Breast Striae After Cosmetic Augmentation

Tsung-Lin Roger Tsai, Alexander Chase Castillo and Clayton L. Moliver

Aesthetic Surgery Journal 2014 34: 1050 originally published online 23 July 2014
DOI: 10.1177/1090820X14541121

Breast Striae After Cosmetic Augmentation

Aesthetic Surgery Journal
2014, Vol. 34(7) 1050–1058

DOI:10.1177/1090820X14541121
www.aestheticsurgeryjournal.com
SAGE

Tsung-Lin Roger Tsai, MD; Alexander Chase Castillo, BS; and Clayton L. Moliver, MD, FACS

Abstract

Background: Breast augmentation is the most popular cosmetic surgery procedure in the United States. Postoperative striae is a known but incompletely understood complication of breast augmentation.

Objectives: The authors investigated their own patient population to discern risk factors for new-onset striae after cosmetic breast augmentation.

Methods: A retrospective chart review was performed for patients who underwent primary breast augmentation from 2005 to 2012 in a single-surgeon practice. Initial chart review revealed that only patients aged ≤25 years exhibited new striae; therefore, only patients from this age group were included. Potential risk factors examined included age, body mass index (BMI), oral contraceptive use, time of last menstrual period (LMP), parity, smoking and alcohol status, diabetes mellitus, and personal history of striae. Implant and surgical factors examined included implant material (silicone vs saline), volume, and location (submuscular vs subglandular placement) and the site of incision.

Results: Of the 549 patients included in the study, 17 (3.10%) had new-onset striae, observed at a mean of 58 days postoperatively. The risk of striae was statistically significantly higher ($P < .05$) among patients who were younger (3.3 times), were nulliparous (14.38 times), began their LMP >14 days before surgery (9.24 times), and had a history of striae (6.11 times). There was a strong correlation between new-onset breast striae and implant size, as well as BMI ($P = .07$).

Conclusions: There is a strong correlation between new-onset striae and hormone levels, genetic factors, and tissue stretch components in patients who undergo cosmetic breast augmentation. This information can be utilized to better educate patients about this potential complication.

Level of Evidence: 4

Keywords

breast augmentation, breast implants, complications, striae, scarring, risk factors, hormonal effects

Accepted for publication January 13, 2014.

Breast augmentation is the most popular cosmetic surgical procedure in the United States; 330 631 procedures were performed in 2012.[1] Any complication can be a significant disappointment for the patient and the surgeon. A known but incompletely understood complication of breast augmentation is striae distensae, also called stretch marks.

Striae are linear dermal scars that form in areas of dermal damage. The histology is similar to that of scar tissue formation, but the exact origin is still unknown. Investigators have hypothesized that these scars are precipitated by an inflammatory reaction that destroys collagen and elastic fibers, with ensuing regeneration in the direction of the mechanical forces. This results in flattening and atrophy of the epidermis. Many theories have been suggested to explain the etiology of striae formation, including infection, effects of mechanical stretching, hormonal influences, genetic factors, immunosuppressed states, and chronic liver disease.[2,3]

Striae usually form within 4 to 12 weeks postoperatively. Immature striae are characterized by linear bands of smooth skin that are erythematous for approximately 8 to

Dr Tsai is a resident, Mr Castillo is a medical student, and Dr Moliver is a Clinical Professor of Plastic Surgery in the Division of Plastic Surgery, Department of Surgery, University of Texas Medical Branch, Galveston.

Corresponding Author:
Dr Clayton L. Moliver, 575 E Medical Center Blvd, Webster, TX 77598, USA.
E-mail: claymoliver@gmail.com

10 months, after which the mature striae become white and flat.

Although striae are rare after breast augmentation, they can be disfiguring and devastating for the patient, especially because the procedure's goal is to improve cosmesis. To date, only 1 publication has addressed the incidence and risk factors of striae after breast augmentation; to our knowledge, all other reports have been case studies. We investigated our patient population to analyze multiple factors and deduce any possible correlations that cause striae after cosmetic breast augmentation. Enhanced understanding of the etiology will enable us to better inform our patients preoperatively about this potential complication.

METHODS

This study was approved by the Institutional Review Board of the University of Texas Medical Branch in Galveston. A retrospective chart review was performed for patients who underwent primary cosmetic breast augmentation mammaplasty from 2005 to 2012 in a single-surgeon practice. Cases of augmentation mastopexy were excluded. One surgeon (C.L.M.) performed all procedures in the same clinical setting and placed either saline or silicone gel implants, all of which were from the same manufacturer (Mentor Worldwide LLC, Santa Barbara, California). All implants were moderate plus, smooth wall, round style, and all were placed in the dual-plane submuscular pocket.

After initial review of patients' medical records, it was noted that new-onset breast striae occurred only in patients aged ≤25 years. Therefore, inclusion criteria were all women ≤25 years of age who underwent primary breast augmentation for cosmetic purposes; 549 patients met these criteria and were included in the study. (Hence, patients who underwent secondary augmentation, augmentation mastopexy, or reconstruction were excluded.) Photographs of all patients were obtained pre- and postoperatively. Striae were determined to be present based on postoperative photographs and notes in the medical records. All photographs were examined by the surgeon who performed the operations (C.L.M.).

The potential risk factors examined included age, body mass index (BMI), oral contraceptive use, time of last menstrual period (LMP), parity, smoking and alcohol status, diabetes mellitus, and personal history of striae. Surgical and implant factors also were examined, including incision site, type of implant (silicone vs saline), volume of implant, implant placement location (subglandular vs submuscular), and the size and actual fill volume of saline implants.

The database was constructed and statistical analysis was performed with SAS version 9.3 (SAS Institute, Cary, North Carolina). Logistic regression was performed to assess whether the various risk factors would predict striae. Variables determined to be significant at the $P < .05$ level via univariate regression analysis were then examined by multivariate regression analysis. Relative risk was analyzed for significant variables. Age was subcategorized into single-year increments from 15 to 25 years. The BMI was defined as underweight (<18.5 kg/m^2), normal weight (18.5-24.9 kg/m^2), or overweight (>25 kg/m^2). The LMP was subcategorized as to whether the surgery occurred in the first half (days 1-14) or second half (after day 14) of the menstrual cycle. Statistical significance was defined as $P < .05$.

RESULTS

Mean follow-up time for the 549 patients was 13 months; 516 (94%) were underweight or normal weight, and 33 (6%) were overweight. Oral contraceptives were taken by 258 patients (47%). For 357 patients (65%), the first day of the LMP occurred >14 days before the surgery. It was noted that 285 patients (52%) were nulliparous compared with 264 (48%) who were at least primiparous. Only 11 patients (2%) had a history of striae. Implant data were as follows: 93 patients (17%) received silicone implants (mean volume, 446 cc; range, 250-800 cc), and 456 (83%) received saline implants (mean volume, 486 cc; range, 225-1000 cc; mean overfill, 75 cc). The mean implant volume was 479 cc. Overall, 159 patients (29%) were smokers, 1 had diabetes mellitus, and 214 (39%) were social alcohol drinkers (the remainder did not drink).

New-onset breast striae after breast augmentation were observed in 17 cases (3.10%). The risk of this complication was found to be statistically significant among the following subsets of patients: those who were 18 to 20 years old, were nulliparous, began their LMP >14 days before surgery, and had a personal history of striae. In this study, there was no association between striae formation and type of implant (silicone vs saline), incision site, implant location, history of smoking, diabetes mellitus, or alcohol use. On average, breast striae developed 57.71 days postoperatively (range, 30-90 days).

The risk of postoperative breast striae was significantly greater among younger patients (18-20 years of age; $P = .0056$), peaking at 9.77% of patients aged 19 years and decreasing to 1.42% of 25-year-olds (Figure 1). The mean age of our cohort was 22 years, and the mean age for breast striae development was 21 years. Table 1 shows the percentage of patients in each age group who experienced striae and their relative risk for developing this complication. The relative risk of breast striae formation for an 18- to 20-year-old was 3.3 times greater than for patients of other ages.

Nulliparous patients were found to have a significant risk for breast striae ($P = .0003$). In our study, 16 (94.75%) of the 17 patients who developed striae were nulliparous (Figure 2). For a nulliparous (vs multiparous) patient, the

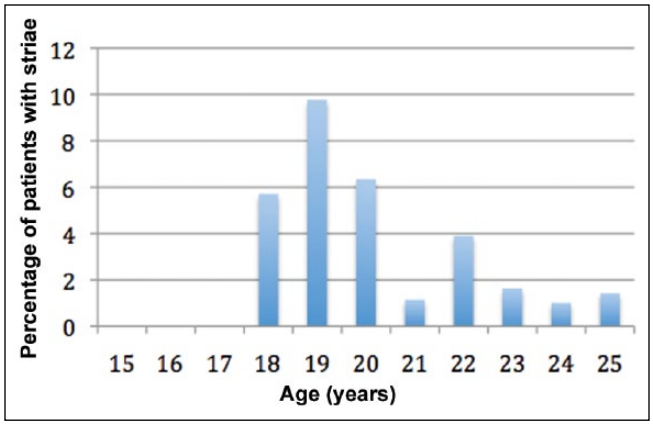

Figure 1. Percentage of patients with new-onset breast striae after breast augmentation, stratified by age.

Table 1. New-Onset Breast Striae: Number of Patients and Relative Risk According to Age

Age, y	No. With Striae /Total (%)	Relative Risk
15	0/1 (0)	0
16	0/2 (0)	0
17	0/4 (0)	0
18	2/35 (5.71)	1.87
19	4/41 (9.77)	2.81
20	4/63 (6.35)	2.3
21	1/88 (1.14)	0.32
22	3/77 (3.90)	1.27
23	1/61 (1.64)	0.48
24	1/99 (1.01)	0.36
25	1/70 (1.42)	0.41
Age range, y		
18-20	10/139 (7.19)	3.3
21-25	7/395 (1.77)	0.52
>25	0/541 (0)	0

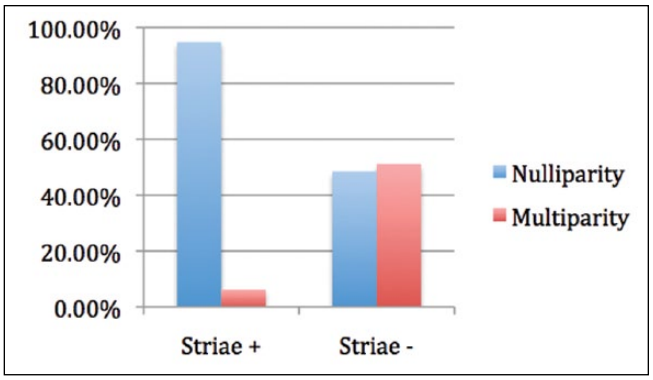

Figure 2. Percentage of nulliparous and multiparous patients with breast striae formation after breast augmentation.

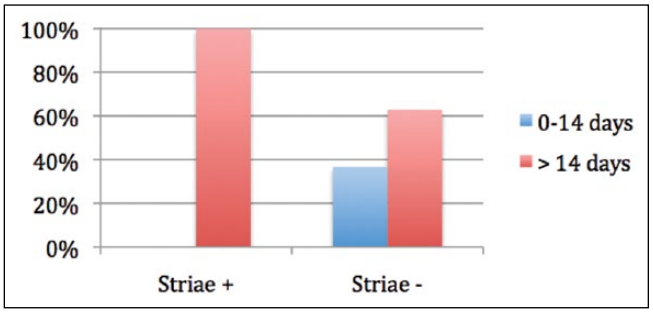

Figure 3. Percentage of breast augmentation patients whose surgery occurred in the first vs second half of the menstrual cycle and the association with breast striae formation.

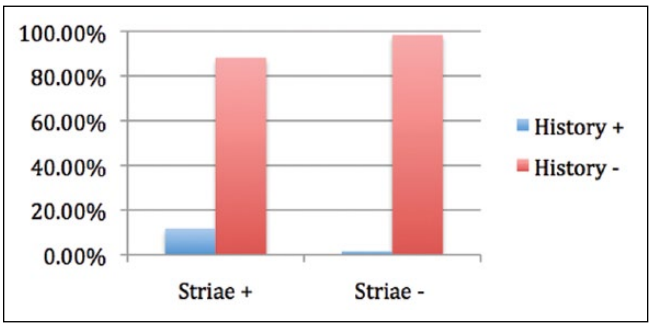

Figure 4. Percentage of breast augmentation patients with or without a history of striae and the association with breast striae formation.

risk of developing breast striae was 14.38 times greater. We did not collect data on parity status for women aged >25 years.

Patients whose LMP began >14 days before surgery (n = 357) had a statistically significant risk of developing breast striae (P = .05). In fact, not a single patient with breast striae in our study had her surgery in the first half of her menstrual cycle (Figure 3). The risk of breast striae development was 9.24 times higher for patients whose surgery occurred >14 days since their LMP began.

Patients with a history of striae anywhere else on their body (17 patients) also had a significant risk for breast striae after augmentation mammaplasty (P = .0391; Figure 4), and 2 (11.76%) of these patients developed breast striae. Patients with a history of striae anywhere on the body had a 6.11 times greater risk of postoperative breast striae. Five of the 532 patients who did not experience breast striae postoperatively already had striae somewhere else on their body.

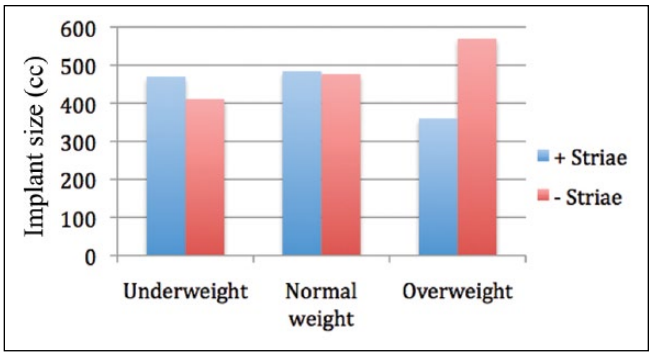

Figure 5. Mean implant volume in relation to body mass index, and the correlation with striae formation.

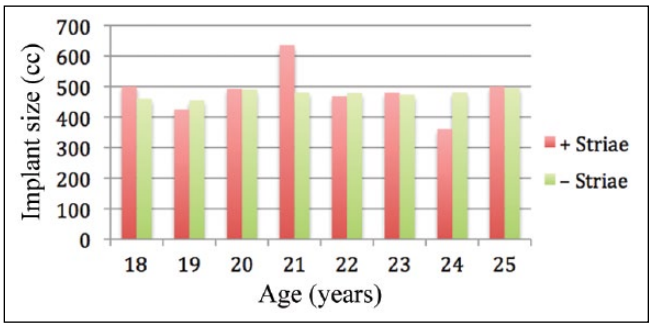

Figure 6. Implant size by age and the correlation with striae formation.

Although not statistically significant, there was a strong correlation between new-onset breast striae and implant size as well as BMI, specifically among underweight and normal-weight patients (P = .07; Figure 5). Analyses of implant size vs age (Figure 6) and BMI vs striae formation (Figure 7) revealed no significant differences. Only 1 patient was found to have diffuse striae of the entire breast; all others had more localized striae (Figures 8-11).

DISCUSSION

Breast striae can be a morbid complication to an otherwise aesthetically pleasing augmented breast. Striae are believed to be dermal scars resulting from intrinsic stretching forces on soft tissues. Pinkus et al[4] were the first to describe the increase in elastic fibers in striae and compare it with scar tissue. Bhangoo et al[5] also observed elastogenesis in scar tissue. Ebert[6] and Breathnach[7] examined striae evolution, from early (striae rubrae) to late (striae albae) phases. Striae rubrae are characterized by inflammation with cuffing of small vessels by lymphocytes and monocytes accompanied by increased mast cells and active fibroblasts, which destroys collagen and elastic fibers. Striae albae are

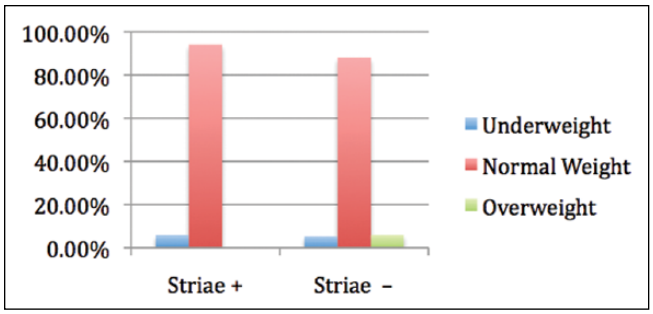

Figure 7. Association between body mass index and striae formation.

characterized by a mature, typical white stretch mark thought to be scar tissue resulting from the regeneration of collagen and elastic fibers in the direction imposed by mechanical forces. Healing takes place under tension and thus may result in linear and atrophic striae. Moreover, hair follicles and other appendages are absent due to the initial damage.

Zheng et al[2] observed mature striae tissue under microscopy. The epidermis over the striae was shown to be flattened and thinned, with an extremely dense packing of thin, eosinophilic collagen bundles in a parallel array, horizontal to the surface. These bundles replaced the papillary dermis located just underneath the epidermis. Elastic fibers were numerous, were densely packed, and had a fine parallel orientation to the surface, mimicking the collagen fibers. Cordeiro et al[8] recently examined hormonal receptors located within striae tissue under frozen processing, protein extraction, and Western blot assay; they noted a significant increase in hormonal receptors in comparison to normal tissue: 2.2-fold increase in estrogen receptors, 1.8-fold increase in androgen receptors, and 1.7-fold increase in glucocorticoid receptors. It is well known that estrogen and corticosteroid products interfere with the mechanisms of wound repair and extracellular matrix reorganization; they also reduce collagen synthesis and induce skin atrophy. These hormones have also been postulated to decrease adhesion between collagen fibers and increase ground substance with degranulation of mast cells, resulting in striae formation in areas of stretching.[9] Burrows and Lowell[10] suggested that striae develop more easily in skin that contains a high amount of rigid cross-linked collagen, which is common in early adulthood. Thomas and Liston[11] demonstrated that the prevalence of striae gravidarum was greater in younger adult obstetric patients. Maia et al[12] and Elsaie et al[13] also observed more striae in younger patients and suggested a relationship to skin stretching caused by microfibril damage to

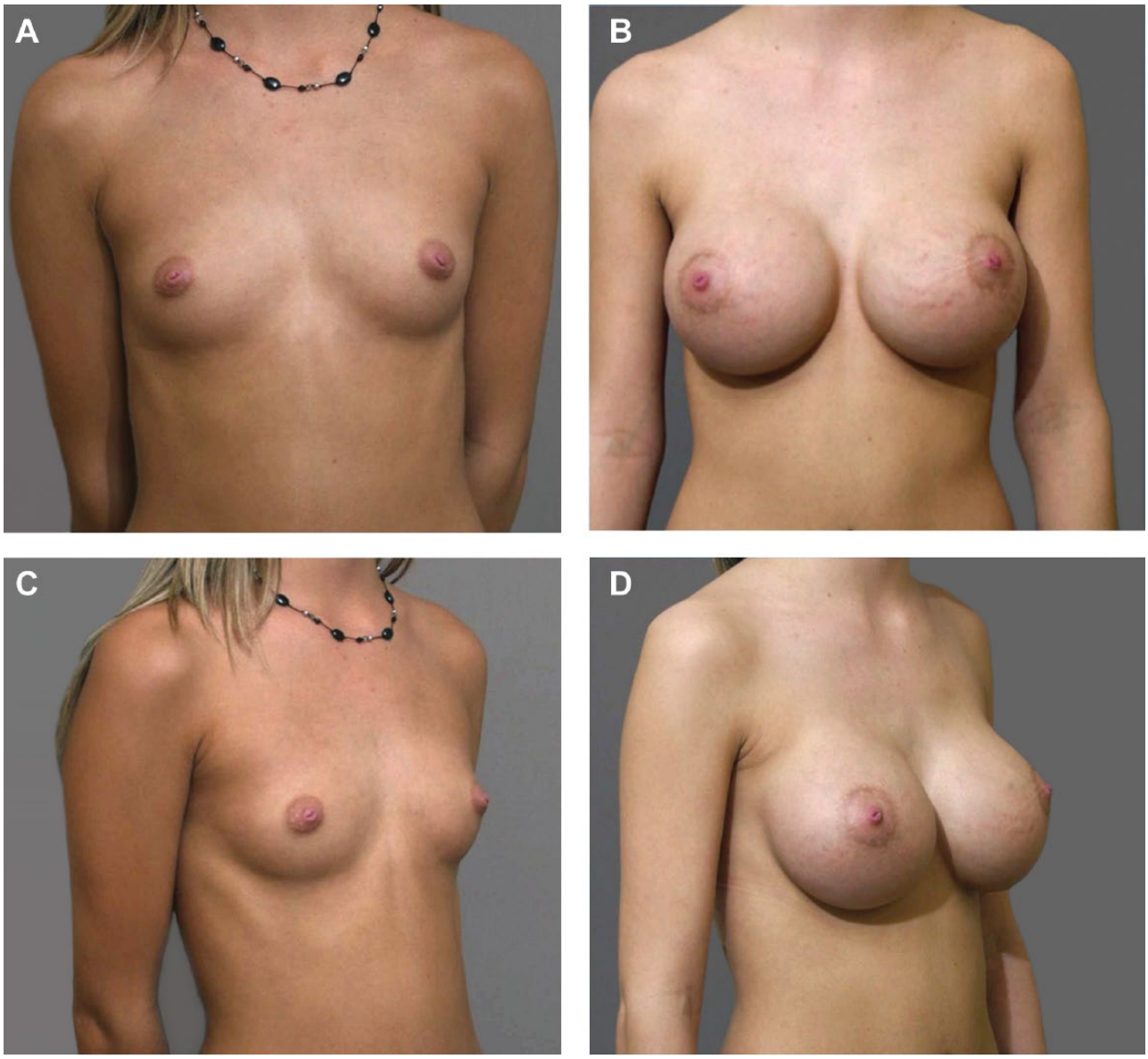

Figure 8. (A, C) This 20-year-old woman with micromastia underwent bilateral augmentation mammaplasty with 530-cc saline implants (Mentor Worldwide LLC, Santa Barbara, California). (B, D) Photographs obtained 6 months postoperatively show mild, localized striae. Onset of striae occurred 42 days postoperatively.

fibrilins, which may be more fragile and susceptible to rupture in younger women.

Various underlying factors may contribute to the formation of striae. The exact pathogenesis is not known; rather, it has been considered multifactorial, involving mechanical, hormonal, and genetic factors. Rosenthal[14] proposed classifying striae according to 4 etiologic mechanisms: (1) insufficient development of the tegument, including a deficiency in its elastic properties; (2) rapid stretching of the skin; (3) endocrine imbalance; and (4) other, possibly toxic, causes.

Our finding that all patients with postoperative breast striae were ≤25 years of age correlates with findings of Burrows and Lowell[10] and Thomas and Liston[11] in younger adults. We believe that this may be attributable to a hormonal influence and to the characteristics of soft tissue in

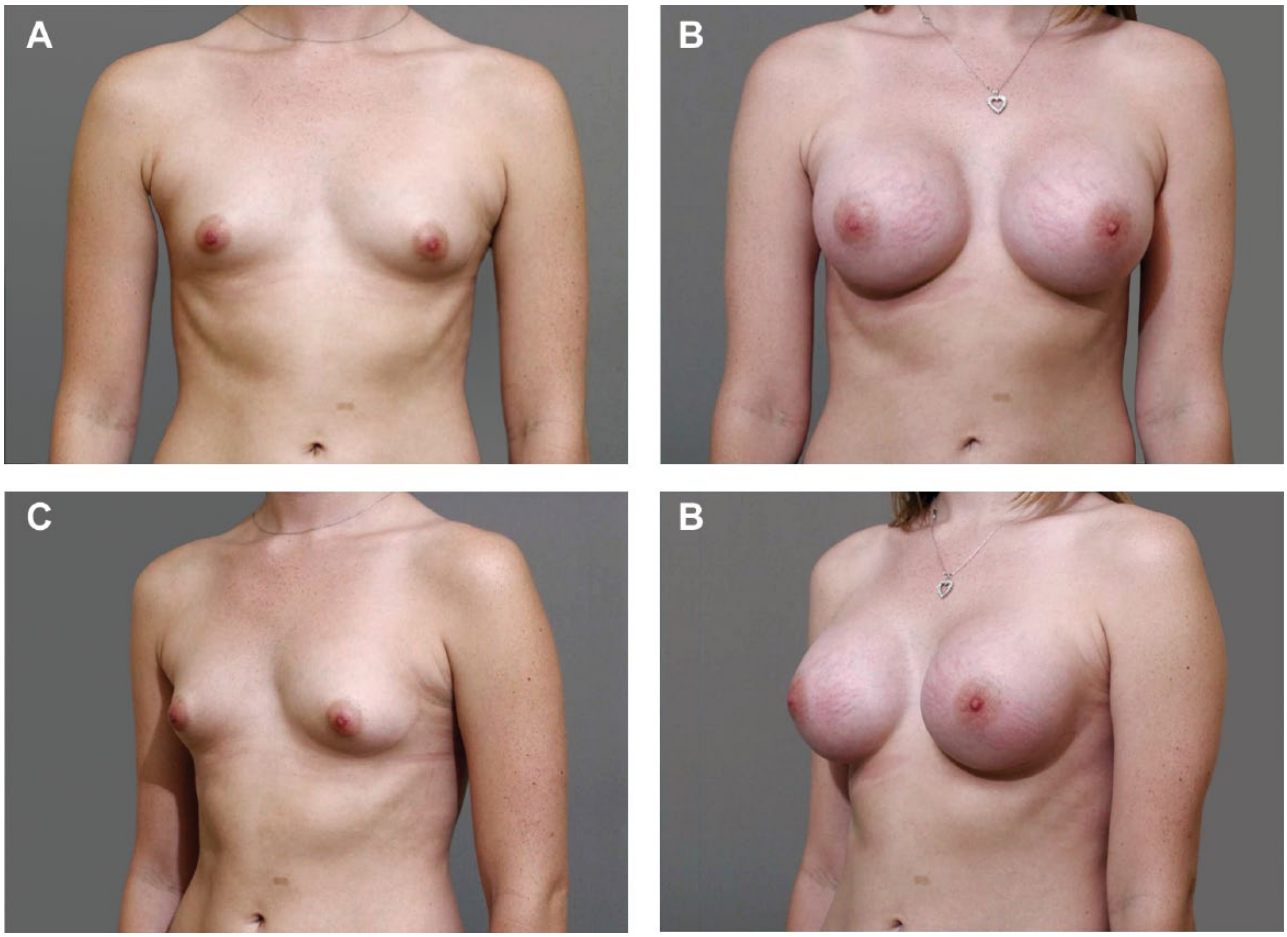

Figure 9. (A, C) This 22-year-old woman with micromastia underwent bilateral augmentation mammaplasty with 405-cc saline implants (Mentor Worldwide LLC, Santa Barbara, California). (B, D) Photographs obtained 3 months postoperatively show moderate striae over half of the breast. Onset of striae occurred 42 days postoperatively.

young adults. Growth hormone, estrogen, and dehydroepiandrosterone (DHEA)—the most abundant circulating steroid in the body and a precursor to many hormones—are at their highest levels between the ages of 18 and 25 years and decrease dramatically with age.

The common denominator for all of our significant findings is a strong hormonal influence. As noted, patients ≤25 years of age are exposed to higher levels of various hormones. It was surprising to discover that the LMP for 100% of our patients with postoperative breast striae had started more than 14 days before surgery, when progesterone levels are predominant and estrogen levels are moderate. Together with the findings of Cordeiro et al,[8] who noted increased estrogen, androgen, and glucocorticoid receptors in striae tissue, our observations of increased risk associated with young age and higher overall estrogen, progesterone, or growth hormone levels indicate that breast striae formation correlates strongly with hormonal influences. In addition, a possible genetic component is suggested by the significant association between personal history of striae and higher risk of new-onset breast striae in our study.

Our findings confirm those of Basile et al[15] for striae distensae after breast augmentation; 4.6% of their patients developed striae, and increased risk was associated with nulliparity, use of oral contraception, being overweight, younger age (peak incidence at age 20 years), and personal history of stretch marks. To our knowledge, this is the only published study similar to ours. The fact that the LMP of all our patients with postoperative breast striae started >14 days before surgery may relate to the disproportionate number of patients (65%) who had surgery during this part of the menstrual cycle. A contributing factor may be oral contraceptive use, which would increase the duration between cycles. Furthermore, the strong

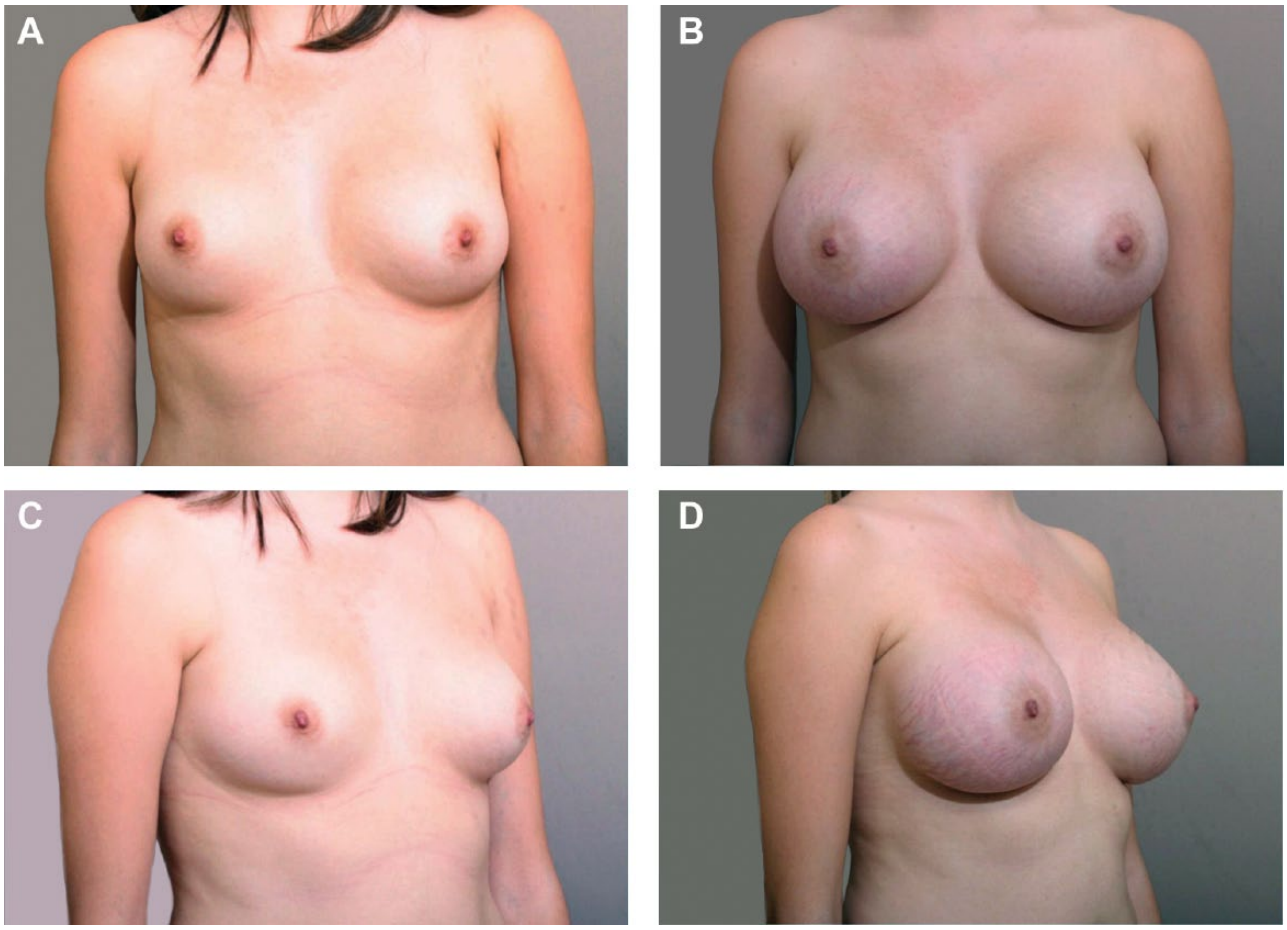

Figure 10. (A, C) This 19-year-old woman with micromastia underwent bilateral augmentation mammaplasty with 432-cc saline implants (Mentor Worldwide LLC, Santa Barbara, California). (B, D) Photographs obtained 3 months postoperatively show diffuse global striae of the entire breast. Onset of striae occurred 56 days postoperatively.

correlation between nulliparity and striae formation may be attributable to selection bias, as women younger than 25 years are more likely to be nulliparous. We did not obtain data for older women and thus cannot deduce whether parity alone, without youth, may be protective. However, this factor alone probably does not account for the majority of the risk, and no nulliparous patient older than 25 years has developed striae in the senior surgeon's practice (C.L.M.).

According to a survey of the American Society for Aesthetic Plastic Surgery (ASAPS) conducted in 2007,[16] the mean implant size used by most surgeons (81%) ranges from 300 to 400 cc. Our mean size was 446 cc for silicone implants and 486 cc for saline implants. We initially hypothesized that a correlation would exist between implant size and the occurrence of breast striae, but our data showed no significant correlation. Our higher-than-average implant size may reflect a regional preference as well as the senior surgeon's practice of routinely overinflating saline implants by 15% beyond the size selected by the patient preoperatively, or similarly increasing the size of silicone implants, all with the patient's understanding and consent.

Our findings provide further evidence of a potentially significant hormonal influence in the development of breast striae after breast augmentation. Our primary goal is to better inform patients about this potential complication. To that end, our results provide a clearer picture of which breast augmentation patients have a greater risk for this complication, and we can now approximate this risk. The lack of detailed postoperative follow-up in our study is a limiting factor in terms of furthering patient education. We did not obtain patient satisfaction scores or document postoperative interviews for our patients with breast striae. It would be beneficial to obtain data on the degree to which patients are concerned about striae and the effects on daily

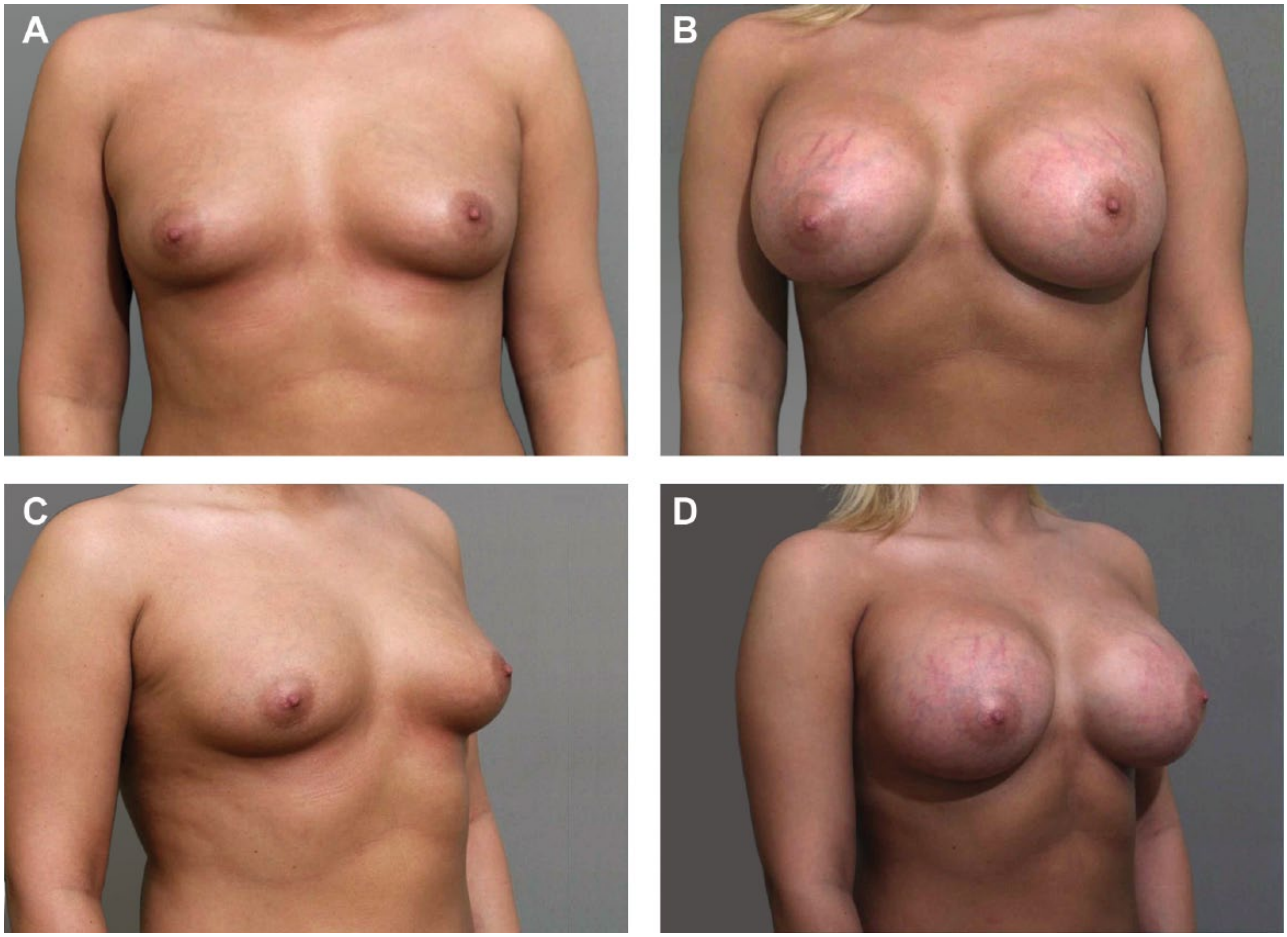

Figure 11. (A, C) This 20-year-old woman with micromastia underwent bilateral augmentation mammaplasty with 490-cc saline implants (Mentor Worldwide LLC, Santa Barbara, California). (B, D) Photographs obtained 4 months postoperatively show early inflamed striae. Onset of striae occurred 82 days postoperatively.

life. Further investigation is needed to elucidate the underlying causes of breast striae and to minimize and ultimately prevent their occurrence.

CONCLUSIONS

Breast striae after cosmetic breast augmentation is a concern for patients and surgeons. The etiology of breast striae appears to be multifactorial and includes mechanical, hormonal, and genetic factors. Our findings demonstrate a strong correlation between hormonal imbalance and new-onset breast striae following cosmetic breast augmentation. The striae developed only in patients aged ≤25 years (peaking at age 19), all of whose last menses occurred >14 days before surgery, and there was a significant association with nulliparity. The greater likelihood of new-onset striae in these patients may relate to higher exposure to estrogen, growth hormone, and steroid products and/or a greater preponderance of receptors that facilitate striae formation. A genetic component is suggested by the significant association with a history of striae elsewhere on the body. This information will serve to better educate and prepare patients during preoperative consultations.

Authors' Note

Previous Presentation: This article was the winning submission for the Resident and Fellows Forum Research Presentation at The Aesthetic Meeting 2013; April 11-16, 2013; New York, NY.

Disclosures

The authors declared no potential conflicts of interest with respect to the research, authorship, and publication of this article.

Funding

The authors received no financial support for the research, authorship, and publication of this article.

REFERENCES

1. Cosmetic Surgery National Data Bank: statistics 2012. *Aesthetic Surg J*. 2013;33(2)(suppl):1S-21S.
2. Zheng P, Lavker RM, Kligman AM. Anatomy of striae. *Br J Dermatol*. 1985;112:185-193.
3. Singh G, Kumar LP. Striae distensae. *Indian J Dermatol Venereol Leprol*. 2005;71:370-372.
4. Pinkus H, Krech MK, Megregan AH. Histopathology of striae distensae, with special reference to striae and wound healing in the Marfan syndrome. *J Invest Dermatol*. 1966;46:283-292.
5. Bhangoo KS, Qiunlivan JK, Connelly JR. Elastin fibers in scar tissue. *Plast Reconstr Surg*. 1976;57:308-313.
6. Ebert MH. Hypertrophic striae distensae. *Arch Dermatol Syphiol*. 1933;28:825.
7. Breathnach AS. Ultrastructure of epidermis and dermis in striae atrophicae. In: Moreti G, Rebora A, eds. *Striae Distensae*. Milan, Italy: Brocades.
8. Cordeiro RCT, Zechin KG, Moraes AM. Expression of estrogen, androgen, and glucocorticoid receptors in recent striae distensae. *Int J Dermatol*. 2010;49:30-32.
9. Osman H, Rubeiz N, Tamim H, Nassar A. Risk factors for the development of striae gravidarum. *Am J Obstet Gynecol*. 2007;196(1):62.e1-62.e5.
10. Burrows NP, Lowell CR. Disorders of connective tissue. In: Burns T, Breathnach S, Cox N, Griffith C, eds. *Rooks Textbook of Dermatology*. Malden, MA: Blackwell Science; 2004:46-47.
11. Thomas RGR, Liston WA. Clinical association of striae gravidarum. *J Obstet Gynecol*. 2004;24:270-271.
12. Maia M, Marcon CR, Rodrigues SB, Aoki T. Striae distensae in pregnancy: risk factors in primiparous women. *An Bras Dermatol*. 2009;84(6):599-605.
13. Elsaie ML, Baumann LS, Elsaaiee LT. Striae distensae (stretch marks) and different modalities of therapy: an update. *Dermatol Surg*. 2009;35:563-573.
14. Rosenthal DB. Striae atrophicae cutis. *Lancet*. 1937;232:557-560.
15. Basile FV, Basile AV, Basile AR. Striae distensae after breast augmentation. *Aesthetic Plast Surg J*. 2012;36:895-900.
16. American Society for Aesthetic Plastic Surgery. Annual meeting survey. March/April 2009. Available from: www.plasticsurgery.org. Accessed May 2013.