Striae Distensae (Stretch Marks) and Different Modalities of Therapy: An Update

Mohamed L. Elsaie MD,* Leslie S. Baumann MD,* and Lotfy T. Elsaaiee, MD†

BACKGROUND Striae distensea (SD; stretch marks) are a well-recognized, common skin condition that rarely causes any significant medical problems but are often a significant source of distress to those affected. The origins of SD are poorly understood, and a number of treatment modalities are available for their treatment, yet none of them is consistently effective, and no single therapy is considered to be pivotal for this problem. With a high incidence and unsatisfactory treatments, stretch marks remain an important target of research for an optimum consensus of treatment.

OBJECTIVE To identify the current treatment modalities and their effectiveness in the treatment of stretch marks.

MATERIALS AND METHODS Review of the recent literature regarding clinical treatment of stretch marks with emphasis on the safety and efficacy of the newer optical devices and laser applications.

RESULTS No current therapeutic option offers complete treatment, although there are a number of emerging new modalities that are encouraging.

CONCLUSION The therapeutic strategies are numerous, and no single modality has been far more consistent than the rest. The long-term future of treatment strategies is encouraging with the advance in laser technologies.

The authors have indicated no significant interest with commercial supporters.

Successfully treating striae distensae (SD; stretch marks) has always been challenging. Nardelli gave the first morphologically correct description of these lesions in 1936, calling them striae atrophicae.[1] The exact origin of stretch marks remains unrevealed, with the factors responsible for its development poorly understood.[2]

Causes of SD are not clear, and a number of theories have been proposed (Table 1). Kogoj anticipated that a striatoxin damages the tissues in a toxic way, resulting in striations.[3] Others had shown that mechanical stretching is the main cause, leading to the rupture of the connective tissue framework.[4] Many authors have denounced this theory, not finding any relationship between growth in abdominal girth in pregnant women and formation of SD.[5] Normal growth has been suggested as another cause, with these marks commonly developing during adolescence and associated with the rapid increase in size of particular regions of the body.[6] Similarly, SD are a feature of high serum levels of steroid hormones. They are a common feature of Cushing's disease and local or systemic steroid therapy may induce them. High steroid hormone levels have a catabolic effect on the activity of fibroblasts and decrease the deposition of collagen in the substance of the dermal matrix. Obesity and rapid increase or decrease in weight have been shown to be associated with the development of SD.[7] Finally, the absence of striae in pregnancy in women with Ehlers-Danlos syndrome and their presence as one of the minor diagnostic

*Department of Dermatology and Cutaneous Surgery, Miller School of Medicine, University of Miami, Miami, Florida; †Department of Dermatology, Venereology and Andrology, Al Azhar University, Cairo, Egypt

ISSN: 1076-0512 • Dermatol Surg 2009;35:563–573 • DOI: 10.1111/j.1524-4725.2009.01094.x

TABLE 1. Different Causes for the Development of Stretch Marks

Different hypotheses on the development of stretch marks in the literature
Infection leading to the release of striatoxin that damages the tissues in a microbial toxic way[3]
Mechanical effect of stretching, which is proposed to lead to rupture of the connective tissue framework (e.g., pregnancy, obesity, weight lifting)[4]
Normal growth as seen in adolescence and the pubertal spurt that leads to increase in sizes of particular body regions[6]
Increase in the levels of body steroid hormones; Cushing's syndrome, local or systemic steroid therapy that has a catabolic effect on fibroblasts[7]
Genetic factors (absence of striae in pregnancy in people with Ehlers-Danlos syndrome and their presence as one of the minor diagnostic criteria for Marfan syndrome suggest an important genetic element)[8,9]
Immunosuppression states associated with pregnancy-induced hypertension medications, human immunodeficiency virus or diseases such as tuberculosis and typhoid[8,11]
Associated with chronic liver disease[13]

criteria for Marfan syndrome emphasize the importance of genetic factors in determining susceptibility of connective tissue.[8] Similarly, there is low expression of collagen and fibronectin genes in affected tissue.[9]

Other rarely reported causes of SD include cachetic states, such as tuberculosis and typhoid and after intense slimming diets.[10] They may also be seen in anorexia nervosa.[11] SD have been reported to occur rarely in patients positive for the human immunodeficiency virus receiving the protease inhibitor indinavir.[8] A case of idiopathic SD was also reported.[12] Men and women with chronic liver disease may also have SD.[13]

Clinical Picture and Histopathology of SD

Recent or immature SD are flattened areas of skin with a pink-red hue that may be itchy and slightly raised. Stretch marks then tend to increase in length and acquire a darker purple color. Over time, they become white, flat, and depressed. Histologically,[14] earlier-stage or immature SD tend to appear pink or red in color (striae rubra) and over time and with atrophic changes attain a white color (striae alba). High-resolution epiluminescence colorimetric assessment of SD identified four distinct types: striae alba, striae rubra, striae caerulea, and striae nigra. The direct and indirect influences of melanocyte mechanobiology appear to have a prominent effect on the various colors of SD.[15]

The histology of stretch marks is that of a scar, and the development of SD has been likened to that of wound healing or scar formation.[16] In the early stages, inflammatory changes may be conspicuous, but later the epidermis is thin and flattened. Recent SD show a deep and superficial perivascular lymphocytic infiltrate around the venules.[17] Collagen bands on the upper third of the reticular dermis are stretched and aligned parallel to the surface of the skin. In the latter stages, there is thinning of the epidermis due to flattening of the rete ridges and loss of collagen and elastin.[18] SD are two and a half times as frequent in women and affect up to 90% of pregnant women. The commonest sites are the outer aspects of the thighs and the lumbosacral region in boys and the thighs, upper arms, buttocks, and breasts in girls.[19] Recently it was demonstrated that SD are associated with loss of fibrillin, a fact that explains the counter replacement of fibrillin upon retinoic acid therapy.[20]

Treatments

Several treatments have been proposed, yet no consistent modality is available. Some authors, have suggested that time is the only treatment for SD and that it returns to normal over years, which is not true.[21] It has always been suggested that effective treatment of SD be instituted during the active stage, well before the scarring process is complete.[22] The first reliable method of treatment involved using tretinoin cream.[23] Subsequent modalities were reported afterward, with variable results, which will be discussed in this section. A summary of major topical treatments is given in Table 2.

TABLE 2. Different Topical Products and Their Speculated Modalities of Action

Product	*Indication*	*Suggested Mode of Action*
Tretinoin	Therapeutic	Exact mechanism unclear, but recent studies suggest fibroblastic stimulation.
Trofolastin	Therapeutic	Active ingredient (centella asiatica) stimulates fibroblasts and inhibits glucocorticoids.
Verum	Preventive	Active ingredient hyaluronic acid is speculated to increase tensile resistance to mechanical forces.
Alphastria	Preventive	Hyaluronic acid, the main ingredient, acts by increasing volume to oppose mechanical atrophy.
Massage with oils	Preventive	Dual action of massage and hydrant action of oils.
Glycolic acid and trichloroacetic acid peels	Therapeutic	Glycolic acid is reported to stimulate collagen production by fibroblasts and to increase their proliferation in vivo and in vitro.

Diet and Exercise

There is lack of data concerning the effect of diet and diet restrictions on stretch marks. Eighty women aged 24 to 53 participated in a 3-month weight-loss program; 29 were on a diet, 31 were on a diet plus aerobic exercise program, and 20 obese women were on a diet plus a resistance exercise program. The data suggested that the degree of SD does not change with weight loss, regardless of the type of weight loss program. SD were prevalent in 79% of the women. The central abdomen was the most common area where SD were present. No significant correlation existed between SD and the number of births, the age of the first birth, weight immediately before pregnancy, weight immediately after pregnancy, or weight at age 20. Forty-five percent of the participants responded that they did not have negative feelings about having SD, and it appeared that SD did not affect the women's psychological mood. It was inferred that a weight loss program using diet alone or a combination of diet and exercise did not change the degree of SD. Further studies are required to establish a clear effect of diet and exercise on SD.[24]

Topical Therapies

Tretinoin

The use of topical tretinoin has yielded variable results, and some of the studies had proven the inefficacy of the vitamin A derivative in the treatment of SD,[25,26] but most of the patients included in these early studies presented with old lesions that had evolved into whitish atrophic scars.[25] More recently, tretinoin has been shown to improve the clinical appearance of stretch marks during the active stage (striae rubra), although with not much effect during the mature stage (striae alba).[27] In the same study, 22 patients applied 0.1% tretinoin ($n = 10$) or a placebo ($n = 12$) daily for 6 months to the affected areas. Patients were evaluated monthly in a physical examination and using analysis of biopsy specimens of stretch marks obtained before and at the end of therapy in comparison with untreated normal skin. Targeted stretch marks in patients treated with tretinoin had a decrease in mean length and width of 14% and 8%, respectively, compared with an increase of 10% and 24%, respectively, in patients who received the placebo. Rangel and colleagues conducted an open multicenter study in Mexico on 20 women with stretch marks after pregnancy. They all applied tretinoin (retinoic acid) cream 0.1% daily for 3 months to pregnancy-related stretch marks in the abdominal area. Efficacy was evaluated according to analysis of one preselected target lesion, which was rated on a 6-point scale (-1 = worse to 4 = cleared). All target lesions decreased in length by 20%, and they demonstrated efficacy of tretinoin as a modality for treating SD of pregnancy.[28]

Hydrant Creams

Anecdotal treatments are numerous and unproven. Despite the general understanding that proper

hydration is necessary to maintain the integrity and barrier function of skin, little in the literature is available on the use of such creams in stretch mark prevention. Three studies involving 130 men in total were found.[29] The active creams in the studies described are not widely available, and it was not clear whether any particular ingredient was helpful. The lack of clarity on the studies and the scientific data available makes it difficult to conclude such creams are effective, and larger studies are needed to determine the efficacy and safety of such products in combating stretch marks.

Trofolastin One study involved 80 women and investigated the effect of massage with a cream containing *Centella asiatica* extract, vitamin E, and collagen-elastin hydrolysates (Trofolastin, Novartis Barcelona, Spain) and its preventive effect on the development of stretch marks in pregnant women.[30] Forty-one subjects used the cream, and 39 used a placebo. Results showed that 56% of the placebo group and 34% of the treated group developed SD in pregnancy. This study demonstrated that the active component, *Centella asiatica*, induced significant prevention of stretch mark development. The exact mechanism of action was identified as the stimulation of fibroblastic activity,[31] and an antagonistic effect against glucocorticoids was also reported.[32]

Verum Another study of 50 women, although lacking a placebo control, examined a cream containing vitamin E, panthenol, hyaluronic acid, elastin and menthol (verum). It was associated with fewer stretch marks during pregnancy than no treatment. One-third of women in the treated group and two-thirds of those who did not receive any treatment developed SD during pregnancy. The results suggest that the product could be helpful, although the trial had no placebo and may show the benefit of massage alone.[33]

Alphastria Alphastria is a cream that is composed of hyaluronic acid, allantoin, vitamin A, vitamin E, and dexpanthenol. The name is composed of the Greek word "alpha" prefix meaning "without," and the Latin word "stria," which means "lines." Hyaluronic acid is an organic substance found in human skin and is the main constituent of the cream. The hyaluronic content stimulates fibroblast activity and collagen production to restore any inhibition and collagen loss induced by hormonal fluctuations or mechanical stretch.[34] Only one study was conducted to demonstrate the efficacy and safety of the cream. Thirty pregnant women were recruited to receive the cream, and 30 others received a placebo as a control group. Three subjects in the exposed group and 21 in the control group developed SD. The study concluded that the product markedly lowered the incidence of stretch mark development after pregnancy.[35,36]

Topical Oil Massage and Herbal Topical Remedies

Some unconventional therapies and anecdotal reports recommend applying unproven oils and natural remedies to stretch marks. The underlying principle for this use would probably be keeping the skin well hydrated. Sweet almond oil, wheat germ oil, olive oil, avocado oil, and castor oil and applying seaweed wraps have these properties.[37] Other remedies such as comfrey, hypericum, maritime pine, equisetum, slippery elm, and wheat grass and eucalyptus tree oil are all used in creams or oils, but no efficacy studies have been performed to support these practices.[38]

Glycolic Acid and Trichloroacetic Acid

Glycolic acid (GA) is an alpha hydroxyl acid. Although there are several reports on the clinical effects of GA in rejuvenation, peeling, and photoaging, no data on the effectiveness of GA to prevent stretch marks could be found in the scientific literature. No epidemiological study on the use of GA in pregnant women has been published. One study comparing topical 20% GA and 0.05% tretinoin with 20% GA and 10% L-ascorbic acid found that both regimens improved the appearance of striae alba and showed no difference in effect from combining 10% ascorbic acid or 0.05% tretinoin with 20% GA, although it was not determined which of the ingredients

provided the effect.[39] The precise mechanism of action of GA is still unknown because the biological effects of GA on cells has not been fully studied, although GA is reported to stimulate collagen production by fibroblasts and to increase their proliferation in vivo and in vitro.[40,41] This mechanism can be useful for stretch mark treatments, but further investigations and studies are required to prove such theory.

Trichloroacetic acid (TCA; 10–35%) has been used for many years and is safe to use at low concentrations. At higher concentrations (e.g., $\geq 50\%$), TCA has a tendency to scar and is less manageable than other agents used for superficial peels. TCA is found in several proprietary peels at varying concentrations, and some kits have instructions and buffering agents so that the peel can be diluted as deemed necessary. Anecdotal reports have indicated the use of TCA in stretch marks, although there is a lack of clarity and absence of data for assessment of this subject. Some authors have had good success using low concentrations (15–20%) of TCA and performing repetitive papillary dermis-level chemexfoliation repeated at monthly intervals with reported improvement in texture and color of marks.[42,43]

Other Topical Products

Hundreds of products are available in the market, but the potential efficacy of these products had never been subjected to any clinical investigation or assessment. Moy has reported improvement with a topical agent alone (Striae Stretch Mark Formula with Regenetrol Complex, Regenterol Labs, Los Angeles, CA). Another formula available is MACROdermabrasion/DermaPhoresis Topical Kit (IntegreMed, Scottsdale, AZ) designed for use with positive-pressure salt microdermabrasion. Salt A-Peel DermaPhoresis system (IntegreMed, LLC) was shown in one study of 12 weeks to attain a 39% improvement along with positive-pressure microdermabrasion in 29 women with SD.[14]

Lasers and Light Devices

Of the many modalities used to ameliorate and improve stretch marks, lasers have recently become a popular therapeutic alternative. The 585-nm flashlamp-pumped pulsed-dye laser (PDL) is the most commonly reported laser used in treatment of SD. The use of ablative technologies such as the short-pulse carbon dioxide and erbium-substituted yttrium aluminium garnet (YAG) enjoyed a brief popularity because of prolonged healing and pigmentary alterations, especially in darker skin tones.[44] Newer applications of other laser modalities such as neodymium-doped YAG (Nd:YAG), diode, and Fraxel are finding a way into treatment of stretch marks (Table 3).

Pulsed-Dye Laser

The dilated blood vessels marked at the early stage of the stretch mark formation render the striae rubrae a good candidate for PDL.[45] According to McDaniel and colleagues and Alster, a clinical improvement in immature SD is achieved after several courses of 585-nm flashlamp PDL therapy using dynamic cooling.[46,47] McDaniel demonstrated that the optimal treatment fluence was 3 J/cm^2 using a 10-mm spot size.[46] This laser has been purported to increase the amount of collagen in the extracellular matrix. Jiminez and colleagues documented the effectiveness of the 585-nm flashlamp PDL in stretch marks of skin types I to IV and demonstrated that collagen changes precede any clinical significant change,[48] although it was also reported that, for darker skin tones (IV–VI), laser treatment of SD should be avoided or used with great caution because of the possibility of pigmentary alterations after treatment.[49]

Excimer Laser

Advances in technology have recently brought the 308-nm xenon chloride (XeCl) excimer laser to the laser arena. This newer technology allows treatment of focal areas with a wavelength close to that of traditional narrow-band ultraviolet B (UVB) light. Recent studies have demonstrated efficacy of the 308-nm XeCl laser in the treatment of

TABLE 3. Summary of Different Laser and Light Source Treatments for Stretch Marks

Type of Laser	*Effectiveness in Striae Distensae*
Pulsed dye laser	Demonstrated to be effective only for the immature element of striae (striae rubrae), targeting the vascular element. Not effective in darker skin and associated with pregnancy-induced hypertension. When combined with radio frequency, it showed a more promising response even on striae alba.[46–48]
Copper bromide laser	A 577-nm laser that showed a mild to moderate effect in one study on skin types II and III; no histological analysis was carried out. Needs much evaluation.[55]
1,450 nm diode laser	Not useful in skin of color (IV–VI) and associated with many complications.[57]
1,064 neodymium-doped yttrium aluminium garnet laser	Targets immature striae and satisfactory results in the few studies so far.[58–60]
Excimer laser	A 308-nm xenon-chloride laser with a good safety profile, although only repigments temporarily and does not have an effect of atrophy.[53,54]
Intense pulsed light	A good alternative that was shown to be an effective tool in striae alba, although with a high incidence of pregnancy-induced hypertension.[63]
Fractional photothermolysis	Fewer studies conducted, although all reported efficacy in mature and immature striae and demonstrated an increase in the number of collagen and elastin fibers and a good safety profile.[70–73]

psoriasis[50] and vitiligo.[51] The 308-nm XeCl laser has the advantage over standard phototherapy of having greater precision and the ability to deliver higher energy fluences to the target tissue in less time. It is also possible that UVB radiation delivered in the form of laser light has a different light–tissue interaction, which may cause greater efficacy.[52] After the use of the excimer in many hypopigmentary conditions, it was used for striae alba. Two studies have shown temporary repigmentation and improvement of leukoderma in SD with excimer laser. Post-laser biopsies showed greater melanin content and hypertrophy of the melanocytes, although it failed to show any improvement in skin atrophy.[53,54]

Copper-Bromide Laser

The copper-bromide laser is a 577-nm laser that is only mentioned once in the literature as being used for stretch marks. One study treated 15 patients with different stretch marks on different areas of the body, exposing them to laser settings of 4 J/cm^2 for SD on the breast in women or 8 J/cm^2 for SD on other parts of the body. The study concluded that the copper-bromide laser was effective in decreasing the size of the SD, although further studies are needed to determine the ideal parameters and the number of sessions needed for an optimum response.[55]

1,450-nm Diode Laser

The diode laser is a midinfrared greater-than-700-nm non-ablative laser technology with an integrated dynamic cooling device. In recent clinical trials, this type of laser has demonstrated efficacy in the diminution of rhytides, treatment of active acne, and improvement of atrophic scars.[56] Only one study examined the efficacy and safety of diode laser in the treatment of 11 patients, Fitzpatrick skin types IV to VI. Patients were assigned randomly to receive 4, 8, or 12 J/cm^2 fluences, and treatment sessions were offered every 6 weeks for a total of three sessions. The incidence of postinflammatory hyperpigmentation was 64%, and there was no improvement in the SD. It was concluded that, for skin types IV to VI, treatment of SD is not useful, and the incidence of postinflammatory hyperpigmentation is significant.[57]

1,064-nm Nd:YAG Laser

The 1,064-nm long-pulse Nd:YAG laser has also led to an increase in dermal collagen when used in the

nonablative treatment of facial wrinkles.[58] In addition, this laser has a strong attraction to vascular targets[59] that, associated with its action on dermal collagen, can lead to the beneficial effects observed in the treatment of immature SD. The histopathologic characteristics present in immature SD are similar to those found in recent scars.[60] This would explain why scars also show a significant improvement after treatment with the 1,064-nm long-pulse Nd:YAG laser. A recent study used the 1,064-nm Nd:YAG on immature SD in 20 patients, and observers and patients identified results as satisfactory.[61] Owing to its physical characteristics, represented mainly by the 1,064-nm wavelength, the laser used is safe. Complications rarely result when the device and parameters are appropriately used in epilation or vascular alterations, even in patients with dark skin. In addition, the cooling of the SD before and immediately after the use of the laser represents another factor in epidermal protection. More research will be devised for a better cosmetic outcome and approach using the Nd:YAG lasers.

Intense Pulsed Light

Intense pulsed light (IPL) seems to be a good alternative treatment for SD. IPL is characterized by a noncoherent filtered flashlamp with a broadband spectrum (515–1,200 nm). Hernandez and colleagues used the IPL for 20 Hispanic patients of skin types III and IV and on long-standing mature SD of the abdomen. All patients received five sessions, and the stretch marks were biopsied before and after therapy. The study demonstrated an overall significant difference in the post-dermal treatment thickness and in the skin textures of the older stretch mark lesions.[62] Studies have demonstrated that IPL replaced dermal elastosis with neo-collagen, which explains its usefulness in improving stretch marks.[63]

UVB/UVA1 Combined Therapy

The MultiClear device (Curelight Ltd., Gladstone, NJ) is a unique device combining UVB and selective UVA1 wavelengths and emitting a high-intensity noncoherent light with peaks at 313, 360, and 420 nm. It is currently approved by the Food and Drug Administration (FDA) for use in UVB phototherapy as well as psoralen plus UVA phototherapy to treat psoriasis, vitiligo, atopic dermatitis, and hypopigmented scars. The device achieves a repigmentary effect of unknown length because of the lack of studies conducted. A study was conducted on nine patients with mature striae alba who received 10 treatment sessions, and biopsies were taken at the baseline and end of the study. At the end of the study, all patients reported some form of hyperpigmentation that was transient and did not affect any surrounding tissues. No changes were seen on biopsy to indicate an effective remodeling collagen effect of the device, although it needs further assessment.[64] There have been no reports of greater incidence of skin cancer development with this form of phototherapy, but further research is prudent before establishing a safer profile of the device.[64]

Fractional Photothermolysis

Fractional photothermolysis is a newer, nonablative resurfacing laser technique. This 1,550-nm laser creates microzones or microthermal zones (MTZs) of "injury" onto the skin. Within these areas, localized epidermal necrosis occurs alongside collagen denaturation. Ultimately, the necrotic debris is expelled, and neocollagenesis occurs. Additionally, because this laser treatment is nonablative, the islands of normal skin serve to speed the healing process. Fractional photothermolysis has been FDA approved for dermatological procedures requiring the coagulation of soft tissue; treatment of periorbital wrinkles; treatment of acne scars and surgical scars; photocoagulation of pigmented lesions such as lentigos (age spots), solar lentigos (sun spots), melasma, and dyschromia; and skin-resurfacing procedures.[65]

There are several studies confirming the efficacy of fractional photothermolysis for treatment of facial scarring. Glaich and colleagues reported on seven patients who were treated with fractional photothermolysis for hypopigmented scars (secondary

to inflammatory acne or gas fire burn). Patients received two to four treatments at 4-week intervals. No adverse events were noted. Independent physician clinical assessment revealed improvements of 51% to 75% in hypopigmentation in six of seven patients 4 weeks after final treatment.[66] Alster and colleagues reported on 53 patients who were treated with fractional laser photothermolysis for atrophic scars. No complications or adverse events were noted. Ninety-one percent of patients had at least 25% to 50% improvement after a single treatment; 87% of patients receiving three treatments had at least 51% to 75% improvement in the appearance of scars after 1 month, with stable improvement after 6 months.[67] Hasegawa and colleagues treated 10 patients with acne scars using fractional photothermolysis. There was no hyperpigmentation reported, and results as seen by patients were successful.[68]

The histopathologic characteristics present in immature SD are similar to those found in recent scars.[60] Some authors using hematoxylin and eosin or orcein stain reported evidence of new collagen formation and demonstrated an overall increase in the density of collagen after fractional photothermolysis.[69] This mechanism is the anticipated mode of reversing the signs and atrophy associated with stretch marks using fractional photothermolysis, the technology receiving the most attention in this regard. There are a few published studies on SD and fractional resurfacing. A 2007 Brazilian clinical study showed that Fraxel improved texture and appearance of mature, white SD in skin types I to IV. Fifteen female patients, skin types I to IV, with mature SD were treated with fractional photothermolysis (1,550 nm Fraxel SL Laser). Treatments included four to five sessions at weekly intervals, pulse energy of 8 to 10 mJ/MTZ, and a final density of 2,000 MTZs/cm^2. The treatment response was assessed by comparing pre- and 2-week post-treatment clinical photography evaluated by two physicians and patient questionnaires. The study demonstrated an early new indication for stretch mark treatment with Fraxel.[70] A Korean study used fractional photothermolysis on four patients of skin type IV with striae gravidarum. All patients received just one treatment and were assessed visually and histologically using skin biopsy. The histology showed an increase in the number of elastic fibers, and no side effects were demonstrated.[71] A more recent study treated six patients with fractional photothermolysis, and they all showed clinical improvement in melanin and erythema indices and in elasticity. The authors demonstrated an increase in collagen and elastin deposition in the dermis.[72] The optimal settings and parameters to use have not been decided upon, but investigators have shown promising results with three to five treatments sessions with their therapeutic approaches.[73]

Microdermabrasion

Aluminum oxide resurfacing has become a popular method of resurfacing. Microdermabrasion is effective in many skin conditions such as acne scars, mottled pigmentation, and fine wrinkles.[74] It has been established too that microdermabrasion induces epidermal signal transduction pathways that are associated with remodeling of the dermal matrix. Microdermabrasion appears to set in motion a cascade of molecular events capable of causing dermal remodeling and repair.[75] There is a paucity of literature about the efficacy of microdermabrasion in stretch mark therapy, but Mahuzier in his text book on microdermabrasion stated that 10 to 20 sessions of microdermabrasion at an interval of not less than 1 month, each session resulting in bleeding points, provide satisfactory improvement in SD.[76] A more recent Egyptian study on the clinical and molecular evaluation of treating SD with microdermabrasion demonstrated a promising effect of dermabrasion on stretch marks. The study used 20 patients with SD receiving five microdermabrasion treatments at weekly intervals on half of the body; SD on the other half of the body served as a control. Biopsies from patients were analyzed using real-time reverse transcriptase polymerase chain reaction for assay of type I procollagen I-mRNA levels. The results showed an

overall good to excellent response in more than half of the subjects, with improvement more marked in striae rubra, and upregulation of type I procollagen mRNA was found in all treated SD samples.[77] Further studies on a larger scale are needed to identify the efficacy of using such techniques for stretch marks.

Radiofrequency Devices

The use of radiofrequency (RF) devices have been reported to be an effective and safe noninvasive technique to tighten the face and neck skin. Unlike lasers, which convert light to heat and target a specific chromophore through the selective photothermolysis, RF devices transfer higher-energy fluences to the skin through a coupling method. The electrical energy transmitted is converted to heat upon reacting with the skin's resistance.[78] It is reported that collagen fibril contraction occurs immediately after RF treatments, which induces new collagen formation.[79] A recent study evaluating the effectiveness of a RF device (Thermage, Thermacool TC, Thermage Inc., Hayward, CA) in combination with PDL subjected 37 Asian patients with darker skin tone with SD to a baseline treatment with a RF device and PDL. This was followed by an additional two sessions of PDL performed at weeks 4 and 8. Histological evaluation was done on nine patients who were selected randomly; 89% of the patients showed good to very good overall improvement, and 59% were graded as good and very good in elasticity. All histological evaluations demonstrated an increase in the amount of collagen fibers, and six of the nine specimens showed an increase in the number of elastic fibers. Hyperpigmentation developed in one study subject only and improved in 3 months.[80] The effects of using RF devices in combination with lasers are yet to be decided, but preliminary studies show a synergistic, effective, safe modality that could be a good alternative for stretch mark therapy.

Summary

The complete evaluation of a patient with SD should include consideration of the SD stage (rubrae or alba) and of the skin type. Expectations must be realistic, and the optimal treatment modality should be carefully selected to avoid any exaggeration of the problem or complications. The therapeutic strategies are numerous, and no single modality has been far more consistent than the rest. Fractional photothermolysis, despite the smaller number of preliminary studies, shows much promise in dermal remodeling and subsequent improvement of white and pigmented SD. Finally, more research and clinical trials should be encouraged to address this cosmetic problem of concern to a large segment of women worldwide.

References

1. Nardelli L. Importanza semiologica delle "striae cutis atrophicae". Boll sez Region Soc Ital Dermatol 1936;1:46.
2. Kim BJ, Lee DH, Kim MN, et al. Fractional photothermolysis for the treatment of striae distensae in Asian skin. Am J Clin Dermatol 2008;9:33–7.
3. Kogoj F. Seitrag zur atiologie und pathogenese der stria cutis distensae. Arch Dermatol Syphiliol 1925;149:667.
4. Agache P, Ovide MT, Kienzler JL, et al. Mechanical factors in striae distensae. In: Morettig G, Rebora A, editors. Stria Distensae. Milan: Brocades; 1976. p. 87–96.
5. Osman H, Rubeiz N, Tamim H, et al. Risk factors for the development of striae gravidarum. Am J Obstet Gynecol 2007;196: 62–e1–5.
6. Weber FP. Idipathic stria atrophicae of puberty. Lancet 1935; 229:1347.
7. Stevanovic DV. Corticosteroid induced atrophy of the skin with telegiectasia: a clinical and experimental study. Br J Dermatol 1972;87:548–56.
8. Burrows NP, Lowell CR. Disorders of connective tissue. In: Burns T, Breathnach S, Cox N, Griffith C, editors. Rooks Text Book of Dermatology. Blackwell science; 2004. p. 46–47.
9. Lee KS, Rho YJ, Jang SI, et al. Decreased expression of collagen and fibronectin genes in striae distensae tissue. Clin Exp Dermatol 1994;19:285–8.
10. Sparker MK, Garcia-Gonzalez E, Sanchez LT. Sclerosing and atrophying conditions. In: Schachner LA, Hansen RC, editors. Pediatric Dermatology. 2nd edn. New York: Churchill Livingstone; 1996. p. 897.
11. Strumia R, Varotti E, Manzato E, Gualandi M. Skin signs in anorexia nervosa. Dermatology 2001;203:314–7.
12. Basak P, Dhar S, Kanwar AJ. Involvement of the legs in idiopathic striae distensae—a case report. Indian J Dermatol 1989;34: 21–2.
13. Johnston GA, Graham-Brown RA. The skin and disorders of the alimentary tract and the hepato biliary system.kidney and cardiopulmonary systems. In: Gilchrest BA, Paller AS, Leffell DJ,

Wolff K, Austen KF, Goldsmith LA, Katz SI, editors. Fitzpatrick's Dermatology in General Medicine. 7th edn. New York: McGraw-Hill; 2007. p. 1445–60.

14. McDaniel DH. Laser therapy of stretch marks. Dermatol Clin 2002;20:67–76.
15. Hermans JF, Pierard GE. High resolution epiluminescnece colorimetry of striae distensae. JEADV 2006;20:282–7.
16. Atwal GS, Manku LK, Griffiths CM, et al. Striae gravidarum in primiparae. Br J Dermatol 2006;155:965–9.
17. Arem AJ, Kischer CW. Analysis of striae. Palst Reconstr Surg 1980;65:22–9.
18. Pierard GE, Nizet JL, Adant JP, et al. Tensile properties of relaxed excised skin exhibiting stria distensae. J Med Engl Technol 1999;23:69–72.
19. Chang AL, Agredano YZ, Kimball AB. Risk factors associated with striae gravidarum. JAAD 2004;51:881–5.
20. Watson RE, Parry EJ, Humphries JD, et al. Fibrillin microfibrils are reduced in skin exhibiting striae distensae. Br J Dermatol 1998;138:931–7.
21. Alaiti S, Obagi ZE. Striae distensae (26 November 2006). Emedicine journal. Available at http://www.emedicine.com/derm/topic406.html (accessed March 2008).
22. Garcia HL. Dermatological complications of obesity. Am J Clin Dermatol 2002;3:497–506.
23. Elson ML. Treatment of striae distensae with topical tretinoin. J Dermatol Surg Oncol 1990;16:267–70.
24. Schwingel AC, Shimura Y, Nataka Y, et al. Exercise and striae distensae in obese women. Med Sci Sports Exerc 2003;35 (suppl):S33.
25. Elson ML. Treatment of striae distensae with topical tretinoin. J Dermatol Surg Oncol 1990;16:267–70.
26. Pribanich S, Simpson FG, Held B, et al. Low-dose tretinoin does not improve striae distensae: a double-blind, placebo controlled study. Cutis 1994;54:121–4.
27. Kang S, Kim KJ, Griffith CE, et al. Topical tretinoin (retinoic acid) improves early stretch marks. Arch Dermatol 1996;132: 519–26.
28. Rangel O, Arias L, Garcia E, et al. Topical tretinoin 0.1% for pregnancy-related abdominal striae: an open-label, multicenter, prospective study. Adv Ther 2001;18: 181–6.
29. Young GL, Jewell D. Creams for preventing stretch marks in pregnancy. (Review).. Cochane Library 2008;1: 1–7.
30. Mallol J, Belda MA, Costa D, et al. Prophylaxis of striae gravidarum with a topical formulation. A double blind trial. Int J Cosm Sci 1991;13:51–7.
31. Velasco M, Romero E. Drug interaction between asiaticoside and some anti inflammatory drugs in wound healing of the rat. Curr Ther Res 1976;18:121–5.
32. Brinkhaus B, Lindner M, Schuppan D, et al. Chemical, pharmacological and clinical profile of the East Asian medical plant *Centella asiatica*. Phytomedicine 2000;7:427–48.
33. Wierrani F, Kozak W, Schramm W, et al. Attempt of preventive treatment of striae gravidarum using preventive massage ointment administration. Wiener Klinishe Wochenschrift 1992;104:42–4.
34. Shah DN, Recktenwall-Work SM, Anseth KS. The effect of bioactive hydrogels on the secretion of extracellular matrix molecules by valvular interstitial cells. Biomaterials 2008;29:2060–72.
35. Zambon pharmaceuticals. Discussion ouverte sur le theme des vergeturres des grossesse. 2007. Available at http://www.zambon ch.ch/prodotti-fra-dettagli-28.html (accessed March 2008).
36. De-Bauman M, Walther M, de-Weck R. Effectivness of alphastria cream in the prevention of pregnancy stretch marks (striae distensae). Results of a double-blind study. Gynakologische Rundschau 1987;27:79–84.
37. Ody P. Herbs for a Healthy Pregnancy. Los Angeles, CA: Keats Publishing; 1999. p. 42–4.
38. Goldberg DJ, Marmur ES, Schmults C, et al. Histologic and ultrastructural analysis of ultraviolet B laser and light source treatment of leukoderma in striae distensae. Dermatol Surg 2005;31:385–7.
39. Ash K, Lord J, Zukowski M, McDaniel DH. Comparison of topical therapy for striae alba (20% glycolic acid/0.05% tretinoin versus 20% glycolic acid/10% L-ascorbic acid). Dermatol Surg 1998;24:849–56.
40. Kim SJ, Park JH, Kim DH, et al. Increased in vivo collagen synthesis and in vitro cell proliferative effect of glycolic acid. Dermatol Surg 1998;24:1054–8.
41. Okano Y, Abe Y, Masake H, et al. Biological effects of glycolic acid on dermal matrix metabolism mediated by dermal fibroblasts and epidermal keratinocytes. Exp Dermatol 2003;12(Suppl 2): 57–63.
42. Obagi ZE, Obagi S, Alaiti S, Stevens MB. TCA-based blue peel: a standardized procedure with depth control. Dermatol Surg 1999;25:773–80.
43. Alaiti S, Obagi E. Striae distensae. (July 24, 2007) emedicine journal. Available at http://www.emedicine.com/derm/topic406.htm (accessed March 2008).
44. McDaniel DH. Laser therapy of stretch marks. Dermatologic clinics 2002;20:67–76.
45. Karsai S, Roos S, Hammes S, et al. Pulsed dye laser: what's new in non-vascular lesions. JEADV 2007;21:877–90.
46. McDaniel DH, Ash K, Zukowoski M. Treatment of stretch marks with the 585 nm flashlamp pumped pulsed dye laser. Dermatol Surg 1996;22:332–7.
47. Alster TS. Laser treatment if hypertrophic scars, keloids and striae rubra. Dermatol Clin 1997;15:419–29.
48. Jiminez GP, Flores F, Berman B, et al. Treatment of striae rubra and stria alba with the 585-nm pulsed-dye laser. Dermatol Surg 2003;29:362–4.
49. Nouri K, Romagosa R, Chartier T, et al. comparison of the 585 nm pulse dye laser and the short pulsed co2 laser in the treatment of striae distensae in skin types IV and VI. Dermatol Surg 1999;25:368–70.
50. Trott J, Gerber W, Hammes S, et al. The effectiveness of PUVA treatment in severe psoriasis is significantly increased by addi-

tional UV 308-nm excimer laser sessions. Eur J Dermatol 2008;18:55–60.

51. Shen Z, Gao TW, Chen L, et al. Optimal frequency of treatment with the 308-nm excimer laser for vitiligo on the face and neck. Photomed Laser Surg 2007;25:418–27.
52. Passeron T, Ortonne JP. The 308 nm excimer laser in dermatology. Press Med 2005;34:301–9.
53. Goldberg DJ, Marmur ES, Hussain M. 308 nm excimer laser treatment of mature hypopigmented striae. Dermatol Surg 2003;29:596–9.
54. Goldberg DJ, Marmur ES, Schmults C, et al. Histologic and ultrastructural analysis of ultraviolet B laser and light source treatment of leukoderma in striae distensae. Dermatol Surg 2005;31:385–7.
55. Longo M, Postiglione MG, Marangoni O, et al. Two-year follow-up results of copper bromide laser treatment of striae. J Clin Laser Med Surg 2003;21:157–60.
56. Tanzi EL, Alster TS. Comparison of a 1450 nm diode laser and a 1320 nm Nd:YAG laser in the treatment of atrophic facial scars: a prospective clinical and histologic study. Dermatol Surg 2004;30:152–7.
57. Tay YK, Kwok C, Tan E. Non-ablative 1,450-nm diode laser treatment of striae distensae. Lasers Surg Med 2006;38:196–9.
58. Trelles MA, Álvarez X, Martín-Vásquez MJ, et al. Assessment of the efficacy of nonablative long-pulsed 1,064 nm Nd:YAG laser treatment of wrinkles compared at 2,4, and 6 months. Facial Plast Surg 2005;21:145–53.
59. Sadick NS. Laser treatment with a 1,064 nm laser for lower extremity class I-III veins employing variable spots and pulse width parameters. Dermatol Surg 2003;29:916–9.
60. Groover IJ, Alster TS. Laser revision of scars and striae. Dermatol Ther 2000;13:50–9.
61. Goldman A, Rossato F, Pratti C. Stretch marks: treatment using the 1,064 nm Nd:YAG laser. Dermatol Surg 2008;34:1–7.
62. Hernandez-Perez E, Charrier EC, Valencia-Ibiett E. Intense pulsed light in the treatment of striae distensae. Dermatol Surg 2002;28:1124–30.
63. Hernandez-Perez E, Valencia-Ibiett E. Gross and microscopic findings in patients submitted to nonablative full-face resurfacing using intense pulsed light: a preliminary study. Dermatol Surg 2002;28:651–5.
64. Sadick N, 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.
65. Wanner M, Tanzi E, Alster T. Fractional photothermolysis: treatment of facial and nonfacial cutaneous photodamage with a 1550 nm erbium-doped fiber laser. Dermatol Surg 2007;33:23–8.
66. Glaich A, Goldberg L, Friedman R, Friedman P. Fractional photothermolysis for the treatment of postinflammatory erythema resulting from acne vulgaris. Dermatol Surg 2007;33: 842–6.
67. Alster T, Tanzi E, Lazarus M. The use of fractional laser photothermolysis for the treatment of atrophic scars. 2007;33: 295–9.
68. Hasegawa T, Matsukura T, Mizuno Y, et al. Clinical trial of a laser device called fractional photothermolysis system for acne scars. J Dermatol 2006;33:623–7.
69. Geronemus RG. Fractional photothermolysis: current and future applications. Lasers Surg Med 2006;38:169–76.
70. Macedo OR, Macedo O, Bussade M, et al. Fractional photothermolysis for the treatment of striae distensae. JAAD 2007;56:204.
71. Petro I. Fractional photothermolysis tackles striae distensae. Dermatol Times 2007;28:94–6.
72. Kim BJ, Lee DH, Kim MN, et al. Fractional photothermolysis for the treatment of striae distensae in Asian skin. Am J Clin Dermatol 2008;9:33–7.
73. Sadick N. Commentary on stretch marks: treatment using the 1,064 nm NdYAG laser. Dermatol Surg 2008;34:1–7.
74. Llyod J. The use of microdermabrasion for acne: a pilot study. Dermatol Surg 2001;27:329–31.
75. Karimipour DJ, Kang S, Johnson TM, et al. Microdermabrasion: a molecular analysis following a single treatment. JAAD 2005;52:215–23.
76. Mahuzier F. Microdermabrasion of stretch marks. In: Microdermabrasion or Parisian Peel in Practice, Mahuzeier F., editor.. Marseille, 1999:25–65.
77. Abdel-Latif AM, Elbendary AS. Treatment of striae distensae with microdermabrasion: a clinical and molecular study. JEWDS 2008;5:24–30.
78. Hsu TS, Kaminer MS. The use of non ablative radiofrequency technology to tighten the lower face and neck. Semin Cutan Med Surg 2003;22:115–23.
79. Zelickson BD, Kist D, Bernstein E, et al. Histological and ultrastructural evaluation of the effect of radiofrequency-based nonablative dermal remodeling device. Arch Dermatol 2004;140:204–9.
80. Suh DH, Chang KY, Son HC, et al. Radiofrequency and 585-nm pulsed dye laser treatment of striae distensae: a report of 37 Asian patients. Dermatol Surg 2007;33:29–34.

Address correspondence and reprint requests to: Mohamed L. Elsaie, MD, MBA, Department of Dermatology and Cutaneous Surgery, University of Miami, 7401 N. Meridian Avenue, Nichol Building, Miami Beach, Fl 33140, or e-mail: egydoc77@yahoo.com