

The Infected Breast Prosthesis after Mastectomy Reconstruction: Successful Salvage of Nine Implants in Eight Consecutive Patients

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Background: The use of tissue expanders and permanent implants has an established role in breast reconstruction after mastectomy. Periprosthetic infection, however, represents a known complication. The most conservative approach to severe or recalcitrant prosthetic infection remains removal of the device. However, removal makes subsequent reinsertion and reexpansion more difficult, with less predictable cosmetic results. The authors believe that timely surgical intervention directed toward salvage of infected breast prostheses can be successful, without demonstrating increased capsular contracture.

Methods: The authors present nine consecutive cases of infected breast implants (nine implants in eight patients). All patients had previously undergone mastectomy for malignancy and immediate expander/implant reconstruction. Six patients had localized infections that failed to respond to oral antibiotics and two women initially presented with systemic infection. All patients were placed on intravenous antibiotics followed by drainage of fluid, manual debridement and curettage of the infected pocket, device exchange, and postoperative antibiotics.

Results: All nine infected breast prostheses responded to this approach and currently remain intact and without recurrent infection. Mean time to follow-up for all patients was 14.6 months (range, 10 to 25 months).

Conclusions: In patients with severely infected breast prostheses, timely operative intervention can salvage the previously "unsalvageable" implant; in addition, the surgically replaced implants did not develop severe capsular contractures. Surgical salvage of severely infected breast prostheses after mastectomy is a treatment option that should be considered when dealing with severe or recalcitrant infection in a suitable patient. (*Plast. Reconstr. Surg.* 120: 581, 2007.)

Since the introduction of silicone gel implants in 1962¹ and the subsequent development of saline-filled implants 3 years later, breast implants remain important tools available to the plastic surgeon. In addition, Radovan's² use of a temporary tissue expander following mastectomy helped launch the concept of the two-stage breast reconstruction. As a result, breast reconstruction after mastectomy has become an option for many women. Currently, an estimated 2 million women have breast implants, with approximately 30 percent used for breast reconstruction.

Implant reconstruction can provide women with excellent cosmetic results and low associated morbidity. Complications specific to breast tissue expanders and implants include malposition, extrusion, capsular contracture, and problems with the fill valve. The most feared, and perhaps least understood, complication is infection of the implant-reconstructed breast. Although much is written about the possible cause of these infections and patient risk factors, there remains uncertainty regarding the treatment of these infections. Classic teaching mandates implant removal and delayed reinsertion after the infection clears.³ This approach can be problematic in practice to the patient and to the reconstructive surgeon. Removal of the expander or implants subjects the patient to additional surgical procedures and the associated risks, and makes delayed reinsertion technically more difficult, with less predictable cosmetic results.

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Also, this approach has major psychological implications for the patient. To many women suffering with breast cancer, breast reconstruction is a critical component in the recovery process. Explantation halts the reconstruction process for upward of 6 months. This can be quite demoralizing to the patient, who now must be without an implant, and may weaken her confidence in the reconstructive surgeon. In fact, it is our experience that many women who require explantation secondary to infectious complications often choose not to undergo any future reconstruction. We have reviewed our own experience with infected breast implants after reconstruction for malignancy and feel that early and aggressive surgical intervention can result in implant salvage, even in those previously considered “unsalvageable.”

PATIENTS AND MATERIALS

A retrospective review of nine consecutive cases of breast implant infection presenting from 2003 to 2005 was performed (nine implants in eight patients). All infected patients had previously undergone immediate reconstruction by the lead author (J.K.C.) after mastectomy for malignancy. The mean age of the patients was 47.3 years (range, 35 to 64 years). None of the eight women had a history of diabetes mellitus. One woman had a distant history of tobacco use (patient 3) and one admitted to smoking occasionally (patient 2). Seven women underwent immediate insertion of temporary tissue expanders (McGhan MV133; McGhan Medical, Santa Barbara, Calif.), which were subsequently exchanged to a permanent textured implant [six McGhan 363LF and one Mentor Siltex (Mentor Corp., Santa Barbara, Calif.)]. One woman underwent immediate reconstruction with a permanent adjustable implant (Mentor Siltex). Two women required adjuvant chemotherapy and one woman required both chemotherapy and radiotherapy.

Five women also underwent contralateral simple mastectomies for prophylaxis or symmetry. Three of these women suffered an infection on the side of the carcinoma; all had undergone axillary lymph node sampling on this side. One woman had an infection in the contralateral or prophylactic side and one woman had bilateral implant infections; neither of these two women underwent axillary lymph node sampling, given the pathologic tumor.

Regarding the nine infected breast prostheses, one was an infected tissue expander near the end of the expansion process, one was an adjustable

implant 4 months after achieving the target volume, and the remaining seven were textured permanent implants. One patient presented with bilateral infected permanent implants (patient 8). The patients are summarized in Table 1.

On initial presentation, six women demonstrated only signs of localized infection that included cellulitis, edema, and tenderness. Five of these women failed to have any improvement in local signs with oral antibiotics, whereas one woman (patient 6) progressed to signs of systemic infection, which included fever, chills, and malaise. Two women (patients 4 and 8) initially presented with both localized and systemic signs of infection. No woman had an exposure or threatened exposure of her implant. The clinical features of the eight patients are listed in Table 2.

These eight patients, with local infection recalcitrant to oral antibiotics or systemic infection, were admitted to our institution under the care of a single reconstructive surgeon. Routine blood work was performed, which included complete blood count, chemistry studies, erythrocyte sedimentation rate, coagulation studies, and blood and urine cultures. All patients were immediately started on intravenous vancomycin after cultures were obtained. In addition, four patients underwent computed tomographic scanning to help determine the presence and nature of any periprosthetic fluid. One patient underwent blunt needle aspiration of periprosthetic fluid.

All eight patients were taken to the operating room. The infected side was explored and seroma fluid was evacuated. Sample fluid was sent for detection of bacteria, fungi, and acid-fast bacilli. The implant was removed and the capsule was mechanically abraded with gauze on a sponge stick or curetted to remove any fibrinous material or infectious peel. The pocket was irrigated copiously with 6 to 9 liters of normal saline administered by means of pulse lavage. No formal capsulectomy was performed. After changing to a sterile setup, a new implant, usually the exact style and size as the one removed, was placed in the pocket. Two flat drains were placed and the wound was closed in layers. The patients remained on intravenous antibiotics during the immediate postoperative period while awaiting microbiologic results. All patients were followed by infectious disease consultants, who usually recommended a total of 4 weeks of postoperative antibiotics.

RESULTS

The above technique was performed in eight consecutive patients with nine infected breast

Table 1. Summary of Cases of Infected Implants

Patient	Age (yr)	Implant Type	Unilateral or Bilateral Reconstruction		ALNS*	Chemotherapy	Radiation	Onset to Infection†	Organism	Length of Stay (days)	Follow-Up (mo)	Baker Classification (grade)
			Unilateral	Bilateral								
1	35	Permanent implant	Bilateral	Bilateral	Yes	Yes	No	10 days	No growth	4	25	I
2	41	Permanent implant	Bilateral	Bilateral	Yes	Yes	No	9 wk	No growth	11	16	II
3	64	Permanent implant	Bilateral	Bilateral	Yes	No	No	20 days	<i>Staphylococcus epidermidis</i> †	9	16	III
4	57	Tissue expander	Bilateral	Bilateral	Yes	Yes	No	5 wk	<i>Staphylococcus aureus</i>	6	15	I
5	41	Adjustable implant	Unilateral	Unilateral	Yes	Yes	Yes	11 mo	No growth	3	13	III
6	42	Permanent implant	Unilateral	Unilateral	Yes	No	No	4 mo	<i>Staphylococcus aureus</i>	6	12	I
7	39	Permanent implant	Bilateral	Bilateral	No	No	No	2 mo	<i>Staphylococcus aureus</i>	5	10	I
8	60	Permanent implant	Bilateral	Bilateral	No	No	No	6 mo	<i>Enterococcus faecalis</i>	3	10	I

ALNS, axillary lymph node sampling.

*On the side with carcinoma.

†Defined as time from the time of most recent implant insertion to clinical signs of infection.

‡Obtained from operative biopsy of thickened capsule; periprosthetic fluid had no growth.

prostheses. On exploration, all nine infected prosthesis were intact and all demonstrated a significant amount of periprosthetic fluid, ranging from slightly turbid to cloudy. The fluid collections were often loculated and surrounded by a gelatinous layer or organized peeled requiring abrasion and curettage. No hematomas were seen in the implant pocket. One patient did have a localized abscess in the axilla that was in continuity with the implant capsule (patient 4). The cultures of the periprosthetic fluid were positive for *Staphylococcus aureus* in three patients and *Enterococcus faecalis* in one patient. In one patient, the capsule was markedly thickened and a small piece was sent for examination. The pathologic findings demonstrated inflammatory changes, and the culture of this tissue was positive for *Staphylococcus epidermidis*; the seroma fluid, however, in this patient demonstrated no growth of organisms.

Postoperatively, most patients reported subjective improvement in pain on postoperative day 1. Over the next several days, the erythema and tenderness of the breast decreased and the white blood cell count normalized. The erythrocyte sedimentation rate was the slowest to normalize, taking up to 3 weeks. All patients remained on intravenous antibiotics during the hospitalization. The mean length of stay was 5.8 days (range, 3 to 11 days). Three patients (patients 2, 6, and 7) were discharged on intravenous antibiotics for 4 weeks, whereas the others were administered oral antibiotics for 4 weeks. Each was followed by an infectious disease consultant, who made antibiotic recommendations on a case-by-case basis.

All patients responded to this approach and had successful salvage of the infected breast prostheses. All patients currently remain with an intact implant and without signs or symptoms of infection. Six of nine implants remain free of capsular contracture (Baker grade I) on long-term follow-up. One patient (patient 3) demonstrates Baker grade III capsular contracture in both the salvaged implant and the contralateral, noninfected side. Another patient (patient 2) also displays Baker grade II capsular contracture of both the salvaged and noninfected implants. The third patient (patient 5) underwent irradiation to the reconstructed side and demonstrates Baker grade III capsular contracture. Mean time to follow-up for all patients was 14.6 months (range, 10 to 25 months).

Table 2. Clinical Features of Cases*

Patient*	Temperature >38.5°C	White Blood Cell Count ($\times 1000/\text{mm}^3$)	ESR (mm/hr)	Failed Oral Antibiotics†
1	No	6.2	NA	Yes
2	No	10.3	96	Yes
3	No	7.4	21	Yes
4	Yes	9.5	NA	No‡
5	No	6.4	35	Yes
6	Yes	11.3	60	Yes
7	No	6.6	44	Yes
8	Yes	15.3	18	No‡

ESR, erythrocyte sedimentation rate (0–20 mm/hr).

*All eight patients had cellulitis, edema, and tenderness.

†Defined as no improvement in localized signs of infection or progression to systemic signs of infection.

‡Initial presentation was of systemic infection and the patient was placed on intravenous antibiotics immediately.

CASE REPORTS

Case 1: Persistent, Indolent Infection

A 64-year-old woman (patient 3) underwent a right modified radical mastectomy with sentinel lymph node biopsy and a left simple mastectomy in August of 2003. McGhan 133MV (400 cc) expanders were placed bilaterally and filled to 50 cc. Within 3 weeks, the patient developed slight erythema of the right breast that resolved with a short course of oral cephalexin, and the expansion process was reinitiated. The erythema returned approximately 1 month later but again responded to a short course of cephalexin. Her expanders were inflated successfully and she underwent a bilateral implant exchange to McGhan textured 363LF saline implants.

She did well for approximately 2 months until the erythema returned on the right and she was again placed on oral antibiotics. The erythema waxed and waned for the next 4 months. During this time, however, the patient remained well, without fever or chills, and her right breast was not swollen or painful.

In May of 2004, her right breast became significantly swollen and cellulitic (Fig. 1). A computed tomographic scan showed



Fig. 1. Case 1. Preoperative view of a 64-year-old woman (patient 3) with an infected right breast implant 9 months after mastectomy. Note the erythema in the lower portion of the right breast. This infection had waxed and waned, and had failed to respond to multiple courses of oral antibiotics.

a fluid collection around the right breast implant. She was admitted to the hospital and underwent blunt needle aspiration of this fluid and placement of a drainage catheter. She was started on intravenous vancomycin; however, her symptoms remained unchanged. She was taken to the operating room on hospital day 5 and underwent the procedure described earlier. Of note was a markedly thickened capsule. A limited capsulectomy was performed and the tissue was sent for examination and culture. Because the thickened capsule prevented reapproximation, a postoperative adjustable implant (Mentor Siltex) was inserted and the fill port was externalized.

The patient remained on vancomycin for an additional 5 days and her symptoms improved. During this immediate postoperative period, the implant was expanded to the target volume and the fill valve was removed at the bedside on postoperative day 4. She was discharged to home on a 4-week course of oral linezolid. The fluid from both the operating room and the initial aspiration was negative for bacteria, fungus, and acid-fast bacilli. The results of the biopsy of the thickened capsule demonstrated fibrous tissue with granulation and was positive for *S. epidermidis*.

Over the next few weeks, the erythema resolved completely and she remained symptom-free. She underwent the nipple-areola reconstruction 6 months later. At 16 months' follow-up, both the salvaged and the noninfected implants showed Baker grade III capsular contractures (Fig. 2).

Case 2: Severe Infection

A 42-year-old woman (patient 6) had a history of right breast cancer for which she underwent a modified radical mastectomy in 1999 and delayed latissimus dorsi with implant reconstruction in 2001. In May of 2004 she was diagnosed with left breast cancer and underwent a left modified radical mastectomy with sentinel lymph node biopsy followed by immediate insertion of a tissue expander (McGhan MVI33). She did well and underwent left implant exchange (McGhan textured 363LF) and nipple reconstruction over a 6-month period.

Several weeks after nipple tattooing, she experienced redness and pain in her left breast while vacationing out of the country. She was placed on oral antibiotics but reported the onset of fever and chills and worsening of her left breast symptoms. She returned immediately to the United States and, on examination, her left breast was cellulitic and swollen (Fig. 3). She also had a fever to 38.5°C and an elevated white blood cell



Fig. 2. Case 1. Patient 3 at 16 months after implant exchange of an infected right breast implant. The patient demonstrates Baker grade III capsular contracture bilaterally. Note that the degree of capsular contracture is the same as on the contralateral, noninfected side.



Fig. 3. Case 2. Preoperative view of a 42-year-old woman (patient 6) with an infected left breast implant 6 months after mastectomy. She had a previous latissimus dorsi and implant reconstruction of the right breast. Note the severely swollen and cellulitic left breast.

count and erythrocyte sedimentation rate, but remained hemodynamically stable. She was immediately given intravenous vancomycin and was taken to the operating room.

She underwent incision and drainage that revealed 275 cc of turbid periprosthetic fluid. A new permanent saline implant (McGhan textured 363LF), identical to her previous implant, was inserted and inflated to 580 cc. Her fever resolved immediately and her white blood cell count trended to normal over the next several days. She remained on vancomycin, her cellulitis resolved, and she was discharged to home 6 days later. She continued vancomycin while at home for 2 weeks and then oral linezolid for 2 weeks. At 12 months, the implant remains soft, without capsular contracture or signs of infection (Fig. 4).

Case 3: Severe Infection with Sepsis

A 57-year-old woman (patient 4) had a history of left breast cancer and modified radical mastectomy in 1990. In June of 2004, she was diagnosed with a right breast cancer and underwent a right modified radical mastectomy with sentinel lymph node biopsy and immediate insertion of a right breast tissue expander (McGhan MV133, 600 cc). She also underwent delayed insertion of a left breast tissue expander at this time (McGhan MV133, 600 cc). At 5 weeks postoperatively, with her expander filled to 550 cc, she experienced sudden onset of fever and chills. She presented to her local hospital with fever to 39°C and cellulitis of her right breast. By report, she was ill-appearing and was admitted to an intensive care setting. She was placed on intravenous antibiotics (vancomycin and ciprofloxacin) and improved over the next 24 to 48 hours. She remained on intravenous antibiotics for 4 days and continued to improve clinically, and was transferred to our institution.

On arrival, she had cellulitis on the anterior right breast, and cellulitis and fluctuance in the right axilla (Figs. 5 and 6). A computed tomographic scan was obtained to determine the extent of the collection and to determine whether this was primarily an axillary or a periprosthetic process. The computed tomographic scan showed a periprosthetic fluid collection and communication to a larger, enhancing collection in the axilla (Fig. 7). She was taken to the operating room on hospital day 2, where the loculated collection was drained, the pocket was



Fig. 4. Case 2. Patient 6 at 12 months after exchange of an infected left breast implant. The implant is soft and without capsular contracture (Baker grade I).

jet-lavaged copiously with saline, and a McGhan MV133 (600 cc) expander was placed and filled to 300 cc. The periprosthetic fluid grew *S. aureus*. She was treated with intravenous antibiotics (vancomycin) for 4 days and was discharged to home on oral antibiotics (linezolid) for an additional 4 weeks. The erythema resolved completely approximately 2 weeks after discharge, and the expansion process was restarted.

She underwent bilateral implant exchange to permanent saline implants (McGhan textured 363LF) in October of 2004 and had bilateral nipple reconstructions in December of 2004. At 15 months, both implants remain soft, without capsular contracture or infection (Fig. 8).

DISCUSSION

Periprosthetic infection is perhaps the most feared and least understood complication of



Fig. 5. Case 3. Preoperative view of a 57-year-old woman (patient 4) who presented with an infected right breast tissue expander after undergoing right modified radical mastectomy and insertion of a tissue expander. At the same time, implant reconstruction of her left breast was delayed. Note the cellulitis of the right breast.

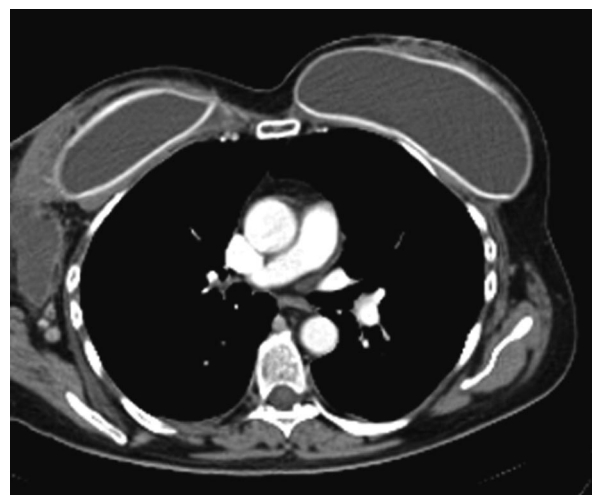


Fig. 7. Case 3. Computed tomographic scan of patient 4 demonstrates right axillary abscess and communication with the breast implant capsule.



Fig. 6. Case 3. The severely cellulitic right axilla of patient 4.

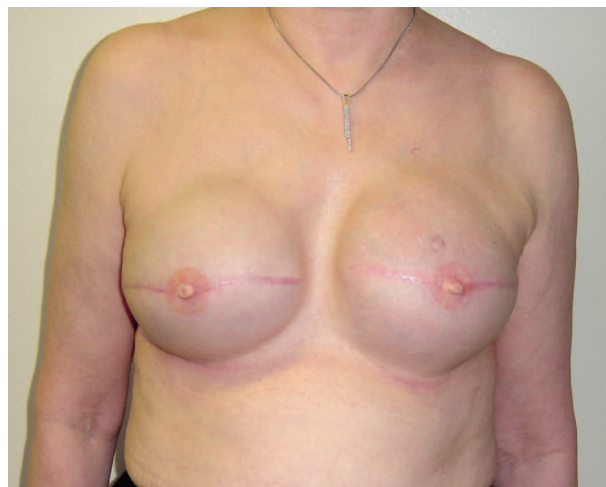


Fig. 8. Case 3. Patient 4 at 15 months after exchange of her infected right breast implant. She has undergone bilateral nipple reconstruction and shows no signs of capsular contracture (Baker grade I).

breast implants. The incidence of periprosthetic infections following breast reconstruction with expanders and permanent implants ranges from 1 to 24 percent.⁴⁻⁹ Armstrong et al. demonstrated a 24 percent infection rate in 33 women who had undergone breast reconstruction with expanders and implants. In a larger series, Disa et al. reviewed 770 women following breast reconstruction with tissue expanders and demonstrated premature removal of the implant attributable to infection in 1 percent of the women. Spear's study demonstrated a similar incidence, with 1.2 percent of 171 expanders requiring removal secondary to infection. More recently, Nahabedian et al. reviewed 130 women who had undergone breast recon-

struction and demonstrated periprosthetic infections in 7.7 percent of the women and 6 percent of the implants.

Periprosthetic infection following augmentation mammoplasty has a reported incidence ranging from 1.7 to 2.5 percent.^{10,11} This is significantly lower than the incidence of periprosthetic infection following breast reconstruction. There may be factors unique to the reconstructive patient that account for the increased rate of infectious complication. Krueger et al.¹² investigated the effect of radiation therapy on implant reconstruction and reported an infection rate of 37 percent

in the irradiated group versus 19 percent in the nonirradiated group. This association between radiation and infection was supported by Nahabedian et al., who found that the infection rate in implants exposed to radiation was 4.88 times greater than in those not irradiated. The same study also found a suggestion ($p < 0.09$) that the incidence of infection was 6.29 times greater when lymph nodes are removed. However, no statistically significant association was found between implant infection and age, diabetes, tobacco use, tumor stage, timing of implant insertion, and chemotherapy. In our series, six of eight patients had undergone lymph node removal. Of these women, three underwent unilateral axillary lymph node sampling with bilateral reconstruction. In all three women, the infected implant occurred on the side of the axillary lymph node sampling.

Although we may never truly understand the cause of breast implant infections, the major question remains: What is the best way to treat an infected implant, regardless of the cause? Classic teaching holds that periprosthetic infection mandates implant removal and delayed reinsertion after the infection clears. However, the concept of implant salvage has become more acknowledged as of late. It is important to note that the concept of implant “salvage” does not refer to the retention of the original prosthetic device; rather, it refers to maintaining the results of the original operation and includes implant exchange.¹³

This concept of salvaging prosthetic devices is not unique to plastic surgery. In fact, salvage of infected prosthetics has become increasingly more successful within other surgical subspecialties. The reported salvage rate for prosthetic vascular grafts exceeds 70 percent when operative debridement is combined with a vascularized muscle flap,¹⁴⁻¹⁶ and the urology literature reports a 70 percent salvage rate in infected penile implants.¹⁷⁻¹⁹ Although the reported salvage rates for infected joint prosthesis is more variable, attempted salvage is still regarded in the orthopedic community as an appropriate treatment strategy.²⁰⁻²²

The first report in the literature describing the successful salvage of a breast implant with periprosthetic infection after augmentation mammoplasty was in 1965.²³ Later, reports by Courtiss et al.¹⁰ and Wilkinson et al.²⁴ further demonstrated the ability to salvage infected implants in the context of breast augmentation and implant insertion following subcutaneous mastectomy. The first report of salvage of infected breast prostheses after complete mastectomy for cancer was by Yii and Khoo²⁵ in 2003. In this study, the authors achieved

successful salvage in nine of 14 patients with a regimen that included explantation, mechanical scrubbing and irrigation of the pocket, capsulotomy, new implant insertion, continuous antibiotic irrigation, and intravenous antibiotics. Yii and Khoo define the infected implants as “those with purulent fluid collection around the prostheses, irrespective of bacterial isolations.” However, no mention was made regarding the severity of local infection or the presence of systemic signs and symptoms of infection.

More recently, Spear et al. examined 24 patients with 26 breast implant infections after mastectomy and augmentation. This study attempted to classify the severity of infection on the basis of initial clinical presentation and offer treatment strategies for each category of infection. Spear et al. define “mild infection” as those with minimal, localized erythema at the surgical site or in the skin overlying the implant that was responsive to initial antibiotic therapy. They define “severe infection” as having one or more of the following: persistent swelling despite antibiotics, purulent drainage with or without cellulitis, aggressive or atypical organisms, or evidence of systemic infection. They offer four patients with “severe” infection without exposure of the implant. Two of these infections were following elective augmentation mammoplasty and were successfully salvaged with antibiotics and device exchange. The other two “severe” infections were following breast reconstruction, and these patients were not offered attempted salvage and the implants were removed. All eight of our patients with nine infected prostheses fall within Spear et al.’s definition of “severe infection” without threatened exposure, or “group 2” according to their categorization. We were uniformly successful in salvaging all nine implants in this group.

Four women underwent preoperative computed tomographic scans. We found this helpful in quantifying the amount of periprosthetic fluid and the nature of this fluid. In one case, computed tomography demonstrated an axillary abscess in addition to a periprosthetic infection; this led us to evacuate the axillary collection through a separate incision. Although ultrasonography is less expensive and can often be obtained quicker in most institutions, computed tomography has the advantage of more clearly determining seroma fluid versus abscess and determining the thickness of the implant capsule. In addition, we have found that ultrasonography is often operator dependent and may be uncomfortable to the patient with an infected, tender breast. Accordingly, in our insti-

tution, we favor computed tomography over ultrasonography for preoperative imaging and surgical planning.

Preoperative hospitalization of the patient is helpful in starting broad-spectrum intravenous antibiotics (i.e., vancomycin) and to determine the optimal time for surgical intervention. Postoperatively, hospitalization is important in determining the success of the implant salvage. If the signs of infection persist or the patient's overall condition worsens, the salvage would be unsuccessful and would mandate implant removal.

Intraoperatively, antibiotic solution is used for the new implants and may be used for the implant pocket as well. However, because we feel that the effect of irrigation of the implant pocket is dilution of contamination, we feel that the volume of the irrigant is more important than the presence of antibiotics in the fluid.

The inflammation associated with periprosthetic infections makes performing a formal capsulectomy without increasing the risk of postoperative bleeding technically difficult. For this reason, a capsulotomy was not performed routinely. Instead, curettage of fibrinous material within the capsule serves as an "internal capsulectomy" by eliminating the capsular peel, which subsequently hardens, resulting in a thickened capsule. Capsular curettage also promotes proper adhesion of the new textured implant to the capsule. However, when faced with a markedly thickened capsule, as in patient 3, we do advocate a limited capsulectomy in addition to capsular curettage.

Also critical to the success of this technique is device exchange. Textured implants are most commonly used in postmastectomy reconstructions and were used in all of our cases. We believe that simply washing and soaking the implant in an antibiotic solution may be inadequate in clearing the bacteria that have become imbedded in the countless crevices. Therefore, in addition to surgically "sterilizing" the implant pocket with curettage and irrigation, placement of a new implant gives best chance of preventing recurrence of infection.

All patients were treated for a total of 4 weeks with antibiotics according to the recommendation of infectious disease consultants. The antibiotic choice, route, and duration of treatment are areas that need to be further investigated. In our patients, there has been a trend away from long-term intravenous antibiotics toward newer oral antibiotics with equivalent bioavailability.

Although three patients went on to develop capsular contracture, we feel that the incidence and severity would have been higher if the infection had been treated with antibiotics alone. Although the exact mechanism of capsular contracture formation is not known, it does anecdotally have a higher incidence in the face of prosthetic infection. In fact, we maintain that those three patients developed contractures unrelated to the prosthetic infection: patients 2 and 3 developed equal degrees of capsular contracture on both the infected and noninfected sides, and patient 5 was irradiated postoperatively.

Although the series of patients salvaged with this technique has been uniformly successful, it is important to note that the decision to attempt implant salvage must consider the patient's overall health and oncologic issues such as need for chemotherapy. We maintain that the patient with localized signs of infection recalcitrant to oral antibiotics would benefit from an early attempt at implant salvage. We also maintain that this approach is appropriate for those patients with systemic signs of infection that improve with a short course of intravenous antibiotics. We would never advocate this approach in a septic patient whose overall condition deteriorates despite intravenous antibiotics.

The condition of the soft tissue is also a factor in whether to attempt implant salvage after mastectomy reconstruction. In all of our cases, there were varying degrees of cellulitis and edema. However, no patient demonstrated prosthesis exposure or impending exposure. We feel that this would be reason to forgo attempted salvage. We have encountered one case of frank pus in the implant pocket communicating with an axillary abscess during exploration and device exchange. In general, the presence of pus in the implant pocket combined with a deteriorated soft-tissue condition may indicate that the window of opportunity for implant salvage has passed. We feel that the presence of frank pus with liquefied capsular tissue on exploration makes successful salvage unlikely and explantation should be considered.

CONCLUSIONS

Periprosthetic infection remains a major issue among plastic and reconstructive surgeons. We have described the successful salvage of nine consecutive severely infected breast prostheses after mastectomy reconstruction. We use a technique of immediate intravenous antibiotics followed by early device exchange and a long course of postoperative antibiotics. Moreover, only one of these

salvaged implants demonstrated the severe capsular contracture often seen after implant infections treated with antibiotics alone.

The theme of this article is to share a series of successful salvage of severely infected breast prostheses after mastectomy reconstruction. We hope that our experience challenges the belief that severe infection mandates explantation. However, the decision to intervene surgically in an attempt to salvage severely infected breast prostheses is a difficult one that must consider patient factors, tissue factors, and the need for chemotherapy. Therefore, one cannot attempt to salvage 100 percent of infected implants. It should be a mutual decision of both the patient and the physician to determine the appropriateness of implant salvage. With careful patient selection and vigilant attention by both the patient and the physician, we feel that a timely surgical intervention can prove successful in salvaging implants previously considered unsalvageable.

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DISCLOSURES

The authors have no financial interest in any of the implant devices or antibiotics used in the care of the patients described in this article. The authors have no commercial or other interest with Mentor or Inamed.

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