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Striae Distensae After Breast Augmentation

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Abstract
Background One known but not fully understood complication after breast augmentation is the new onset of stretch marks (striae distensae) on the surgically treated breast. To date, all publications on this subject have been case reports. No report has fully described the actual incidence, risk factors, or management of striae distensae after breast surgery.
Methods This study prospectively followed patients who underwent primary breast augmentation using silicone implants in a single group practice from 2007 to 2011. New-onset striae distensae were actively investigated. Time from surgery to the moment of striae onset, patient age, nulliparity, use of oral contraceptives, overweight, personal history of stretch marks, and other variables were evaluated.
Results A total of 409 patients were included in the study. In 19 cases (4.6 %), new-onset striae distensae after breast augmentation were observed. The population with striae distensae was significantly younger than the total population (29.56 vs 20.91 years; $p = 0.012$). Striae distensae also were more common in nulliparous than in multiparous women (8.29 vs 0.52 %; $p = 0.006$), overweight women (17.77 vs 3.02 %; $p = 0.016$), women using oral contraceptives (7.89 vs 0.55 %; $p = 0.008$), and women with a personal history of stretch marks (8.97 vs 3.36 %; $p = 0.031$). No relation was shown regarding implant pocket type, size, or profile.
Conclusion Striae distensae may be a common but underreported complication after breast augmentation. In this series, striae distensae developed in 4.6 % of the patients within 1 year after breast augmentation. Severity may vary from inconspicuous small marks (classifications 1 and 2) to wide red and active striae rubra (classifications 3 and 4). Nulliparity, use of oral contraceptives, overweight, personal history of stretch marks, and younger age were related to a higher incidence of striae distensae. The increased rates in these groups may be associated with their exposure to higher estrogen levels and the important role of this hormone in facilitating the formation of striae distensae. Further studies are needed to show whether changes in these risk factors (i.e., weight loss, contraceptive withdrawal) may help to decrease striae distensae rates is these populations.
Level of Evidence IV This journal requires that authors assign a level of evidence to each article. For a full description of these Evidence-Based Medicine ratings, please refer to the Table of Contents or the online Instructions to Authors at www.springer.com/00266.

Keywords Striae distensae breast augmentation · Stretch marks · Striae breast · Striae breast implants · Striae treatment · Stretch mark breast

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Breast augmentation currently is among the procedures performed most commonly by plastic surgeons around the world. It was the number one cosmetic operation performed by plastic surgeons in the United States in 2008, with more than 307,000 patients undergoing cosmetic breast augmentation. Given the frequency of breast augmentation, a clear understanding of the current benefits,

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risks, and potential complications is essential to providing accurate and complete consultations to patients.

Several complications can follow breast augmentation, with most of them already well documented. They include but are not limited to capsular contracture, seroma, asymmetry, undesirable waviness and palpability, and leakage or rupture of the implant [1, 2]. The incidence of these complications varies widely among the several articles on the subject [2–4]. One known but not fully understood complication is the new onset of stretch marks (striae distensae) on the surgically treated breast.

To date, all publications on this subject have been case reports. Consequently, a retrospective study that fully investigates the actual incidence, risk factors, and management of striae distensae after breast surgery is essential. Although the cause of striae distensae after breast augmentation is not yet completely understood, it is hypothesized that the marks 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.

Considering the rapid distension of the breast skin envelope as a collective common factor, it is not clear why only some women experience striae distensae after augmentation. Some case reports also have suggested that striae distensae are related to nulliparity, young age, and oral contraceptive use [2, 4–7]. Other factors that could be implicated such as implant position and size have not been thoroughly investigated [1, 2, 4, 6].

Although historically seldom reported as a common complication in the literature (only 30 cases are reported to date), striae distensae may become a significant cause of patient dissatisfaction after surgery. We hypothesized that a deeper analysis of the subject may show that it is, in fact, highly underreported because of its minor nature. This study aimed to evaluate the actual incidence and epidemiology of striae distensae after breast augmentation.

Methods

This study prospectively followed patients who underwent primary breast augmentation using silicone implants in a single group practice from 2007 to 2011. All the surgical procedures were performed by one of two board-certified plastic surgeons (A.R.B., F.V.B.) working as part of an integrated, multidisciplinary team. Secondary and mastopexy cases were excluded. All cases had pre- and postoperative photography and a breast skin exam performed by a board certified dermatologist (A.V.B.). The time from surgery to the moment of striae onset and patient ages were identified. In addition, nulliparity, use of oral contraceptives, overweight, and personal history of stretch marks were evaluated. Oral contraceptive use was defined as use at the time of surgery. Overweight was defined as a body mass index (BMI) higher than 25 kg/m^2. After new-onset stria was identified, a dermatologist consultation was warranted for all the patients.

The incision used and the details regarding the implant type, size, and pocket plane were gathered. Before the operation, all the patients were informed about the risks and benefits of the respective procedure. Smooth and textured silicone gel-filled implants were used. The patients were followed for at least 12 months after surgery. All the patients had a combination of continuous pump-controlled propofol infusion and local anesthesia. A routine of surgical steps was maintained when possible, as described briefly. The routine was a modification of a previously described surgical technique outlined elsewhere [5]. Decisions regarding placement of the incision, volume, and pocket depended on the preferences of the patient and a tissue-based analysis performed by the attending surgeon.

Before the skin incision, the dissection planes were symmetrically infiltrated on each side with 150 ml of a 1:200.00 epinephrine solution comprising 10 ml of bupivacaine 2 % and 20 ml of lidocaine 2 %. After the skin incision, all further dissection was performed with a Colorado needlepoint-tip electrocautery (Stryker, Kalamazoo, Michigan) with blended cut and coagulation current (50:50). Only electrocautery needlepoint pocket dissection under direct visualization was used. No blunt or blind dissection was performed at any time.

The patients were discharged the same day as the surgery. No drains were used. During the postoperative period, we recommended the use of an adjustable strap across the upper pole of the breasts and/or a supportive bra for 2 weeks. Antibiotics, pain medicine, and breast skin moisturizer were prescribed.

Details regarding location and severity of striae were analyzed. Patients were grouped according to our proposed severity of striae distensae classification (Fig. 1):

Class 0: No new striae distensae
Class 1: Uni- or bilateral striae distensae limited to one quadrant of the breast and not longer than the diameter of the areola
Class 2: Uni- or bilateral striae distensae limited to two quadrants of the breast and not longer than the diameter of the areola
Class 3: Uni- or bilateral striae distensae limited to three quadrants of the breast and not longer than the diameter of the areola
Class 4: Uni- or bilateral striae distensae in all four quadrants of the breast and/or longer than the diameter of the areola.

Treatment protocols followed the severity classification.

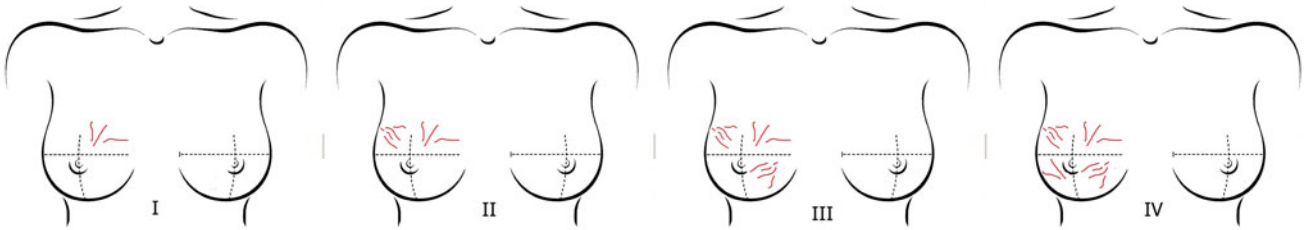

Fig. 1 Striae distensae classification

The database was constructed with Excel (Microsoft Corporation, Redmond, WA). Descriptive statistics tabulated the frequency of striae distensae within several groups in terms of different patient variables (patient age, nulliparity, overweight, use of oral contraceptives, and personal history of stretch marks) and surgical variables (implant volume, implant type, and pocket plane and incision choice). Statistical analysis and calculation of sample size were performed using the Statistical Package for the Social Sciences Windows version 13.0 (SPSS, Chicago, IL, USA). The descriptive statistics used for quantitative continuous variables (implant size) and the qualitative ordinal variables were the mean and standard deviation after confirmation of the normal distribution. All p values less than 0.05 were considered significant. The p values from contrast groups after the overall χ^2 tests are reported, with one p value shown for each two-group comparison.

Results

The study included 409 augmentation patients, who received a total of 818 implants. The follow-up period ranged from 0 to 52 months (mean, 16 months). With regard to surface texture, 40 smooth implants (20 cases) and 768 mechanically textured prostheses were used.

A total of 19 patients (4.6 %) experienced striae distensae after breast augmentation during the follow-up period. Of these 19 patients, 17 had bilateral involvement, and 2 (both classified as type 1 in our classification system) had unilateral involvement (36 breasts affected or 4.4 % of the surgically treated breasts). Details regarding severity are shown in Table 1.

Among the 19 affected patients, 5 patients (26.3 % of all striae distensae cases, 1.2 % of all cases that had surgery) were classified as type 1 in our classification system, 3 patients (15.8 % of all striae distensae cases, 0.7 % of all cases that had surgery) as type 2, 5 patients (26.3 % of all striae distensae cases, 1.2 % of all cases that had surgery) as type 3, and 6 patients (31.6 % of all striae distensae cases, 1.5 % of all cases that had surgery) as type 4. Figures 2 and 3 show two of these cases.

Table 1 Occurrence of striae distensae according to classifications

Striae distensae classification	All cases		SD cases	
	($n = 409$)	n (%)	($n = 19$)	n (%)
1	5	1.2	5	26.3
2	3	0.7	3	15.8
3	5	1.2	5	26.3
4	6	1.5	6	31.6
Total		4.6		100.0

The mean time from surgery to the onset of striae was 1.83 months (range, 1–3 months). The patients ranged in age from 17 to 64 years. The mean age in the total population was 29.56 years. The patients with striae distensae ranged in age from 17 to 25 years (mean, 20.91 years). The population with striae distensae was 8 years younger, on the average, than the total population, a difference that was significant ($p = 0.012$).

The distribution of new-onset striae distensae by implant volume, type, and pocket plane and incision choice are listed in Table 2. No association between striae distensae and implant size, type, or pocket or incision choice was established in this series. It is important to highlight that the anecdotal relation between the size of the implant and the striae distensae was not confirmed in this series (average, 282 vs 300.8 ml; $p > 0.05$). However, striae distensae were more common in nulliparous than in multiparous women (8.29 vs 0.52 %; $p = 0.006$), overweight women (17.77 vs 3.02 %; $p = 0.016$), women using oral contraceptives (7.89 vs 0.55 %; $p = 0.008$), and women with a personal history of stretch marks (8.97 vs 3.36 %; $p = 0.031$). New-onset striae distensae rates within several clinical variables are listed in Table 3.

Discussion

Striae distensae are linear scars that form in areas of dermal damage produced by stretching of the skin. The etiology and pathophysiology of striae distensae are poorly understood. However, rapid mechanical stretch produced by

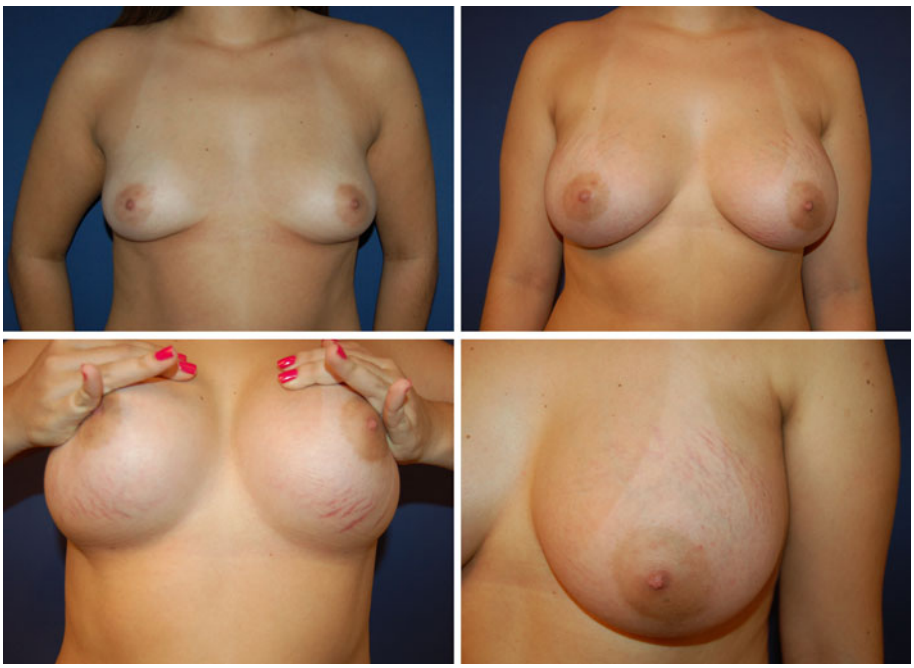

Fig. 2 An overweight (BMI, 28 kg/m^2) 21-year-old woman nullipara with transaxillary breast augmentation (subglandular, 300-ml, highprofile texturized implants) presented 36 days after surgery with bilateral striae distensae longer than the diameter of the areola (type 4) in all four quadrants of her breast. *Upper right* striae distensae at the erythematous and inflammatory stage (striae rubrae) can be seen in detail in her lower and upper quadrants

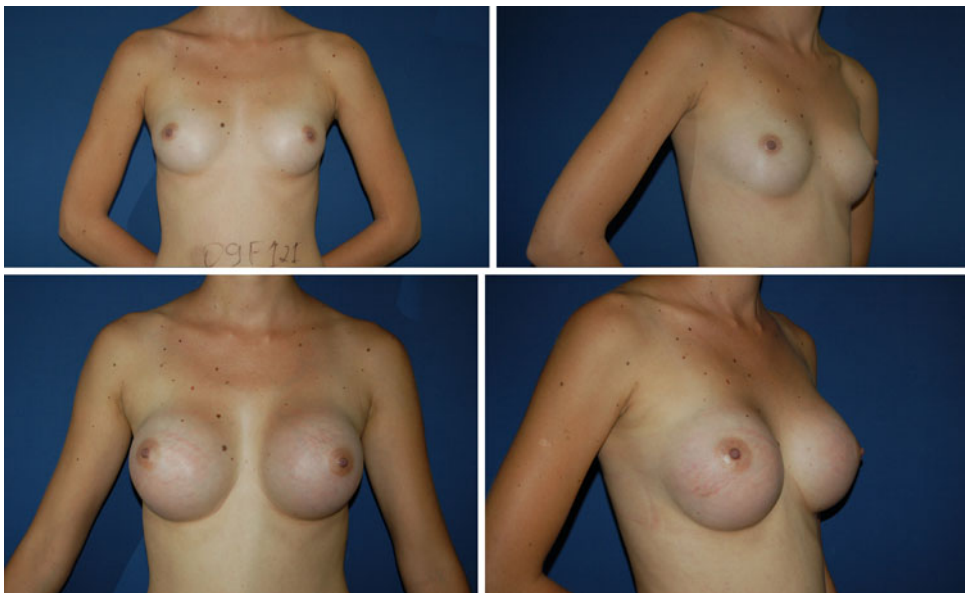

Fig. 3 A 20-year-old woman with transaxillary breast augmentation (subglandular, 310-ml, high-profile texturized implants) presented 58 days after surgery with bilateral striae distensae in two quadrants of the breast (type 2). *Upper right* striae distensae redness and texture can be seen in detail in her lower and upper quadrants

implant introduction and hormonal factors are known contributing factors [6–10]. Recent findings indicate that under certain conditions, hormonal receptor expression is increased, suggesting that regions undergoing greater mechanical stretching of the skin may express greater hormonal receptor activity [7]. This activity may influence the metabolism of the extracellular matrix, causing the formation of striae distensae [7, 9, 11].

Alterations in hormone receptors occur within a well-defined period during the formation of striae distensae. However, the functionality of hormone receptors differs during different stages in the development of the lesions. One study showed that estrogen receptors doubled in skin with striae distensae compared with healthy skin [7]. The androgen and glucocorticoid receptors in the striae distensae skin also increased.

Obesity is known to cause several abnormalities of sex hormone production and metabolism. Androstenedione production rates are elevated and serve as prehormones of both testosterone and estrogens. Extragonadal aromatization of androgens to form estrogens (androstenedione to estrone and testosterone to estradiol) is elevated, resulting in increased estrogen production rates. Obese persons are thus chronically exposed to hyperestrogenemia, which may

Table 2 Distribution of striae distensae (SD) by implant type, pocket plane, and incision choice

	All cases ($n = 409$)		SD cases ($n = 19$)	
Implant volume average: ml (range)	282 (180–480)		300.8 (235–350)	
Implant type: *n* (%)				
Textured round high projection	220	53.8	14	73.7
Textured round extra-high projection	169	41.3	5	26.3
Smooth round high projection	20	4.9	0	0
Pocket plane				
Subglandular pocket	320	78.2	16	84.21
Retropectoral pocket	89	21.8	3	15.79
Incision choice				
Axillary incision	342	83.6	16	84.21
Inframammary incision	45	11.0	2	10.53
Areola incision	22	5.4	1	5.26

Table 3 New-onset striae distensae (SD) rates for several clinical variables

Medical history factors	All patients ($n = 409$) % (*n*)	SD patients ($n = 19$) % (*n*)	SD rate per 100 patients	Chi-square *p* value
Parity				
Nulliparity	53 (217)	95 (18)	8.29	0.012
Parity	47 (192)	5 (1)	0.05	
BMI (kg/m^2)				
Normal	89 (364)	58 (11)	3.02	0.016
Overweight/obese	11 (45)	42 (8)	17.77	
Oral contraceptives				
Yes	56 (228)	95 (18)	7.89	0.008
No	44 (181)	5 (1)	0.55	
History of SD				
Yes	19 (78)	37 (7)	8.97	0.031
No	81 (331)	63 (12)	3.36	

BMI body mass index

explain their higher incidence of striae distensae. The use of oral contraceptives also causes the same hormonal imbalance [9–12]. In nulliparous and young patients, striae distensae may be related to the fact that these groups also may present with higher levels of estrogen and a tighter skin envelope more susceptible to the rapid mechanical stretch.

In summary, the role of estrogen receptors in striae distensae formation recently has become clear, and hyperestrogenic states (absolute or relative) such as obesity, use of oral contraceptives, young age, and nulliparity may increase the risk of striae distensae. Clinically, the condition goes through two stages: an initial raised erythematous, inflammatory stage (striae rubrae, Figs. 2 and 3) and a white, depressed, finely wrinkled second stage (striae albae).

The histology of stretch marks is that of a scar, and the development of striae distensae has been likened to wound healing or scar formation. In the early stages, inflammatory changes may be conspicuous, but later, the epidermis is thin and flattened.

The development of striae distensae after breast augmentation is a complication that must be recognized. Complication rates after breast augmentation range from 0.6 to 20 % [1–4]. Most of the series, however, do not include striae distensae rates. In our case series, striae distensae developed in 4.6 % of all the women within 3 months after surgery. Due to the minor nature of striae distensae, we believe it has been underreported in the literature until now. Although minor, striae distensae may be considered one of the most common complications after breast augmentation.

We also were able to measure the risk for the development of striae distensae within several risk factor groups. The role of young age became clear. Despite patient ages ranging from 17 to 64, 100 % of the striae distensae patients were younger than 25 years. Patients with striae distensae ranged in age from 17 to 25 years, with most

patients younger than 22 years (82 %). The average age was 20.91 years in the affected group, statistically lower ($p = 0.012$) than the average in the general group (29.56 years). The pathophysiology of this mechanism in younger patients still is uncertain, but it may be related to skin stretching caused by microfibril damage to fibrilins, which in younger women may be more fragile and thus more susceptible to rupture [9, 10]. However, further investigation is necessary to show whether younger skin has fewer fibrilins or a less resistant form with consequent predisposition to formation of striae in their stretching.

Nulliparity, use of oral contraceptives, and overweight also were found to be related to striae distensae after breast augmentation. These findings are in line with those of most studies that have investigated the striae distensae problem in the general population and with all case reports published previously on the subject. The rationale behind these risk factors is that excess weight and the use of oral contraceptives may cause a hormone imbalance. Some authors have found that although estrogen accelerates healing, the actions of androgens are primarily deleterious. The shift that occurs in the balance between serum estrogen and androgen levels in these patients may therefore have important consequences for fundamental tissue repair processes and striae distensae formation [7].

We believe that cases with a higher risk of striae distensae would benefit from a dermatologist consultation, preoperative skin treatment, and withdrawal of oral contraceptives if possible. It seems plausible to us that weight loss also may reduce the risk of striae distensae development. We therefore advise our high-risk patients (with 2 or more positive risk factors) to stop their use of oral contraceptives if possible and to lose weight before the surgery if necessary. The current study, however, did not evaluate the effect of oral contraceptive withdrawal or weight loss on the striae distensae development risk. Further studies are necessary to show whether these measures are effective.

We believe that patients should be informed about their estimated striae distensae risk before surgery. This study may help surgeons to provide better information to patients in this regard. Prevention should also include adequate skin care after surgery. Use of skin moisturizer may be helpful in all cases. Dermatologists have long stated that treatment is more effective if started soon during the inflammatory phase. Treatment during the Alba phase is ineffective [11–14]. Consequently, a high rate of suspicion is important and may help the surgeon to treat striae distensae adequately when present.

We note that the data in this report reflect a single-center experience with specific patient characteristics and may differ among different populations and skin types. Unknown causes of hyperestrogenic states and other known causes of striae distensae such as steroid use, Cushing disease, pituitary or adrenal tumors, rapid growth during puberty, and pregnancy should be recognized and differentiated from the breast augmentation-related striae formation.

Conclusion

Striae distensae may be a common but underreported complication after breast augmentation. In the current series, striae distensae developed in 4.6 % of the patients within 1 year after breast augmentation. Its severity may vary from inconspicuous small marks (classifications 1 and 2) to wide red and active striae rubra (classifications 3 and 4). Nulliparity, use of oral contraceptives, overweight, a personal history of stretch marks, and younger age were related to a higher incidence of striae distensae. The increased rates in these groups may be associated with their exposure to higher estrogen levels and the important role of this hormone in facilitating the formation of striae distensae. Further studies are needed to show whether changes in these risk factors (i.e., weight loss, contraceptive withdrawal) may help to decrease striae distensae rates in these populations.

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Conflicts of interest The authors declare that they have no conflicts of interest to disclose.

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