УДК 616.5-003.92-085](048.8)
DOI: http://doi.org/10.30978/UJDVK2021-2-68
ISSN 1727-5741 (Print), ISSN 2522-1035 (Online)

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Stretch marks: a review

Stretch marks (SM) feature visible linear scars developing on the body in the areas of excessive stretching of the skin. This is a benign skin condition, but because of their aesthetic implications, they can cause distress among their sufferers. SM show high prevalence, probably more than 50 % among pregnant women, and 50 to 90 % in the general population. SM affect more likely certain ethnic groups, especially dark-skinned patients. During pregnancy, risk factors include family history, but also important weight gain whilst more generally elevated body mass index also constitutes a risk factor. Clinically, the initial erythematous and violaceous lesions referred to as striae rubrae fade into wrinkled, hypo-pigmented, atrophic scar-like marks named striae albae. Four main theories support SM formation: 1) mechanical stretching of the skin; 2) hormonal changes; 3) an innate structural disturbance of the integument; 4) genetic predispositions. Histologically various abnormalities of collagen and elastic fibres are described at dermal level. The treatment of SM is always deceptive. Topical treatments are the commonest, among them Centella asiatica or hyaluronic acid creams, almond oil, topical retinoids, cocoa butter or olive oil are the most popular. Chemical peels may also be used with limited success. Various office procedures may also be performed, such as microdermabrasion, radiofrequency, laser/light therapy, platelet-rich plasma and others, but the results are often limited and deceptive for both the patient and the dermatologist. More remains to be done about the study of this frequent dermatological disorder.

Key words

Stretch marks, striae, striae albae, striae rubrae, collagen, elastin, fibulin, treatment.

1. INTRODUCTION

Striae distensae (SD), striae, stretch marks, striae atrophicans are synonyms to design visible linear scars which develop in areas of dermal damage as a result of excessive stretching of the skin. In case of occurring during pregnancy, they are named striae gravidarum (SG). They are a common benign skin condition but are often a significant source of distress to those affected. The first histologic description of SD appeared in the medical literature in 1889 [1].

2. EPIDEMIOLOGY

SD usually occur in adolescence, pregnancy and obesity [2]. In adolescents, the prevalence ranges between 6 and 86 %, whilst during pregnancy it occurs in 43 to 88 % of women. Amongst obese individuals, of BMI 27—51, the prevalence is reported to be 43 % [3]. In adult non-pregnant women, the average prevalence is 35 % and in adult males it is reported as 11 % [3]. It has been reported in the literature that the prevalence in the general population ranges from 50 to 90 % [4]. Interracial differences have been observed in the severity of SD. Of forty-eight women evaluated, Black African women were more severely affected than Caucasians within the same geographical region [5]. In another study in Australia, the prevalence in Chinese women was lower than in Malay and Indians (69 *vs.* 85 %) [6].

3. RISK FACTORS

In a study on 161 women who had given birth, 48.3 % of women with SD *vs.* 19.4 % without SD reported mothers with SD, and 47 % of women with SD *vs.* 18.1 % without SD reported relatives with SD [7]. 81 % of women with SD *vs.* 30.5 % without SD reported an history of SD. 47 % of women with SD vs. 17% without SD were non-white, confirming the results in previously mentioned studies [7]. In 191 Turkish primiparous women, family history, maternal weight gain and maternal age were found to be significantly associated with SD [8]. The group at higher risk of developing striae is younger women with maternal obesity who have a positive family history of SD. Among 164 Brazilian primiparous women who had had a single foetus pregnancy, 59.8 % developed striae during pregnancy. SD were more frequently observed in younger women, in those who gained more weight during pregnancy and/or those who had babies with higher birth weight [9]. In 80 female students

(40 with SD and 40 without SD), history of contraceptives intake and a family history of striae were risk factors of SD occurrence, while weight loss can reduce the risk of these lesions [10]. Among 112 primiparous women delivering at a private teaching hospital in Beirut (Lebanon), 60 % had developed SD. Women who developed SD were significantly younger and had gained significantly more weight during pregnancy. Birthweight, gestational age at delivery, and family history of SG were associated with moderate/severe SD [11].

4. CLINICAL FEATURES

Two clinically and histopathologically recognisable forms of SD have been described: striae rubrae SR) and striae albae (SA) [12]. Although they appear after each other, without transition, they constitute two very different clinical entities. The initial erythematous and violaceous appearing lesions are referred to as striae rubrae (SR) (Fig. 1). These fade into wrinkled, hypo-pigmented, atrophic scar-like marks named striae albae (SA) (Fig. 2). The latter of these has been described as a permanent form of SD [13]. Clinically SD appear as multiple, symmetric, well defined, irregularly linear, red to pale coloured (depending upon the stage) atrophic scars which follows the lines of cleavage and lies parallel to the skin surface [14]. During puberty, SD are present over thighs, buttocks and breasts in girls. In boys, they often develop over lumbosacral region and outer aspect of the thighs. Striae gravidarum (SG) are commonly seen over abdomen, breast and thighs in third Trimester of pregnancy.

5. AETIOLOGY

Four main theories relating to SD formation are described: Mechanical stretching of the skin, hormonal changes, an innate structural disturbance of the integument and genetic predispositions.

5.1. Mechanical stretching of the skin

This cause was postulated due to the perpendicularity of SD to the direction of the skin (Fig. 3).

According to Shuster [15] 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.

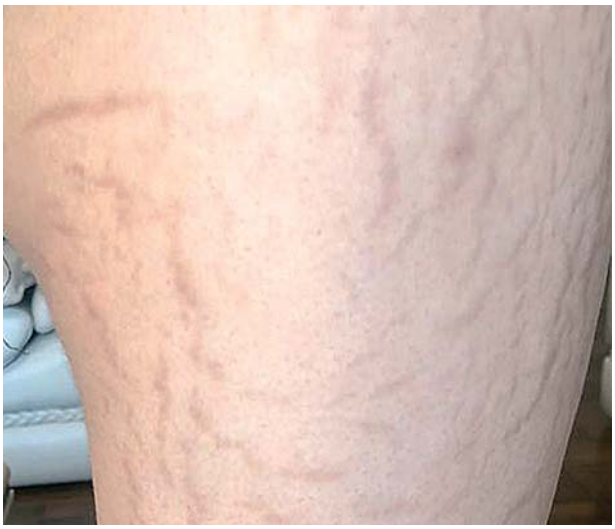

Fig. 1. **Striae rubrae on the tight**

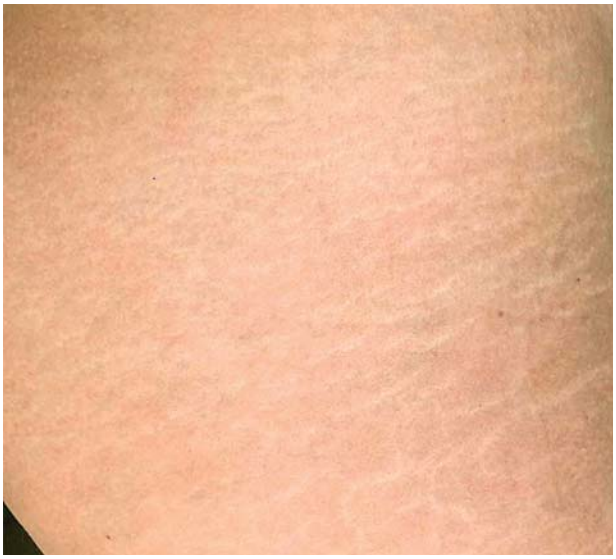

Fig. 2. **Striae albae on the hip**

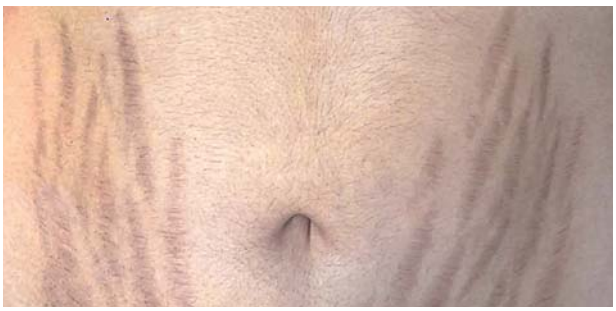

Fig. 3. **Striae rubrae on the abdomen. Note their orientation perpendicular to the direction of the skin**

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 crosslinked 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. [16] suggest that striae result from disruption of elastic fibres 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. Henry et al. [17] 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. [18] 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. [19] 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.

5.2. Hormonal changes

The fact that SD are mainly related to pregnancy, puberty, and the use of corticosteroids suggest the involvement of hormonal factors in their occurrence.

Adrenocorticotrophic hormone (ACTH) and cortisol are thought to promote fibroblast activity, leading to increased protein catabolism and thus alterations to collagen and elastin fibres [20]. A significant increase in the expression of androgen, glucocorticoid, and especially oestrogen receptors was observed in skin with early SD, compared to skin without cutaneous striae [21]. 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, SD lesions demonstrated in another work a significant increase in the expression of androgen and glucocorticoid receptors and a declined expression of oestrogen receptors, indicating their involvement in the development of early SD [22]. In another study, the intensity of ER-β expression was significantly related with the site of the stretch marks. The abdominal striae showed stronger staining and the buttocks showed the least expression of ER-β, suggesting that using hormonal replacement therapy on the skin showing early stretch marks can be helpful as prophylactic and therapeutic modality for stretch marks [23].

5.3. Innate structural disturbance of the integument

Lower serum relaxin levels were demonstrated in pregnant women with SD compared to those without SD at 36 weeks gestation [24]. The connective tissue in skin type with less relaxin would be expected to be less lax and thus at greater risk of structural disruption of the elastic fibre network during stretching than more lax skin with greater relaxin content.

5.4. Genetic predispositions

In a retrospective study genetic factors, such as family history, personal background and ethnicity were found as important predictors for the onset of SD [4].

The onset of striae rubrae was reported 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 presented no haematological or endocrinal changes [25].

Genetic factors may be related with the presence of striae, and may be associated with syndromes such as Ehlers Danlos, Marfan, ectodermal dysplasia, and autosomal dominant familial striae distensae [26].

6. HISTOPATHOGENESIS

In an interesting study [27], skin samples of recently developed, erythematous abdominal SG from pregnant women were taken to examine the organization of collagen fibrils. In control (hip) and stretched, normal-appearing perilesional abdominal skin, dermal collagen fibrils were organized as tightly packed, interwoven bundles. In SG, collagen bundles appeared markedly separated, especially in the midto-deep dermis. In the spaces separating

bundles, loosely packed wavy collagen fibrils lacking organization as bundles were present. These disorganized fibrils persisted into the postpartum period and failed to form densely packed bundles. The authors concluded that early SG display marked separation of collagen bundles and emergence of disorganized collagen fibrils that fail to form bundles. These alterations may reflect ineffective repair of collagen bundles disrupted by intense skin stretching. Persistent disruption of the collagenous extracellular matrix likely promotes formation and atrophy of SG [27]. In another study [28], the lesions of nine patients with early SD during puberty were examined by light and electron microscopy. Specific changes were seen in very early stage SD, and in clinically uninvolved skin 0.5 to 3 cm remote from the edge of the long axis of the SD lesions. Sequential changes of elastolysis accompanied by mast cell degranulation appeared first, followed by an influx of activated macrophages that enveloped fragmented elastic fibres, signing the occurrence of inflammation. Skin biopsies from normal subjects (NS), stretch-marked skin (SM), and normal-looking skin from patients with stretch marks (NL) were analysed by histochemistry [29]. It was found that NL skin contained less DNA, protein, and elastin than NS skin (–16 %, –36 %, –44 %, respectively) and that such deficiencies were more profound in SM skin (–55 %, –64 %, –80 %, respectively). Both NL- and SM-derived cells had slower than normal outgrowth of their fibroblasts, which also demonstrated low migration and proliferation rates, and produced less elastin, fibrillin 1, collagen 1, and fibronectin than NS-derived cells in primary cultures [29]. The molecular differences between skin with and without stretch marks (rubra [SR] and alba [SA]) of female volunteers were analysed using DNA microarray analyses of cutaneous biopsies (2 mm) and in vivo confocal Raman spectroscopy of selected buttock regions [30]. The main differences observed when comparing skin with and without stretch marks were at depths between 75 and 95 μm, corresponding to the dermal epidermal junction and dermis regions and showing differences between normal skin and stretched skin regarding collagen, collagen hydration, and elastin fibres [30]. Another group of investigators demonstrated that fibroblasts from striae exhibit increased pro-fibrotic and decreased anti-fibrotic signalling pathways [31].

A potential role of fibulin

Wang examined the elastic fibre network of skin samples of newly developed, erythematous abdominal SG by Verhoeff staining and immunofluorescence [32].

The normal elastic fibre network appeared markedly disrupted in SG, compared with perilesional abdominal skin or control (normal-appearing hip skin). This disruption was accompanied by the emergence of short, disorganized, thin, thread-like 'fibrils', which were observed prominently in the mid-to-deep dermis. These fibrils were rich in tropoelastin. The emergence of these fibrils was accompanied by increased gene expression of tropoelastin and fibrillin-1, but not other elastic fibre components, including fibrillin-2 and fibulin-1, -2 or -5 [32].

During the elastogenesis process, the tropoelastin monomer is synthesized by the fibroblast on the rough endoplasmic reticulum and undergoes little intracellular posttranslational modification [33]. During this phase, the elastin-binding protein associates with tropoelastin monomers to become a complex form before being released on the cell surface [34]. This association of elastin binding protein and tropoelastin monomers protects the insoluble elastin from proteolysis and gives it extraordinary stability [35]. During self-assembly of the tropoelastin, fibulin-5 and fibulin-4 have the potential to induce elastic fibre formation and also promote the coacervation process [36, 37]. Fibulin-5 thus serves as an adaptor molecule between monomeric elastin and the matrix scaffold to aid in elastic fibre assembly [38].

Interestingly, patients with Fibulin-5 mutations presented mainly with cutis laxa and showed disruption of elastic fibres in skin [39]. Additionally, these mutants showed decreased affinity for tropoelastin [40].

7. TREATMENT

7.1. Topical treatments

Topical treatments are the commonest in SD, they being SR or SA. In a report aimed to assess the evidence for the use of topicals in SD and to propose a structured approach in managing SD, the results showed that there are few studies (n = 11) which investigate the efficacy of topicals in management of SD [41]. The majority of them are of poor quality and the results are always deceptive.

Centella asiatica

This plant found in South-Asia is widely used in topical treatments for striae. While the mechanism of action of Centella is unclear, this ingredient may stimulate fibroblasts and inhibit glucocorticoid activity [42]. Topical application accelerates wound healing and improves the tensile strength of scars [43]. In a randomized, double-blind, placebo-controlled trial involving 80 women, Mallol et al.

showed that daily massage with a cream containing Centella to the abdomen, breasts, buttocks and hips from the 12th week of pregnancy until delivery was associated with decreased SG incidence (56 *vs.* 34 % for control) [44]. These results are controversial, as in a randomized, double-blind, placebo-controlled trial, García Hernández et al. enrolled 183 women, who applied the cream containing Centella triterpenes twice a day to the abdomen, thighs, hips, buttocks and breasts starting in the 12th week of pregnancy [45]. The incidence of SG was similar between the treatment and placebo groups (38 *vs.* 33 %, respectively). As Centella is often combined with others ingredients, well-designed controlled studies are required to determine the specific efficacy of Centella for SG prevention [42].

Almond oil

Almond oil is traditionally used in the prevention of striae during pregnancy, especially in Mediterranean countries. In a nonrandomized, comparative study of 141 women with no history of SG, Timur Tashan et al. [46] divided subjects into three groups (group 1 bitter almond oil with a 15-min massage every other day, group 2 bitter almond oil without massage and group 3 no treatment). The development of abdominal SG was significantly reduced in the oil plus massage group (16 of 47), compared with oil alone (31 of 48) and the control group (33 of 46), suggesting that massage, but not almond oil could be beneficial for the prevention of striae during pregnancy. In a more recent randomized controlled trial [47], in 150 nulliparous women in the second trimester, no difference was observed in the incidence or severity of abdominal SG between no intervention, olive oil without massage, or a cream containing almond oil without massage. Lesions developed in 60, 64 and 64 % of participants, respectively [47].

Hyaluronic acid (HA)

Hyaluronic acid is thought to improve tensile resistance to mechanical forces and counteract atrophy by stimulating fibroblast activity and collagen production to increase skin volume [48]. In a double-blind study involving pregnant women, de Buman et al. demonstrated that a cream containing HA reduced the incidence of SG, compared with placebo (three of 30 *vs.* 11 of 30 subjects, respectively) [49]. Wierrani et al. enrolled 50 pregnant women at 20 weeks' gestation and found that SG developed in 29 % of women who applied a similar to the abdomen, thighs and breasts with massage, compared with 62 % of control subjects, who performed no massage or topical application [50]. However, this study was not placebo controlled and was poorly randomized.

Tretinoin

Topical tretinoin partially restores decreased collagen formation in photoaged skin by stimulating the synthetic activity of dermal fibroblasts. As damage of structural proteins such as collagen may also occur in SG, topical tretinoin has been studied for the treatment of this condition, but in this context, its exact mechanisms remain unclear [42]. Studies report conflicting results. In an observational study by Elson, tretinoin led to 'significant improvement' of SD from a variety of causes in 15 of 16 patients [51]. In a double-blind, placebo-controlled study by Pribanich et al., tretinoin cream 0.025 % was applied daily for 7 months to abdominal SG at various stages of development, with six subjects assigned to treatment and five to placebo [52]. No improvement occurred with treatment compared with placebo. More recently, in a double-blind, randomized controlled study by Kang et al., 22 patients with early (erythematous) SD from a variety of causes, including pregnancy, applied a higher strength of tretinoin cream (0.1 %) or vehicle nightly [53]. After 6 months, eight of 10 subjects in the tretinoin group demonstrated marked or definite improvement of lesions, compared with one of 12 subjects in the vehicle group. Lesions treated with tretinoin showed a mean decrease in length and width by 14 and 8 %, respectively, whereas lesions treated with vehicle increased by 10 and 24 %, respectively [53].

Finally, in an open-label, prospective study by Rangel et al., tretinoin 0.1 % cream was applied nightly to pregnancy-related abdominal SG starting 1 week after delivery [54]. After 12 weeks of application, 16 of 20 subjects demonstrated moderate to marked improvement, and the mean length and width of target lesions decreased by 20 and 23 %, respectively. However, topical tretinoin is a pregnancy category C drug, and its use during pregnancy and lactation is not generally recommended.

Cocoa butter

Cocoa butter is a natural fat derived from cocoa beans. Cocoa butter has emollient properties, although its mechanism of action is not known. It was sometimes suggested applying topical cocoa butter before, during and after pregnancy to prevent SG development. In a double-blind, randomized, placebo-controlled trial, Osman et al. studied 175 nulliparous pregnant women, 91 of whom applied a lotion containing cocoa butter and vitamin E daily to the abdomen, breasts and thighs from 12–18 weeks' gestation until delivery [55]. The

remaining subjects applied a placebo lotion lacking cocoa butter and vitamin E. There was no difference in SG development between the intervention and control groups. If SG developed, the severity of lesions was similar between the two groups [55]. Buchanan et al. conducted a similar randomized, double-blind, placebocontrolled study in 300 pregnant Afro-Caribbean women with no previous history of SD [56]. Of these subjects, 150 applied a cream containing 25 % cocoa butter and vitamin E oil daily from 16 weeks' gestation to delivery, while the remaining applied a placebo cream. No statistically significant difference was noticed in the development of SG between the treatment and placebo groups, with lesions developing in 44 *vs.* 55 % of subjects, respectively.

Olive oil

The use of olive oil for preventing SG is popular. Early research produced conflicting results. In an observational study of 116 primagravid women, Poidevin noted that 36 of 50 pregnant women who applied olive oil nightly developed abdominal SG, compared with 36 of 66 pregnant women who did not [57], suggesting that olive oil was not effective as prophylaxis for SG development. In another early observational study, Davey found that the incidence of SG was reduced by up to 42 % in primiparous women who had used olive oil during pregnancy, compared with those who did not (26 *vs.* 68 %, respectively) [58]. In a more recent randomized controlled trial, Taavoni et al. studied the use of olive oil for a short duration in 70 nulliparous pregnant women [59]. Participants at 18–20 weeks of gestation were randomized to no treatment or application of olive oil to the abdomen without massage twice a day for 8 weeks. Although the incidence of SG was 46 % (16 of 35) in the intervention group vs. 63 % (22 of 35) in the control group, this difference was not statistically significant, with the conclusion that initiation of olive oil in the second trimester did not prevent SG onset.

As a conclusion regarding the efficacy of topical interventions in the prevention of SG, in the two most recently published Cochrane reviews [60, 61] in 2009 and 2012 respectively, the authors' conclusions were as follows: «We found no high-quality evidence to support the use of any of the topical preparations in the prevention of stretch marks during pregnancy. There is a clear need for robust, methodologically rigorous randomised trials involving larger sample sizes to evaluate the effects of topical preparations on the development of stretch marks in pregnancy. In addition, it is important that preparations commonly used by women to prevent and treat stretch marks are evaluated within the context of robust, methodologically rigorous and adequately powered randomised trials» [61].

7.2. Chemical Peel Treatments

Chemical peel treatments involve the application of trichloroacetic acid or glycolic acid (GCA). They are thought to induce an initial inflammatory response, with subsequent increased collagen production [62]. A nonrandomized, controlled trial investigating GCA reported decreases in striae furrow width but concluded that it may yield better results when used in combination with other products [63]. GCA combined with tretinoin and L-ascorbic acid [66] and trichloroacetic acid combined with the use of sand abrasion [64] or a postpeel cream [65] are such examples, all of which produced improvements in the appearance of striae. Post-inflammatory hyperpigmentation (PIH) remains a concern [62].

7.3. Mechanical treatments

Aluminum oxide microdermabrasion mechanically ablates damaged Skin [62]. A study investigating its use in SD reported clinical improvements and increased type 1 procollagen formation [67]. Reported side effects included PIH.

7.4. Radiofrequency treatments

Radiofrequency (RF) (RF) devices deliver RF current to the skin, which is converted to heat in the dermis as the result of its electrical resistance. After initial collagen denaturation with its use, there is subsequent increased collagen production. The majority of trials investigating RF for the treatment of SD have reported clinical improvements [62]. However, side effects include erythema and oedema, and the majority of trials had small cohorts.

7.5. Laser/Light therapy

Fractional lasers

Improvements in SD after treatment with a 1540-nm fractional non-ablative erbium glass laser have been reported [68–73]. Malekzad et al. [74] observed only a fair or poor improvement in 70 % of patients with its use, and, although improvements in SR have been described [74–77] the literature suggests that non-ablative lasers are most effective on SA [70]. Concerns surrounding PIH also remain. Fractional ablative carbon dioxide lasers have primarily been used in SA, with reported clinical improvements [78–82]. Side effects include PIH. Gungor et al. [83] compared the efficacy of an ablative erbium-yttrium aluminium garnet laser with a non-ablative neodymium-doped yttrium aluminium garnet laser and found poor clinical results with both. The lit-

erature suggests that, when compared with non-ablative lasers, ablative lasers are less well-tolerated and produce inconsistent results [76].

Diode lasers

1450-nm diode laser is a nonfractional laser that has been shown to increase dermal collagen. However, an RCT investigating its use in Fitzpatrick skin types IV-VI reported no improvements in SD but demonstrated high rates of PIH [84].

Intense Pulsed light (IPL)

Intense pulsed light consists of a broad-spectrum (515—1200 nm) visible beam of high-intensity light. Studies investigating its use in SD have demonstrated increased dermal collagen levels after treatment [85, 86]. However, a study comparing intense pulsed light against a fractional carbon dioxide laser for the treatment of SD concluded that the laser was more effective [87]. No RCTs have yet been performed, and PIH remains a cause for concern [85, 87, 88].

7.6. Percutaneous induction therapy

Percutaneous collagen induction therapy, or needling therapy, involves the creation of microclefts extending to the papillary dermis, resulting in increased production of collagen and elastin [89, 90]. Aust et al. [89] reported improvements in skin texture and tightening after treatment. More recently, percutaneous collagen induction therapy compared favourably against microdermabrasion combined with sonophoresis [91] and against a carbon dioxide laser [92]. However, there are no RCTs, and side effects include erythema [90—92].

7.7. Platelet-rich plasma

Platelet-rich plasma (PRP) is a concentrated solution of autologous platelets containing growth factors and cytokines injected intradermally [93]. Ibrahim et al [93] investigated its use in SD with microdermabrasion, and, despite increased collagen levels after PRP treatment alone, 13 % had worsening of their striae. They concluded that it is best to use PRP in combination with microdermabrasion. Other studies have combined PRP with RF [94, 95] and microneedling [96] all reporting varying degrees of clinical improvement.

However, small sample sizes and no RCTs make drawing definitive conclusions difficult. Side effects include bruising [93, 94].

7.8. Infrared light

Infrared light applied to skin causes heating of the dermis and collagen denaturation, with subsequent neocollagenesis. Trelles et al. [97] investigated its use in the treatment of SA. Despite positive histologic findings, including more pronounced rete processes, detection of improvements clinically remained low. Side effects were limited to erythema of the skin.

7.9. Galvanopuncture

Galvanopuncture is a needling therapy that applies a continuous microcurrent, inducing an inflammatory reaction with subsequent collagen production [98]. Bitencourt et al. [98] investigated its use in SA. All patients demonstrated clinical improvements, and erythema was the only side effect.

Further trials, with histologic analysis, are needed to further assess its efficacy.

7.10. Vascular lasers

The 585-nm pulsed dye laser (PDL) is a commonly used vascular laser. Because of its high affinity for haemoglobin, which is present in the microvasculature of SR, it can reduce the erythema of these lesions [99]. PDL appears to have minimal benefit in the treatment of SA [100—102]. Longo et al. [103] tested the 577-nm copper bromide laser, which has higher rates of absorption by haemoglobin than PDL, with 33 % complete resolution of SD.

7.11. UV light

In an intent to repigment SA, UVB (296—315 nm) and UVA1 (360—370 nm) light was used in 9 individuals. Improvement was only temporary and only in 50 % of patients, and transient hyperpigmentation was observed as a side effect [104].

7.12. Excimer laser (311 nm)

Studies using excimer laser in SA report conflicting results. Some of them reported improvements in striae pigmentation after its use [105, 106]. However, poor results were observed in others [107].

8. CONCLUSION

Stretch marks are a frequent reason of consultation in dermatology as they affect a high number of individuals, especially dark-skinned people. Obviously, this is a benign pathology, but it may cause distress of the patients by their aesthetic patterns. However, little interest is born to their study; their aetiology remains unclear, their pathogenesis is not well-known, and most important, there is no convincing treatment, and not even efficient prevention at the dawn of the 21st century. The treatments are usually not dissociating striae rubrae and striae albae, although they are completely different entities. Much remains to be done before we can bring adequate response to our patients in the prevention and management of stretch marks.

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Розтяжки (стрії): огляд

Розтяжки — це видимі лінійні рубці, що розвиваються на тілі в місцях надмірного розтягування шкіри. Це доброякісне шкірне захворювання, але через естетичне значення вони можуть завдавати страждань пацієнтам. Розтяжки дуже поширені у понад 50 % вагітних жінок і у 50—90 % населення загалом. Вони частіше уражають певні етнічні групи, особливо темношкірих пацієнтів. Факторами ризику під час вагітності є сімейний анамнез, але також має місце і зайва вага, в той час як підвищений індекс маси тіла також є фактором ризику. Клінічно початкові еритематозні і фіолетові ураження, або так звані червоні смуги, переходять у зморшкуваті, гіпопігментовані, атрофічні рубцеві відмітини, так звані білі смуги. Утворення розтяжок підтверджується чотирма основними теоріями: 1) механічне розтягнення шкіри; 2) гормональні зміни; 3) вроджене структурне порушення покривів; 4) генетична схильність. Гістологічно описані різні аномалії колагенових і еластичних волокон на рівні дерми. Лікування розтяжок завжди оманливе. Найпоширеніші місцеві методи лікування, серед яких популярні креми з центелою азійською або гіалуроновою кислотою, мигдальна олія, ретиноїди для місцевого застосування, масло какао або оливкова олія. Хімічний пілінг також можна використовувати. Можуть бути виконані різні процедури, такі як мікродермабразія, радіочастотна терапія, лазерна/світлова терапія, збагачена тромбоцитами плазми, інші, але результати часто обмежені і оманливі як для пацієнта, так і для дерматолога. Ще багато чого треба зробити для вивчення цього розповсюдженого дерматологічного захворювання.

Ключові слова: розтяжки, стрії, білі смуги, червоні стрії, колаген, еластин, фібулін, лікування.

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Растяжки (стрии): обзор

Растяжки представляют собой видимые линейные рубцы, развивающиеся на теле в местах чрезмерного растяжения кожи. Это доброкачественное кожное заболевание, но из-за своего эстетического значения они могут причинять страдания пациентам. Растяжки демонстрируют высокую распространенность у более 50 % беременных женщин и у 50—90 % населения в целом. Они чаще поражают определенные этнические группы, особенно темнокожих пациентов. Факторами риска во время беременности являются семейный анамнез, но также имеет место и прибавка в весе, в то время как в целом повышенный индекс массы тела также является фактором риска. Клинически первоначальные эритематозные и фиолетовые поражения, называемые красными полосами (SR), переходят в морщинистые, гипопигментированные, атрофические рубцовые отметины, называемые белыми полосами. Формирование растяжек подтверждается четырьмя основными теориями: 1) механическое растяжение кожи; 2) гормональные изменения; 3) врожденное структурное нарушение покровов; 4) генетическая предрасположенность. Гистологически описаны различные аномалии коллагеновых и эластических волокон на уровне дермы. Лечение растяжек всегда обманчиво. Наиболее распространены местные методы лечения, среди которых популярны кремы с центеллой азиатской или гиалуроновой кислотой, миндальное масло, ретиноиды для местного применения, масло какао или оливковое масло. Химический пилинг также можно использовать. Могут быть выполнены различные процедуры, такие как микродермабразия, радиочастотная терапия, лазерная/световая терапия, обогащенная тромбоцитами плазмы, и другие, но результаты часто ограничены и обманчивы как для пациента, так и для дерматолога. Еще многое предстоит сделать для изучения этого распространенного дерматологического заболевания.

Ключевые слова: растяжки, стрии, белые полосы, красные стрии, коллаген, эластин, фибулин, лечение.

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