Br J Dermatol. 2018;178:749-760.
6. Wang F, Calderone K, Smith NR, et al. Marked disruption and aberrant regulation of elastic fibres in early striae gravidarum. Br J Dermatol. 2015;173:1420-1430.
7. Keen MA. Striae distensae: What's new at the horizon? BJMP 2016;9:a919
8. Cho S, Park ES, Lee DH, Li K, Chung JH. Clinical features and risk factors for striae distensae in Korean adolescents. J Eur Acad Dermatol Venereol. 2006;20:1108-1113.
9. Al-Himdani S, Ud-Din S, Gilmore S, Bayat A. Striae distensae: a comprehensive review and evidence-based evaluation of prophylaxis and treatment. Br J Dermatol. 2014;170:527-547.
10. Cordeiro RC, Zecchin KG, de Moraes AM. Expression of estrogen, androgen, and glucocorticoid receptors in recent striae distensae. Int J Dermatol. 2010;49:30-32.
11. McKusick VA. Transverse striae distensae in the lumbar area in father and two sons. Birth Defects Orig Artic Ser. 1971;7:260-261.
12. Cordeiro RC, de Moraes AM. Striae Distensae: Physiopathology. Surg Cosmet Dermatol. 2009;1:137-140.
13. Tung JY, Kiefer AK, Mullins M, Francke U, Eriksson N. Genome-wide association analysis implicates elastic microfibrils in the development of nonsyndromic striae distensae. J Invest Dermatol. 2013;133:2628-2631.
14. Leung AK, Barankin B. Physiological striae atrophicae of adolescence with involvement of the upper back. Case Rep Pediatr. 2013;2013:386094. doi:10.1155/2013/386094
15. Elshimy N, Gandhi A. A teenager with lumbar striae distensae (when a bruise is not a bruise). BMJ Case Rep. 2013;2013:bcr2013201962. Published 2013 Dec 18. doi:10.1136/bcr-2013-201962.
16. Lokhande AJ, Mysore V. Striae distensae treatment review and update. Indian Dermatol Online J. 2019;10:380-395.
17. Boozalis E, Grossberg AL, Puttgen KB, Heath CR, Cohen BA. Demographic characteristics of teenage boys with horizontal striae distensae of the lower back. Pediatr Dermatol. 2018;35:59-63.
18. Youssef SES, El-Khateeb EA, Aly DG, Moussa MH. Striae distensae: Immunohistochemical assessment of hormone receptors in multigravida and nulligravida. J Cosmet Dermatol. 2017;16:279-286.
19. Valente DS, Zanella RK, Doncatto LF, Padoin AV. Incidence and risk factors of Striae Distensae following breast augmentation surgery: a cohort study. PLoS One. 2014;9(5):e97493. doi: 10.1371/journal.pone.0097493. eCollection 2014.
20. Adam JE, Craig G. Striae and their relation to topical steroid therapy. Can Med Assoc J. 1965;92:289-291.
21. Atwal GS, Manku LK, Griffiths CE, Polson DW. Striae gravidarum in primiparae. Br J Dermatol. 2006;155:965-969.
22. Ud-Din S, McGeorge D, Bayat A. Topical management of striae distensae (stretch marks): prevention and therapy of striae rubrae and albae. J Eur Acad Dermatol Venereol. 2016;30:211-222.
23. Hague A, Bayat A. Therapeutic targets in the management of striae distensae: A systematic review. J Am Acad Dermatol. 2017;77:559-568.
24. Lokhande AJ, Mysore V. Striae Distensae Treatment Review and Update. Indian Dermatol Online J. 2019;10:380-395.
25. Liu L, Ma H, Li Y. Interventions for the treatment of stretch marks: a systematic review. Cutis. 2014;94:66-72.
26. Farahnik B, Park K, Kroumpouzos G, Murase J. Striae gravidarum: Risk factors, prevention, and management. Int J Womens Dermatol. 2016;3:77-85.
27. O'Connor AA, Lowe PM, Shumack S, Lim AC. Chemical peels: A review of current practice. Australas J Dermatol. 2018;59:171-181.
28. Karia UK, Padhiar BB, Shah BJ. Evaluation of Various Therapeutic measures in striae rubra. J Cutan Aesthet Surg. 2016;9:101-105.
29. Kim IS, Park KY, Kim BJ, Kim MN, Kim CW, Kim SE. Efficacy of intradermal radiofrequency combined with autologous platelet-rich plasma in striae distensae: a pilot study. Int J Dermatol. 2012;51:1253-1258.
30. Gold MH. Update on fractional laser technology. J Clin Aesthet Dermatol. 2010;3:42-50.

31. Hou A, Cohen B, Haimovic A, Elbuluk N. Microneedling: A Comprehensive Review. Dermatol Surg. 2017;43:321-339.
32. Bitencourt S, Lunardelli A, Amaral RH, Dias HB, Boschi ES, de Oliveira JR. Safety and patient subjective efficacy of using galvanopuncture for the treatment of striae distensae. J Cosmet Dermatol. 2016;15:393-398.
33. Faurschou A, Olesen AB, Leonardi-Bee J, Haedersdal M. Lasers or light sources for treating port-wine stains. Cochrane Database Syst Rev. 2011 Nov 9;(11):CD007152. doi: 10.1002/14651858.CD007152.pub2.
34. Wollina U, Goldman A. Management of stretch marks (with a focus on striae rubrae). J Cutan Aesthet Surg. 2017;10:124-129.
35. Sadick NS, Magro C, Hoenig A. Prospective clinical and histological study to evaluate the efficacy and safety of a targeted high-intensity narrow band UVB/UVA1 therapy for striae alba. J Cosmet Laser Ther. 2007;9:79-83.
36. Elsaie ML, Baumann LS, Elsaaiee LT. Striae distensae (stretch marks) and different modalities of therapy: an update. Dermatol Surg. 2009;35:563-573.

Received: 4 January 2020
Accepted: 5 February 2020
Correspondence:
Heba Elsedfy
Pediatrics Department
Ain Shams University - Cairo (Egypt)
Tel. +201005189166
E-mail: hebased@yahoo.com