Mycobacterium fortuitum Infection following Reconstructive Breast Surgery: Differentiation from Classically Described Red Breast Syndrome

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Background: Red breast syndrome (RBS) has been described as an erythema that may be associated with 2-stage prosthetic reconstructive breast surgery using biologic mesh. RBS is differentiated from infectious cellulitis through absence of fever and laboratory abnormalities and usually has a self-limiting course. There have been no clinical reports on etiology, risk factors, or management of RBS. This report describes patient data that raise the need to rule out mycobacterial infection when RBS is being considered as a diagnosis.

Methods: We present 6 cases of *Mycobacterium fortuitum* infection occurring after prosthetic breast reconstruction performed with a human-derived acellular dermal matrix, including the timing and course of erythema, laboratory results, treatments used, and long-term outcomes. We also describe the differential diagnoses of RBS in the context of these cases, including emergence of acid-fast bacilli and diagnostic and treatment considerations. Exact two-tailed 95% confidence intervals based on the F-distribution are provided with estimates of the incidence rates of infection.

Results: The 6 cases presented here do not fit the typical description of RBS and were caused by mycobacterium infection. Statistical evaluation of the estimated incidence rate of *M. fortuitum* infection in a patient thought to have RBS, which occurred 100% of the time in this series, revealed a 95% confidence interval of 54.1–100%.

Conclusions: When presented with possible RBS, surgeons must rule out cellulitis, culture for acid-fast bacilli such as mycobacterium species, and then determine the best course of treatment. Patient counseling regarding potential household sources of infection is warranted to minimize postoperative infection risk. *(Plast Reconstr Surg Glob Open 2013;1:e50; doi: 10.1097/GOX.0b013e3182a939ed; Published online 10 October 2013.)*

Approximately 70,000 2-stage reconstructive breast surgeries using a tissue expander and breast implant were performed in the United States in 2011.[1] These procedures now more commonly involve the use of acellular dermal matrix (ADM) products, which were introduced in 2005.[2] Inserting an ADM to help reinforce the soft-tissue pocket for the expander and the later implant has been shown to provide enhanced soft-tissue coverage of the prosthesis and improve control of the in-

framammary and lateral mammary folds and implant position, resulting in better cosmetic outcomes.[2–5]

In recent years, there have been several reports of a delayed erythema on the reconstructed breast.[6–9] This infrequent finding is reported in 5–10% of breast reconstructions performed with ADM,[8] and it has been called both delayed breast cellulitis[10,11] and red breast syndrome (RBS).[7,9,12] The erythema tends to appear a few weeks or more after surgery on the skin overlying biologic mesh products[7] and then usually resolves spontaneously within 2 months.[7,8] As described in published reports, cases may mimic cellulitis but differ in several respects. In most cases, the reddened area of skin blanches under pressure, but there is no induration. Pain, tenderness, localized calor, and fever are typically absent, as are elevated serum markers, such as white blood cell and neutrophil count, C-reactive protein, and erythrocyte sedimentation rate.[7]

The cause of RBS has not been determined.[7] RBS is reported to occur in the presence of expanders or implants with biologic mesh products such as ADM.[6,8] The condition does not appear to be linked to product sterility or processing.[8] It is not known if any subgroup of patients is at greater risk for RBS or whether radiation and chemotherapy are factors. It is feasible that some cases may represent allergic or inflammatory reactions, unidentified infection, or possibly revascularization of the ADM, but there are no clinical data to support any of these potential causes.[6–8]

Differentiation from infectious cellulitis has been described in the limited volume of literature as a dilemma for surgeons considering a diagnosis of RBS. Although the literature states that RBS does not seem to require any treatment other than watchful waiting, infectious cellulitis or infection in the expander or implant pocket requires more aggressive treatment. In cases of RBS, the surgeon may be forced to remove the expander because the patient has to begin chemotherapy, and the oncologist is

concerned about the erythema being a sign of infection. In many cases, a true diagnosis is never made. Lack of intervention in a scenario of undiagnosed infectious cellulitis could allow progression to a serious infection, necessitating invasive procedures and possibly removal of the expander or implant.[7]

We present 6 cases of acid-fast bacillus (AFB) infection with *Mycobacterium fortuitum* in patients who underwent prosthetic breast reconstruction performed with human-derived ADM (HADM; AlloDerm Regenerative Tissue Matrix; LifeCell, Branchburg, N.J.; DermaMatrix, Synthes, West Chester, Pa.). These cases, in which RBS was a leading diagnosis, occurred over the last 6 years among 3109 reconstructive procedures that involved HADM. No other patients had signs and symptoms characteristic of RBS. The 6 cases reported here do not match previous published descriptions of RBS in several respects; we believe that they may have been caused by mycobacterial infections acquired after surgery that ascended through surgical drain sites. Our objective is to evaluate atypical infections due to AFB, an organism that often eludes detection, in patients in whom RBS is being considered as a diagnosis. The differential diagnoses of RBS, including potential causes and impact on treatment plans, will be discussed in the context of these patients. Exact two-tailed 95% confidence intervals (CI) based on the F-distribution are provided, with estimates of the incidence rates of infection.

TECHNIQUE

All 6 patients in this report underwent mastectomy as part of treatment for breast cancer. Patient demographic information is presented in Table 1, and therapeutic and surgical information is presented in Table 2. Patients underwent expander/implant-based reconstruction following unilateral mastectomy ($n = 2$), bilateral therapeutic mastectomy ($n = 2$), and bilateral mastectomy due to unilateral cancer and contralateral prophylaxis ($n = 2$). One case involved the use of DermaMatrix and 5 cases involved AlloDerm Regenerative Tissue Matrix.

The authors performed 2-stage reconstruction with a standard approach. The first stage was performed under general anesthesia immediately following mastectomy. Patients received prophylactic cefazolin as a single intravenous dose <30 minutes before surgery and cephalexin 500 mg every 6 hours for 2 weeks postoperatively.

HADM (either 8×16 or 8×20 cm) was used as an extension of the released pectoralis major muscle to reinforce the inferior breast flap over a tissue pocket for insertion of a tissue expander.[13] The subpecto-

Table 1. Patient Demographic Information

Case No.	1	2	3	4	5	6
Age, race	52, white	47, white	46, black	54, white	67, white	59, white
Weight (lb)/ body mass index (kg/m²)	145/23.4	190/30.7	250/35.9	104/19.6	201/36.8	182/29.4
Side and stage of breast cancer	Left: clinical stage I (T2N0M0)	Right: invasive ductal carcinoma; clinical stage III (T2N1M0)	Bilateral lobular carcinoma in situ + atypical ductal hyperplasia on left, clinical stage I (T1N0M0)	Right: history of ductal carcinoma in situ and lumpectomy + chemotherapy/ radiation; left: invasive ductal stage I (T1cN0M0)	Right: stage 2A (T1N1M0)	Right: stage IIIA (T3N1Mx)
Radiation or chemotherapy	None	Radiation: none; chemotherapy: cyclophosphamide and docetaxel, 6 cycles; postoperative, starting after expander removed due to infection	Radiation: NA; chemotherapy: none	Radiation: history of radiation to right breast for ductal carcinoma in situ 2004, not concurrent with treatment/ infection; chemotherapy: taxotere, carboplatin, trastuzumab; postoperative, 9/30/11–1/ 2012, 6 cycles + additional trastuzumab cycle	Radiation: postoperative; chemotherapy: 4 cycles of cyclophosphamide/ docetaxel	Radiation: postoperative; chemotherapy: taxotere, carboplatin, trastuzumab, 4 cycles; preoperative (2/2011–3/ 2011) × 4 cycles; postoperative 2 cycles, 8/2011
Comorbidities	Hypertension	Acne, gastroesophageal reflux disease, anxiety	Asthma, endometriosis	NA	Obesity, hypertension	Asthma, tobacco use
Risk factors for infection	NA	Left pneumothorax from chemoport requiring chest tube during postoperative period	Obesity	Radiation, but to breast contralateral to infection site	Axillary dissection, high output Jackson-Pratt drain for 3 wk	Well water, multiple household cats
Time to symptoms (wk) after initial surgery	8	4	5	4	7	4 wk: incision not healing; 7 wk: erythema, edema draining
Patient symptoms/findings		Pain, erythema; post implant removal, chronic nonhealing wound with drainage	Left breast erythema, pain, purulent drainage	Mild erythema (4 wk), worsening erythema (8 wk), calor, edema	Erythema, elevated temperature, serous drainage around Jackson-Pratt drain site, edema	Erythema, edema, drainage, malaise

NA, information not available.

ralis pocket was prepared and then the pocket and wounds were rinsed with antibiotic solution (bacitracin or vancomycin/gentamicin). The sheet of HADM was positioned with the dermal side toward the mastectomy flap and then sutured to the chest wall along the arc formed by the desired location of the inframammary and lateral mammary folds with running 3-0 polydioxanone (PDS) sutures (Ethicon, Somerville, N.J.). The tissue expander was rinsed with the same antibiotic solution used for the wounds and then placed into the pocket deep to the pectoralis major muscle and HADM. A textured anatomic tissue expander was inserted into the space under the muscle. The HADM was sutured to the pectoralis major muscle with appropriate tension using a running 3-0 PDS suture. Tissue expanders were filled with normal saline to an appropriate volume as determined by clinically acceptable tension of the mastectomy skin

Table 2. Surgical Information by Patient

Case No.	1	2	3	4	5	6
Hospital site	Florida Hospital Orlando Outpatient Medical Plaza	Florida Hospital Altamonte	Orlando Health Main Hospital	Winter Park Memorial Hospital	Florida Hospital Orlando Outpatient Medical Plaza	Winter Park Memorial Hospital
Date of surgery	01/01/10	07/29/08 (expander)	12/12/08	06/21/11	09/06/11	05/17/11
Side reconstructed	Bilateral	Bilateral	Bilateral	Bilateral	Right	Right
Expander brand/model	Allergan/ 133FX-13	Inamed/ 133FV-14	Inamed/ 133FV-16	Allergan/ 133 FX 101-T	Allergan/ 133 FX-15-7	Allergan/ 133FX-15-T
Expander volume (mL)	700	600	850	350	650	800
Initial expander fill volume (mL)	200	200	450	125	300	400
ADM brand/lot number	AlloDerm*/ C332566-027, C32572-015	DermaMatrix†/ serial code(s) 065692541001, 065665711006	AlloDerm/ C27835-039 (L), B27227-079	AlloDerm/ B39524-061	AlloDerm/ B40851-024	AlloDerm/ B40558-081, B36813-054
Presoak solution used for ADM	Vancomycin + gentamicin + NS	Bacitracin + NS	Bacitracin + NS	Bacitracin	Bacitracin	Vancomycin + gentamicin + NS
Time between first and second surgeries (wk)	11 (implant removed)	5 (left expander removed)	5 (left expander removed, transverse rectus abdominis myocutaneous first stage)	9.5 (left expander removed in office)	8 (expander removed)	8 (expander removed)

*AlloDerm Regenerative Tissue Matrix, LifeCell, Branchburg, N.J.
†DermaMatrix, Synthes, West Chester, Pa.
NS, normal saline.

flaps. One or two size #10 flat Jackson-Pratt drains were inserted between the HADM and mastectomy flap. Mastectomy incisions were closed with 4-0 Monocryl sutures (Ethicon). The pocket and all expanders were irrigated with antibiotic solutions.

Drains were removed once drainage was less than 20 mL/d. Expansions were begun 1 week postoperatively if the incisions were sealed and appeared to be healing well. Expansions were performed sequentially with normal saline, in the office, over the course of several weeks. The second stage of reconstruction, when performed, consisted of removing the expander and inserting a permanent breast implant. Perioperative antibiotics, antibiotic rinses of implants and pockets, and suture selection were similar to first-stage surgeries. Cultures were obtained from all patients and sent to the reference laboratory (Quest Diagnostics or Florida Hospital pathology laboratory).

ERYTHEMA AND PATIENT COURSE

A total of 28 patients among 3109 reconstructive procedures (0.9%; 95% CI, 0.6%, 1.3%) experienced serious infections requiring removal of the breast implants. Of these, 27 were bacterial and 1 was fungal. The 6 patients reported here represent 6 of 28 or 21.4% (95% CI, 8.3%, 41.0%) of all patients who were diagnosed with surgical-site infections requiring implant removal during a 6-year period.

The 6 of 3109 patients (0.2%; 95% CI, 0.1%, 0.4%) developed localized erythema of the breast initially characteristic of RBS. All 6 patients developed the erythema 4–6 weeks after surgery, and all subsequently had positive cultures for *M. fortuitum*. Statistical evaluation of the estimated incidence rate of *M. fortuitum* infection in a patient thought to have RBS, which occurred 100% of the time in this series, revealed a 95% CI of 54.1–100% by exact two-tailed analysis.

Five of these patients developed the erythema after the first stage of surgery and 1 patient (patient 1) after the second stage. In 1 patient, portions of the same sheet of HADM implant were used in both breasts, but RBS developed in only one (nonirradiated) breast. The other 3 bilateral reconstruction cases involved separate sheet implants of HADM and developed unilateral erythema. Findings included fever (*n* = 1), edema (*n* = 3), and serous wound drainage (*n* = 3). Examination did not reveal evidence of seroma or physical failure on the part of the expander prosthesis or HADM. Complete blood count values were within normal to high-normal range for all patients.

Cultures were obtained in all 6 patients, 3 by fluid aspiration percutaneously in the office and 3 by direct intraoperative cultures from the implant that were obtained in the operating room at the time of removal. All patients had cultures that grew *M. fortuitum* from these aspirations. The elapsed time from the last surgery until presentation of signs of infection was 4 weeks in 3 patients, 5 weeks in 1 patient, 7 weeks in 1 patient, and 8 weeks in 1 patient.

The clinical course of these 6 patients varied. In all 6 cases, patients were treated with antibiotics (Table 3), and the prosthetic implant or tissue expanders were removed, along with all nonincorporated or nonadherent portions of the HADM. In 2 cases, additional debridement of soft tissue (skin, subcutaneous tissue, and pectoral muscle) was required. RBS resolved in all patients within 2 months. At present, successful reconstruction has been completed in 5 of the 6 patients. One patient has chosen not to have additional surgery until she can successfully quit smoking. As examples, Figures 1 and 2 show patients 4 and 5 before mastectomy, at the time of expander infection, and at 2–3 months after reconstruction with breast implants.

All 5 patients who underwent additional stages of reconstruction were offered autologous techniques, such as transverse rectus abdominis myocutaneous flaps or latissimus dorsi flaps. However, only 2 patients were willing to have autologous reconstruction performed. One patient underwent reconstruction with bilateral transverse rectus abdominis myocutaneous flaps and silicone prostheses, and 1 patient underwent 2-stage reconstruction with a latissimus dorsi flap and tissue expander, followed by placement of a permanent breast implant. Three patients underwent 2-stage prosthetic reconstruction with expanders and HADM and successfully went on to have placement of permanent implants without recurrent mycobacterial infection. Of these 3 patients, 2 had excellent results without any sign of encapsulation, and one developed Baker level III encapsulation postradiation. The encapsulation is likely to be related to the radiation and not to the history of previous infection. All patients are still being seen yearly for routine follow-up.

DISCUSSION

The AFB *M. fortuitum* is considered an atypical mycobacterial pathogen. Mycobacteria are ubiquitous, can survive in hostile environments, and can resist commonly used biocides.[14] They are commonly found in soil and in both processed and natural water supplies.[14,15] Strains of mycobacteria, including *M. fortuitum*, have been found to be resistant to several types of disinfectants, including chlorine, povidone-iodine, formaldehyde, and alkaline glutaraldehyde.[14,16] Testing is complicated because *M. fortuitum* and other mycobacteria have relatively long

Table 3. Antibiotic Use

Patient	Laboratory Values (WBC, CRP, ESR, Other)	Antibiotics Used for Treatment of Infection	Other Interventions and Current Status
1*	WBC 7000/µL; CRP 70 mg/L; ESR 60 mm/h	Minocycline + sulfamethoxazole	Left mammary prosthesis removal; successful 2-stage reconstruction with silicone gel implant performed after antibiotic treatment completed; contralateral implant remained in place during course of treatment without infection
2	WBC 4500/µL; CRP not obtained	Amikacin + doxycycline + moxifloxacin	Left mammary prosthesis removal followed by multiple surgical debridements; eventual 2-stage reconstruction performed successfully with silicone gel implant and HADM after antibiotic treatment completed
3	WBC 10,300/µL; CRP not obtained	Amikacin + clarithromycin + imipenem-cilastatin + moxifloxacin + TMP-SMX	Surgical debridement of left mastectomy wound following purulent drainage; left expander removed; eventual successful reconstruction with bilateral transverse rectus abdominis myocutaneous flaps and silicone prostheses
4	WBC 5800/µL; CRP not obtained	Minocycline + moxifloxacin + vancomycin	Left expander removed; after successful antibiotic treatment, 2-stage reconstruction was performed with latissimus dorsi flap and anatomic silicone breast implants; contralateral expander remained in place during entire course of antibiotic treatment without complication or infections
5	WBC 6740/µL, CRP 119 mg/L	Ciprofloxacin + minocycline + moxifloxacin + tobramycin + vancomycin	Multiple percutaneous drainages with worsening symptoms; expander removal; patient completed chemotherapy, followed by placement of expander and HADM; after radiation therapy, a permanent implant was successfully placed
6	WBC 6700/µL, ESR 61 mm/h	Ciprofloxacin + gentamicin + minocycline + TMP-SMX + vancomycin	Incision debridement, expander removal, and successful antibiotic treatment; patient decided not to undergo further reconstruction procedures until completing smoking cessation program

*This patient developed erythema after the second stage of surgery (placement of silicone breast implant).
CRP, C-reactive protein; ESR, erythrocyte sedimentation rate; HADM, human-derived acellular dermal matrix TMP-SMX, trimethoprim-sulfamethoxazole; WBC, white blood cell count.

Fig. 1. Patient 4. A, Presentation before bilateral mastectomy; B, expander infection; and C, 2 mo after reconstruction with silicone implants.

Fig. 2. Patient 5. A, Presentation before mastectomy of right breast; B, expander infection; and C, 3 mo after reconstruction with saline implant.

incubation periods compared with bacterial species more commonly associated with postoperative infections. Mycobacteria frequently go undetected when using standard tests for infectious agents (eg, Gram stain, commonly used aerobic and anaerobic culture methods). The best tests to detect mycobacterial infection feature staining solutions (eg, Ziehl-Neelsen stain) or Lowenstein-Jensen medium.[17]

Mycobacterial infections with a delayed onset have been reported after breast augmentation procedures[18–25] and other types of reconstructive and cosmetic plastic surgeries, including periocular and facial surgery[16,26] and cataract extraction with intraocular lens implantation.[27]

Six of 3109 patients (0.2%; 95% CI, 0.1%, 0.4%) who underwent 2-stage breast reconstruction performed over a 6-year period developed erythema thought to be RBS and were eventually diagnosed with *M. fortuitum* infection. Statistical evaluation of the estimated incidence rate of *M. fortuitum* infection in a patient thought to have RBS was 6 of 6 or 100% in this series, with a two-tailed 95% CI of 54.1–100%. Although this makes such an infection extremely rare in postmastectomy breast reconstruction, these 6 of 28 cases also represented 21% (95% CI, 8.3%, 41.0%) of all complications due to

infection ($n = 28$) occurring in these surgeries that led to breast implant removal. The etiology of *M. fortuitum* in this case series is unknown. However, it may have been introduced during postoperative exposure of the surgical wound and/or drain sites to microorganisms (eg, from the shower, environmental surfaces, pet exposure, or an inadvertent lapse of sterile technique during dressing changes at home). Postoperative infections may also occur as a result of intraoperative contamination or immunosuppression. Three patients presented with acute onset of pain and erythema initially localized at the drain site and subsequently progressing to the breast, leading to the hypothesis that ascending drain site infections may have been the etiology, at least in some of these cases.

By definition, the cases of *M. fortuitum* infection included in this report do not match the general description of RBS, as the limited literature addressing RBS describes it as a diagnosis of exclusion. RBS is an unusual and infrequent complication of breast reconstructive surgery. It usually appears a few weeks to a month after surgery, visibly resembles cellulitis (but without warmth, tenderness, or fever), causes concern among surgeons and patients, and then slowly resolves. Discharge, fever, and laboratory ab-

normalities are generally not present. Some authors have suggested that RBS is a reaction caused by some factor related to the ADM.[7,8,13] In an exchange of letters, Newman et al[7] and Nahabedian[8] discussed some possible etiologies, including dependent erythema in the lower breast, interruption of lymphatic flow, an unknown factor in ADM, a generalized histamine release, an inflammatory response to the stress on tissues of creating the pocket for an expander, the pressure of the expansion, or the initiation of revascularization. Confounding factors in determining the etiology of RBS include the effects of chemotherapy and radiation that patients received before or during reconstruction. Most discussions of RBS conclude that it is unlikely to be caused by an infection.[6,12,13]

We believe it is unlikely that the mycobacterial infections observed in our series were all related to a lapse in sterile technique by a specific surgeon or surgical site, as the overall rate of infection was very low and the surgeries were performed at multiple hospital sites in conjunction with several different breast cancer surgeons. We also believe HADM to be an unlikely source of the infections, as there have been no previous case reports involving HADM as a source of mycobacterial infection following breast augmentation in the published literature. The infections observed in these 6 patients were probably due to postsurgical exposure of drain sites to environmental sources of mycobacteria. All 6 patients had surgical drains placed during their procedure; 3 of the 6 had overt signs of infection at the drain site during follow-up. Mycobacteria are frequently associated with catheter-related infections,[14] and the surgical drains may have served as a conduit for bacterial entry following mycobacterial exposure in our patients' households. Showerheads provide a moist, dark, warm environment which can enrich growth of microbes, such as mycobacteria, that are present in the water supply. A recent study of microbial swabs of 45 showerhead surfaces from diverse geographical areas in the United States showed mycobacterium to be the most highly prevalent of all bacterial species isolated in the study,[28] and mycobacterial contamination of patient catheters via shower/water exposure has been reported in several studies.[29] Domestic pets, including cats and dogs, are another potential source of mycobacterium,[30,31] and it is possible that wound and/or drain exposure to pet hair or pet saliva could be the source of these mycobacterial infections.

Several methods have been suggested to prevent or reduce the incidence of RBS. Some authors[12,13] have reported variations in the way they prepare ADM before use in breast reconstruction. Newman[12] soaks nonhydrated ADM in a triple antibiotic solu-tion but uses prehydrated ADM straight from the packaging and rinses it in the pocket with either normal saline or triple antibiotic solution. Rawlani et al[13] suggest that placing the ADM in the pocket with the smooth (basement membrane) surface facing the expander or implant may reduce the incidence of RBS.

The 6 cases included in this report deviate from the previous descriptions of RBS in several respects. All patients had culture-positive infections, which would preclude any entity defined as a "diagnosis of exclusion." Clinical presentations were also more severe, including cases of fever ($n = 1$), localized edema ($n = 3$), and high-volume serous wound drainage ($n = 3$). Rather than being self-limited, these infections led to prosthesis explantation in all patients.

We believe that undiagnosed AFB infection may explain some of the cases thought to be RBS (ie, some cases diagnosed as RBS may be caused by low-grade and self-limited infections by *M. fortuitum* or milder strains of mycobacteria). The diagnosis of AFB may be missed in these cases as it is difficult to establish, partly due to the fluid sample acquisition challenges in such patients. In addition, surgeons not considering AFB infection may not request appropriate cultures. Moreover, AFB cultures must be kept refrigerated during transport to the laboratory. Laboratory personnel must be alerted when they have an AFB specimen for transport and processing.

The possibility of infection by *M. fortuitum* or other AFB presents implications for the management of patients when a diagnosis of RBS is being considered. Ruling out seroma, mechanical failure of the HADM, and life-threatening disease such as inflammatory carcinoma remain important. Patients with postoperative erythema should be treated initially with conventional regimens providing coverage of common Gram-positive organisms. When erythema persists in a case of suspected RBS, there should be a low threshold for initiating AFB coverage. As many *Staphylococcus* species are methicillin resistant, such organisms should also be considered. Antibiotics such as minocycline, ciprofloxacin, moxifloxacin, and trimethoprim/sulfamethoxazole may be good choices for initial oral therapy in such cases. The patient should be carefully monitored for spreading erythema, fever, and tenderness in the area. Fluid should be aspirated for culture, which should include AFB testing.

When erythema persists in a case of suspected RBS, antibiotics should be stopped for a day or so if possible, after which cultures of deep infected tissue should be obtained and sent for aerobic, anaerobic, fungal, and mycobacterial stains and cultures. If mycobacteria are isolated, susceptibility studies

should be obtained from a reference laboratory. Initial empiric treatment should be directed against methicillin-resistant *Staphylococci* with vancomycin, daptomycin, or linezolid intravenously or doxycycline or trimethoprim/sulfamethoxazole if the oral route is chosen. If mycobacteria are identified, initial treatment would be empiric and might include cefoxitin, amikacin, clarithromycin, trimethoprim/sulfamethoxazole, or quinolones, and definitive treatment should be based on the results of susceptibility studies. An infectious disease consultation is usually indicated for complex mycobacterial infections. Generally speaking, 4–6 months of treatment or longer may be required.

Based on our anecdotal observations and our suspicions regarding potential bacterial exposure from patients' home environments as a potential source of mycobacterial infection, we now identify pet ownership status during preoperative consultations in our practice and we counsel patients regarding strategies for avoidance of pet-mediated infection during recovery. Patients are counseled to sterilize or replace showerheads in their homes, and we recommend they avoid allowing tap water to come into contact with their surgical wound and drain site during the postsurgical recovery period. In the early postoperative period, patients should maintain sterile techniques as much as possible in caring for their wounds. It is best to avoid the bathroom in performing wound care, as multiple wet areas promote the growth of potentially pathogenic bacteria. We have also incorporated Biopatch antimicrobial dressings (Ethicon) with chlorhexidine gluconate (Ethicon) to cover drain sites in our patients undergoing implant-based breast reconstruction. Chlorhexidine displays bacteriostatic activity against mycobacteria in vitro,[32] and reported data indicate that bacteriostatic activity may be sufficient to treat several types of infections in a clinical setting.[33] Use of these dressings has been shown to reduce the rate of postoperative infections associated with periprosthetic drains following breast reconstruction procedures.[34] In our experience over the past 6 months since incorporating Biopatch antimicrobial dressings into our postoperative care regimen, we have observed no cases of *M. fortuitum* infection or any other cases that may be diagnosed as RBS. Further studies are warranted to assess the effectiveness of this approach.

It seems likely that multiple etiologies may explain RBS as currently described in the literature. Over time, the ability to better diagnose AFB may lead to a subset of patients considered to have RBS, such as those reported here, being excluded from the category of RBS due to documented infection. It remains important to consider such AFB infections when assessing a patient with possible RBS.

CONCLUSIONS

Six cases of *M. fortuitum* infection in 3109 2-stage prosthetic breast reconstructions with HADM were reported. These cases raise the possibility that atypical acid-fast mycobacteria infections may be present in patients otherwise thought to have RBS and thus have implications for the diagnosis and management of this entity.

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