Retrospective Case Review of Capsular Contracture After Two-Stage Breast Reconstruction

Is Colonization of the Tissue Expander Pocket Associated With Subsequent Implant Capsular Contracture?

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Abstract: Periprosthetic capsular contracture is a common problem associated with implant-based breast reconstruction. The purpose of this study was to determine if bacterial colonization of the tissue expander contributes to contracture of the permanent implant. Medical records were reviewed for 86 patients (124 tissue expanders) between 1997 and 2001 in 1 institution. Three specimens taken from the expander were cultured. The overall incidence of colonization was 42.7%; 49.4% (38.8–60.0) of immediate and 28.2% (14.1–42.3) of delayed expanders had at least 1 positive culture site (P = 0.043). The most common organisms were *Propionibacterium acnes* (57.6%), *Staphylococcus epidermidis* (31.0%), and *Peptostreptococcus* (5.8%). Statistical analysis revealed no significant difference between colonization of the expander and capsular contracture of the permanent prosthesis (P = 0.59). 45.8% (25.9–65.8) of breasts irradiated preoperatively developed contracture versus 14% (7.2–20.8) with no irradiation (P = 0.0013). These results suggest that colonization of the expander occurs frequently, irradiation predisposes to contracture, and colonization did not contribute to secondary implant contracture in this study population.

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Fibrous contracture of the prosthesis capsule is a relatively common complication following breast reconstruction.[1–3] Etiologic theories have considered periprosthetic infection,[4–7] hematoma,[8] and a host-implant response[9,10] as possible causes. The occurrence of unilateral capsular contracture contradicts the theory that a host-implant reaction is responsible, and many studies have failed to show hematoma as a direct etiologic factor.[8,11,12]

Several studies suggest that bacterial contamination of the periprosthetic capsule may play a role in causing contracture of permanent breast implants.[4–7] The subject of microbial growth, both within the saline medium and the periprosthetic tissue around breast implants, has been addressed by a number of authors. Burkhardt et al[4] cultured a series of capsules at the time of capsulectomy for capsular contracture of breast implants used in augmentation mammaplasty. This group found a positive culture rate of 71%, and an 87% incidence of *Staphylococcus epidermidis*. They suggested that the cause of fibrous capsular contracture is most likely a subacute periprosthetic infection. They also hypothesized that the source of the pathogen may be from the breast ducts, a claim which has been substantiated by studies examining the bacteriology of nipple secretions.[6]

Another possible source of capsular colonization is translocation of bacteria introduced to the lumen from skin during the expansion phase of 2-stage reconstruction. A number of authors have reported viability of bacteria within the intraluminal saline of explanted devices.[13–18] Truppman et al[15] cultured *S. epidermidis* in the saline of an implant in place for 5 years. Nordstrom[16] described growth of *Serratia marcescens* in the saline of tissue expanders. Liang et al[17] described a patient in which cultures from within and external to a tissue expander grew *Pseudomonas aeruginosa*. Peters et al[18] described cultures from the saline within Simaplast implants which grew *S. cohnii*, *Proprionibacacterium acnes*, and *Diphtheroid* species. The possibility of transmigration of pathogens from the lumen to the periprosthetic environment has also been addressed.[19] Liang et al[17] employed scanning electron microscopy to show that the expandable silicone membrane of tissue expanders is impermeable to bacteria; however, holes within the tissue expander port made by

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injection needles of recommended size were large enough to allow bacterial to translocate between the intraluminal and periprosthetic environments.

Bacteria may colonize the capsule surrounding the tissue expander whether the route is from the breast ducts or from migration of intraluminal bacteria introduced during filling of the device.[16] There have been 2 studies in the literature which have studied the incidence of bacterial contamination in a series of tissue expanders.[20,21] Becker and Hartman[20] studied the intraluminal fluid of 45 tissue expanders and found a 0% incidence of contamination. Similarly, Brown et al[21] cultured the saline from 32 tissue expanders and found no evidence of microbial contamination. To date, no studies have addressed the question of whether contamination of the tissue expander may have an effect on the permanent prosthesis used in 2-stage breast reconstruction. As bacterial contamination has been suggested as a plausible cause of capsular contracture, device colonization during the tissue expansion stage may contribute to capsular contracture of the secondary implant.

The purpose of this study was to determine if colonization of the tissue expander is significant and if colonization of this device correlates with subsequent increased incidence of capsular contracture of the second prosthesis.

MATERIALS AND METHODS

Patients

All patients undergoing 2-stage breast reconstruction following mastectomy for breast carcinoma or prophylaxis against breast carcinoma between 1997 and 2001 were included in the study. A total of 86 patients and 124 tissue expanders were submitted for study.

Patients included in this study (Tables 1, 2) were between the ages of 25 and 71, with a mean age of 48.9 ± 9.1 years (47.0–50.9) at the time of tissue expander removal. Fifty-six percent of reconstructions were unilateral and 44% were bilateral procedures. Eighty-five tissue expanders were inserted at the time of mastectomy, and the remaining 39 were inserted after a delay ranging from 5 months to 13 years, with an average delay of 30.9 ± 25.4 months (22.7–39.1). There was one case of frank clinical infection of a tissue expander in which the tissue expander incisions dehisced and the prosthesis was replaced; 19.4% (n = 24) of the reconstructed breasts were irradiated preoperatively. The average follow-up time was 12.1 ± 7.1 months (9.9–11.9).

TABLE 1. Patient Characteristics

Variable	Mean ± SD (95% CI) Percent (95% CI)
Age at removal of tissue expander, y	49 ± 9 (47–51)
Follow-up, mo	12 ± 7 (11–14)
Delay time, mo	31 ± 25 (23–39)
Immediate procedures, %	68.5 (60.4–76.7)
Delayed procedures, %	31.5 (23.3–39.6)
Breasts exposed to preoperative irradiation, %	19.4 (12.4–26.3)

CI, confidence interval.

Procedural Technique

The mastectomy, if done on an immediate basis, was typically done via a skin-sparing elliptical incision. In immediate cases, the tissue expander was placed in a total submuscular pocket and in delayed cases a subpectoral pocket. The pocket in both cases was irrigated with half-strength Betadine followed by saline until the returns were clear prior to placement of the tissue expanders. Drains were used and remained in place until the volume of drainage decreased to less than 35 mL/24 hours. All patients received perioperative antibiotics until their drains were removed. Patients received a cephalosporin unless allergic to penicillin, in which case they received clindamycin. All tissue expanders were filled at the time of insertion via a sterile closed filling system to a volume determined by the quality of the overlying skin flaps. The patients then began postoperative expansion 3 to 4 weeks following insertion. The expansion was carried out every 2 weeks under sterile filling conditions. The second-stage procedure was performed 2 months following the end of the expansion phase. At the time of second-stage surgery, the patient was given a single intraoperative dose of antibiotics after the 3 routine cultures had been obtained. All second-stage implants were smooth-walled saline devices except for 3 cases late in the study period, in which McGhan 410 cohesive gel implants were placed.

Specimen Collection

At the time of explantation 3 specimens were taken. These included intracapsular fluid, scrapings of the biofilm of the implant surface, and a 1 × 1 cm specimen of capsule. These were examined under the supervision of a microbiologist for gram smear and aerobic and anaerobic cultures.

Specimens were centrifuged at 3000*g* for 10 minutes. The sediment was then used to prepare direct stains and inoculate agar plates. Each specimen was examined microscopically for gram stain. Aerobic cultures were plated on 5% sheep blood agar, and MacConkey agar with Crystal Violet. Blood agar and MacConkey agar were incubated at 35°C in 5% CO_2. Anaerobic cultures were plated on colistin nalidixic acid agar enriched with vitamin K. Plates were incubated anaerobically at 35°C. All plates were cultured for 5 days.

Colonization of the tissue expander device was considered positive if any one of the 3 sites examined produced a

TABLE 2. Patient Characteristics by Group

Group	Age, y Mean ± SD (95% CI)	Procedure		Delay Time, mo Mean ± SD (95% CI)	Follow-up, mo Mean ± SD (95% CI)
		Delayed (%)	Immediate (%)		
I	45 ± 7 (38–52)	11	89	Not applicable (n = 1)	12 ± 6 (7–18)
II	50 ± 10 (47–53)	44	56	31 ± 32 (16–45)	11 ± 5 (10–12)
III	46 ± 9 (43–49)	23	77	38 ± 20 (19–56)	11 ± 6 (9–13)
IV	51 ± 7 (47–55)	25	75	24 ± 10 (8–40)	10 ± 3 (8–12)

CI, confidence interval.

positive bacterial culture after a maximum of 5 days incubation.

Chart Review

A retrospective review of the clinical charts of all patients included in the study was performed. Follow-up visits after the second stage of reconstruction were scheduled at 2 weeks, 2 months, 6 months, and then yearly. The average follow-up time was 12 ± 7 months. At each visit, a subjective rating of contracture was recorded by the responsible surgeon. Baker's classification of capsular contracture, modified to include the results of prosthetic breast reconstruction, was used as the basis for this clinical assessment.[19] The presence of capsular contracture of class II to IV at any time point during follow-up was recorded. The microbiology records for each patient were reviewed, and a positive culture from at least 1 of the 3 specimens taken at the time of explantation was recorded. Patients were then grouped according to presence or absence of colonization of the tissue expander and presence or absence of capsular contracture (Table 3). Preoperative irradiation was also recorded.

Statistical Methods

Data were grouped based on infection by any organism and presence or absence of capsular contracture (Table 3). For these 4 groups, 1-way analysis of variance was used to compare patient age, follow-up time, and delay time. We found that these patient characteristics were statistically homogeneous across the 4 groups. Continuous variables were presented as mean ± SD and range. Categorical variables were evaluated by χ^2 statistics. Ordinary Pearson χ^2 was applied for the 4 groups related to variables of interest and χ^2 statistics with Yates correction was used for the 2 by 2 contingency table analysis. All variables were reported with their 95% confidence intervals. A set level of significance, $\alpha = 0.05$, was chosen for decision making. Logistic regression was employed to analyze the effect of irradiation and infection on capsular contracture. The SPSS Version 11 (Chicago, IL) statistical software package was used to evaluate the data.

TABLE 3. Colonization of Tissue Expanders by Any Organism and Corresponding Capsular Contracture of the Permanent Implant

Group			Tissue Expanders, n	Number Receiving Radiotherapy
Number	Colonized	Contracture		
I	+	+	9	2
II	−	−	55	8
III	+	−	44	5
IV	−	+	16	9

RESULTS

Of the 85 tissue expanders inserted immediately, 49.4% (38.8–60.0) were culture positive. Of the 39 delayed specimens, 28.2% (14.1–42.3) were culture positive ($P = 0.043$). The organisms cultured (Fig. 1) included *Propionibacterium acnes* (57.6%), *S. epidermidis* (31.0%), *Peptostreptococcus* (5.8%), *Bacillus* species (2.9%), *Diphtheroid* species (0.9%), *Enterococcus* (0.9%), and *Viridans streptococcus* (0.9%);

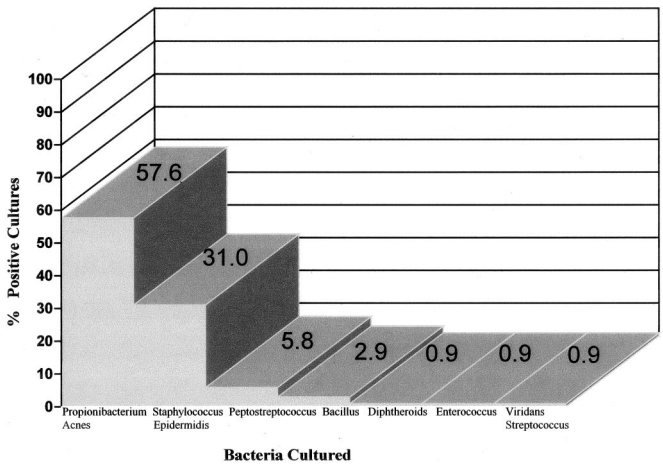

FIGURE 1. Frequency of bacteria cultured from tissue expanders.

47.9% of the positive cultures were from the tissue expander scrapings, 30.2% from the capsule specimen, and 21.8% from the intracapsular fluid. Scrapings of the tissue expander biofilm appeared to be the most sensitive of the culture techniques.

In this study, there were 25 cases of class II to IV contracture, for an overall incidence of 20.2% (n = 25/124). Of those patients developing capsular contracture, 16% were bilateral and 84% were unilateral. This pattern is consistent with random distribution of a breast-based phenomenon as reported in Burkardt et al.[7,22] In the absence of irradiation, the overall rate of capsular contracture was 11.2% (n = 14/124).

Overall, 17% (6.9–27.1) of breasts with positive culture versus 22.5% (12.8–32.3) of breasts with negative culture developed capsular contracture. Statistical analysis revealed no significant difference between culture-positive and culture-negative tissue expanders when compared with capsular contracture of the permanent prosthesis ($P = 0.59$).

Of the breasts in this study, 19.4% (n = 24) were irradiated prior to 2-stage reconstruction. When compared with capsular contracture, 45.8% (n = 11/24) of breasts exposed to preoperative irradiation developed capsular contracture, versus 14.0% (n = 14/100) of breasts with no previous irradiation.

Stepwise logistic regression was employed to compare the effect of both infection and radiation on capsular contracture. This analysis again revealed that positive culture was not a significant predictor for capsular contracture ($P = 0.75$), whereas radiation was a significant predictor ($P = 0.001$).

Of the 25 breasts that developed capsular contracture in this study, 42% (n = 11) required capsulectomy and replacement of the permanent prosthesis. Of these, 27.2% (n = 3) had been irradiated prior to reconstruction. The remaining patients elected not to have revisions despite having some degree of capsular contracture.

DISCUSSION

Periprosthetic capsular contracture continues to be a difficult problem associated with breast reconstruction. A number of etiologic theories have been studied in an effort to prove factors such as infection, premastectomy irradiation, duration of the prosthesis operation, the patient's menopausal state, and the presence of hematoma as causal to capsular contracture.[23] To prevent this complication, it is necessary to identify the primary mechanism responsible. This study examined periprosthetic colonization of the tissue expander and its possible role in contributing to capsular contracture of the permanent implant placed at the second stage of breast reconstruction.

The data presented in this study did not support the hypothesis that colonization of the tissue expander contributes to capsular contracture of the secondary implant ($P = 0.59$). In fact, in our sample, the incidence of contracture was somewhat higher with negative culture (n = 16/71, 22.5%) versus with positive culture (n = 9/53, 17%). Colonization of tissue expanders does appear to occur frequently, especially in tissue expanders inserted immediately in conjunction with mastectomy; 49.4% (n = 42/85) of the immediate tissue expanders in this study were colonized at the time of explantation, and 28.2% (n = 11/39) of delayed specimens were culture positive. Of these, 88% were colonized by *P. acnes* or *S. epidermidis,* which are the primary skin organisms. Similar pathogens were identified in a study by Peters et al,[24] which examined 186 silicone implants removed between 1992 and 1995. The authors reported a 42% incidence of capsular colonization. Organisms cultured in this study included *S. epidermidis*, *Diphtheroid species*, *Streptococcus species*, *Proprionibacterium acnes*, and *Enterococcus*. This study also showed that capsular contracture was not significantly associated with capsular colonization ($P > 0.05$).

Within our study population, periprosthetic infection requiring operative removal of the device occurred in only 1 patient. This suggests that although subacute colonization of tissue expanders is common, it is rarely associated with clinically evident infection. The source of the organisms which colonize the periprosthetic environment is yet to be proven, but inoculation from the skin during filling or from the breast ducts during placement of the tissue expander are possibilities.

Our data confirm the findings of previous studies showing that preoperative irradiation increases the likelihood of capsular contracture[25]; 19.4% (n = 24) of breasts in this study were irradiated prior to 2-stage reconstruction, and 45.8% (n = 11/24) of breasts exposed to irradiation developed capsular contracture versus 14.0% (n = 14/100) with no previous irradiation ($P = 0.0013$). The overall incidence of contracture in this study was 20.2% (n = 25/124). This compares closely to the contracture rates reported in the literature (12–29%) following 2-stage reconstruction.[1–3] In the absence of irradiation, the overall rate of capsular contracture was 11.2% (n = 14/124). Although capsular contracture was more common in the irradiated patients, this did not appear to increase the rate of subsequent need for revisionary surgery in this study population. Irradiation of the breast prior to reconstruction is an obvious confounder when studying variables contributing to contracture. Logistic regression was employed to account for this effect, and colonization of the tissue expander was still found to be a nonsignificant predictor of capsular contracture of the permanent prosthesis.

The data generated in this study suggest that the presence of bacteria in the periprosthetic environment around the tissue expander is not associated with an increased incidence of capsular contracture of the permanent implant. This study has shown that the periprosthetic environment of tissue expanders is colonized frequently, and whether this has any long-term negative effects on the patient has yet to be proven.

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