An Outbreak of *Mycobacterium jacuzzii* Infection following Insertion of Breast Implants

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**Background.** Surgical wound infections caused by rapidly growing mycobacteria developed in 15 women after insertion of breast implants from August to November 2003 at a single medical center.

**Methods.** A case-control study was conducted that included the identified patients, as well as women who underwent breast operations at the same center who did not develop infections. The study was accompanied by an extensive environmental investigation. Isolates were identified by standard bacteriological methods and by comparison of their 16S rRNA, HSP65, RPOB, SODA, and RECA gene sequences. Isolates were compared by random amplified polymorphic DNA analysis and by pulsed-field gel electrophoresis.

**Results.** The risk factors for infection included surgery performed by 1 specific surgeon (odds ratio, 21.3; 95% confidence interval, 3.64–125.6). Identical strains of mycobacteria were isolated from the infected wounds of the patients; from the eyebrows, hair, face, nose, ears, and groin of this particular surgeon; and from this surgeon’s outdoor whirlpool. The isolates exhibited a biochemical profile overlapping that of *Mycobacterium wolinskyi*, but their sequences of 16S rRNA and HSP65, RPOB, SODA, and RECA genes differed. We propose the name “*Mycobacterium jacuzzii*” for this new species. DNA fingerprints of cultured isolates from the surgical wounds, areas of the surgeon’s body that grow hair, and the surgeon’s whirlpool were identical. When the surgeon discontinued his use of the whirlpool and began cleaning the hairy areas of his body with a shampoo containing triclosan, the outbreak ended.

**Conclusions.** This outbreak brings to light the possibility of the colonization of human skin and human-to-human transmission of environmental mycobacteria during surgery that involves implant insertion.

More than 2 million women in the United States have breast implants, and a 5-fold increase in the performance of breast augmentation surgery was reported from 1992 to 2000 [1, 2]. The rates of infection following augmentation mammoplasty (AM) and following breast reconstruction after mastectomy range from 0.5% to 2.5% and 1% to 24%, respectively [2–4]. *Staphylococcus aureus* and coagulase-negative staphylococci are the most common organisms involved; however, *Pseudomonas aeruginosa* and nontuberculous mycobacteria (NTM) have also been implicated [2, 3].

**Background.** From August to November 2003, a cluster of surgical wound infections occurred in 10 women following surgery for insertion of breast implants performed in a single medical center. Specimens obtained from the infected surgical sites were initially analyzed by a private laboratory and were found to be sterile.

The modern, well-equipped, certified outpatient medical center has 2 operating suites located on the uppermost floor. The building’s water supply is connected to the municipal water system. The air conditioning of the medical center is an autonomous closed system, but an open air conditioning system servicing the rest of the building is located on the roof. Thirty surgeons, including 15 who specialize in plastic surgery, and 15 nurses work in the center and perform 250–300 clean, elective operations each month. One hundred operations involve the breast; of these operations, 90% are AM, 5% are breast reconstruction after mastectomy, and 5% are surgical correction of breast asymmetry and other anomalies. AM involves the insertion of silicon gel implants, whereas implants used for breast...
reconstruction and asymmetry correction are shaped and adjustable. Cefonicid was routinely administered on the day of surgery and 1–7 days later.

At the time of investigation, 7 (70%) of 10 infected women had persistent discharge from the surgical wound site, from which we succeeded in isolating rapidly growing mycobacteria (RGM). RGM were also isolated from the tap water of the operating rooms and from water in the open air conditioning system.

Closer examination of the routine infection control measures found that surgeon A systematically requested that the air conditioning in the operating room be temporarily turned off (from the time the patients entered the room until they were fully anesthetized, to reduce their exposure to the low temperature). This transiently interrupted the operating room’s positive pressure. Initially, we hypothesized that the combination of local environmental contamination with RGM and the disrupted positive pressure facilitated entry of mycobacteria into the surgical site wounds. The medical center’s water supply was then separated from that of the rest of the building, and all water sources were hyperchlorinated. The air conditioning systems were thoroughly cleaned, and all high-efficiency particulate air filters were changed. Despite these measures, 5 additional cases of surgical wound infection occurred during the subsequent month, prompting further investigation.

METHODS

Definition and ascertainment of cases. Definite cases involved patients who had breast surgery involving implants in the medical center from 1 July to 30 November 2003 and developed a surgical site infection (SSI) that was culture-positive for RGM. Presumptive cases involved patients who fulfilled the same criteria, except the infection was not assessed by mycobacterial stains or cultures.

Epidemiologic investigation. The extent of the outbreak was established from a review of the medical records of all patients who had undergone breast surgery involving implants from July to November 2003. All patients were notified and asked to report any symptoms of SSI. Patient care was reviewed by interviewing operating room personnel and by observing surgical procedures involving breast implants performed at the medical center.

In all, 202 women who underwent breast operations involving implants during the study period were enrolled in the case-control study. Control subjects were the patients who did not develop SSI.

Risk analysis included age, type and duration of surgery, type of implant, the operating room used, and the number and identities of the involved personnel. Statistical analysis was performed using SPSS software, version 10 (SPSS). Continuous variables were compared using Student’s t test. ORs and their 95% CIs were calculated for categorical variables and were compared using Fisher’s exact test. Variables with a P value <.05 were entered into a logistic regression model, where statistical significance was set at P < .05.

Environmental investigation. Samples of tap water from all sinks, water from air conditioning systems, and sterile saline used for expansion of implants, vials of lidocaine and epinephrine, and surgical dressings were collected. Swabbed samples of surfaces of surgical instruments, implants, skin markers, and air conditioning filters were also collected. Air sampling was performed with an air sampler. Finger, nasal, hair, and axillary swab samples were obtained from the operating room staff. Samples were also obtained from surgeon A’s family members, dogs, and objects in his house.

Laboratory methods. Patient specimens, obtained by swabs, needle aspirates, and tissue biopsies, as well as all environmental specimens, were forwarded to the microbiological laboratory. Specimens were processed by standard methods and inoculated onto a mycobacterium growth indicator tube (Bactec MGIT 960; Becton-Dickinson) or MB/BacT bottle (BacT Alert System; Organon Teknika), a Löwenstein-Jensen slant, and a Middlebrook 7H11 selective agar plate [5], and were incubated at 36°C either until growth was observed or up to 7 weeks. Water samples were concentrated, decontaminated, and inoculated onto Löwenstein-Jensen slants and MB/BacT bottles [6]. Smears from colonies were stained with Ziehl-Neelsen stain. Species identification was performed by conventional biochemical methods [6–8] and by determining antimicrobial susceptibility patterns using the resistance ratio method and Etest [6, 7, 9]. Species were further identified by PCR amplification and by sequencing of the 16S rRNA and the HSP65, RPOB, SODA,
Table 2. Clinical and demographic characteristics of case patients.

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age, years</th>
<th>Date of surgery</th>
<th>Type of operation</th>
<th>Breast involved</th>
<th>Incubation period, days&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Treatment and outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1&lt;sup&gt;b&lt;/sup&gt;</td>
<td>18</td>
<td>1 Jul 2003</td>
<td>AC</td>
<td>Right</td>
<td>30</td>
<td>Asymptomatic after removal of implants</td>
</tr>
<tr>
<td>2</td>
<td>42</td>
<td>1 Jul 2003</td>
<td>AM</td>
<td>Left</td>
<td>35</td>
<td>Recurrent infection and debridment; Cpfx and Dox therapy for 6 weeks</td>
</tr>
<tr>
<td>3&lt;sup&gt;b&lt;/sup&gt;</td>
<td>70</td>
<td>2 Jul 2003</td>
<td>BRM</td>
<td>Left</td>
<td>39</td>
<td>Asymptomatic after removal of implants</td>
</tr>
<tr>
<td>4</td>
<td>18</td>
<td>29 Jul 2003</td>
<td>AM</td>
<td>Bilateral</td>
<td>32</td>
<td>Asymptomatic after removal of implants; Cpfx therapy for 3 weeks</td>
</tr>
<tr>
<td>5</td>
<td>29</td>
<td>30 Jul 2003</td>
<td>AC</td>
<td>Bilateral</td>
<td>34</td>
<td>Recurrent infection and debridment; Cpfx and Dox therapy for 6 weeks</td>
</tr>
<tr>
<td>6&lt;sup&gt;b&lt;/sup&gt;</td>
<td>44</td>
<td>10 Aug 03</td>
<td>BRM</td>
<td>Left</td>
<td>16</td>
<td>Asymptomatic after removal of implants</td>
</tr>
<tr>
<td>7</td>
<td>20</td>
<td>2 Sep 2003</td>
<td>AM</td>
<td>Left</td>
<td>27</td>
<td>Recurrent infection and debridment; Cpfx and Dox therapy for 6 weeks</td>
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<tr>
<td>8</td>
<td>18</td>
<td>10 Sep 2003</td>
<td>AM</td>
<td>Left</td>
<td>32</td>
<td>Asymptomatic after removal of implants; Cpfx therapy for 6 weeks</td>
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<tr>
<td>9</td>
<td>40</td>
<td>10 Sep 2003</td>
<td>BRM</td>
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<tr>
<td>10&lt;sup&gt;c&lt;/sup&gt;</td>
<td>22</td>
<td>10 Sep 2003</td>
<td>AM</td>
<td>Left</td>
<td>52</td>
<td>Asymptomatic after removal of implants; Cpfx therapy for 3 weeks</td>
</tr>
<tr>
<td>11&lt;sup&gt;d&lt;/sup&gt;</td>
<td>24</td>
<td>14 Sep 2003</td>
<td>AM</td>
<td>Left</td>
<td>19</td>
<td>Recurrent infection and debridment; Cpfx and Dox therapy for 12 weeks</td>
</tr>
<tr>
<td>12&lt;sup&gt;c&lt;/sup&gt;</td>
<td>36</td>
<td>14 Sep 2003</td>
<td>AM</td>
<td>Right</td>
<td>37</td>
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**NOTE.** AC, asymmetry correction; AM, augmentation mammoplasty; BRM, breast reconstruction after mastectomy; Cpfx, ciprofloxacin; Dox, doxycycline.

* Defined as the interval between the operation and onset of infection.
* Patients with presumptive cases, in which mycobacteria were not detected by stain or culture.
* Patients whose operations were not performed by surgeon A. Patient 10’s operation was performed by surgeon B, and patient 12’s operation was performed by surgeon C.
* An adjustable implant manufactured by Inamed, style 410, was inserted.
* Patient underwent breast surgery without implant insertion.

and *RECA* genes. In brief, DNA was extracted from colonies grown on Mueller-Hinton broth medium using the Wizard genomic DNA purification kit (Promega), in accordance with the manufacturer’s instructions. Five molecular targets, including a 1338–base pair (bp) sequence of 16S rRNA [10], a 387-bp sequence of *HSP65* [11, 12], a 524-bp sequence of *SODA* [13], a 951-bp sequence of *RECA* [13], and a 738-bp sequence of *RPOB* [13, 14], were amplified and sequenced bidirectionally using sequencing primers and Big Dye Terminator v1.1 on an ABI Prism 3100 automated sequencer (Applied Biosystems). The similarity among sequences was determined using the Clustal W program with the Accelrys Gene 2.0 program (Accelrys). Randomly amplified polymorphic DNA analysis was applied for genotypic comparison of isolates. Ten random primers, described in previous studies of mycobacteria, were chosen for evaluation [15, 16], and the 3 that generated the best amplification patterns were used thereafter for strain comparison (table 1). PFGE was performed with restriction enzyme *Xba*I, as described elsewhere [17].

**RESULTS**

**Description of case patients.** Fifteen women with a median age of 31.7 years (range, 18–70 years) developed SSI. Eleven women (73%) had definite cases, and 4 (27%) had presumptive cases. Three of the presumptive cases occurred early in the outbreak, when mycobacteria were not suspected, and consequently, neither acid-fast smears nor mycobacterial cultures were performed.

No clustering of cases by day of the week or time of surgery was found, but all patients except 1 underwent breast operations involving implants (table 2). All adjustable implants were
The mean interval between surgery and the onset of infection was 28 days (range, 16–39 days). All patients had symptoms and signs localized at the surgical site, including clear discharge, tenderness, occasional pain, swelling, and erythema, but none experienced fever or other systemic symptoms. Early during the outbreak, patients were treated with surgical exploration, lavage, drainage, and broad-spectrum antibiotics. Eventually, all implants had to be removed because of the ongoing infection. Three presumptive case patients recovered completely after implant removal without any specific antibiotic therapy. Six patients remained asymptomatic after removal of implants but received 3–6 weeks of ciprofloxacin, and 6 patients experienced recurrent wound infection that was treated by surgical debridement, ciprofloxacin, and doxycycline for 6–12 weeks.

**Case-control study.** The attack rate was 6.9%, with 14 case patients and 188 control subjects. Patient 13 was excluded from the case-control study, because no implant was inserted during breast reconstruction. The attack rate for patients whose operations were performed by surgeon A was 28.6% (12 of 42 operations) performed by other surgeons b 12 (85.7):2 30 (15.96):158 31.25 (6.71–142.8) <.001 21.3 (3.64–125.6) <.001

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**NOTE.** Data are no. of patients, unless otherwise indicated. Patient 13 was excluded from the case-control study, because no implant was inserted during breast reconstruction. AC, asymmetry correction; AM, augmentation mammoplasty; BRM, breast reconstruction following mastectomy.

a Adjusted for surgeon, type of operation, and implant type.

b The infection rate was not increased when other operating room staff was involved in surgeries.

style 150, manufactured by Inamed; 6 silicon gel implants were manufactured by Sebbin, and 1 was manufactured by Inamed. Surgeon A performed 13 of the operations, and surgeons B and C performed 1 operation each (surgeon C performed the operation for patient 12, from whom mycobacteria were not isolated).

The mean interval between surgery and the onset of infection was 28 days (range, 16–39 days). All patients had symptoms and signs localized at the surgical site, including clear discharge, tenderness, occasional pain, swelling, and erythema, but none experienced fever or other systemic symptoms. Early during the outbreak, patients were treated with surgical exploration, lavage, drainage, and broad-spectrum antibiotics. Eventually, all implants had to be removed because of the ongoing infection. Three presumptive case patients recovered completely after implant removal without any specific antibiotic therapy. Six patients remained asymptomatic after removal of implants but received 3–6 weeks of ciprofloxacin, and 6 patients experienced recurrent wound infection that was treated by surgical debridement, ciprofloxacin, and doxycycline for 6–12 weeks.

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more significant than the association between AM and infection (P <.001 vs. P <.005). Adjustable implants were also associated with increased rate of infection (P <.001). In multivariate analysis, the only statistically significant risk factor was performance of the procedure by surgeon A. No such increased risk was found for operations not requiring implants performed by surgeon A.

**Microbiological study.** Specimens of surgical wound discharge yielded small colonies of acid-fast bacilli 3 days after inoculation on chocolate agar. These isolates, as well those grown on selective mycobacterial media, were identified as belonging to the Mycobacterium smegmatis group on the basis of the typical growth of smooth nonpigmented colonies, growth in <7 days, negative results for arylsulfatase at 3 days, growth at 45°C, presence of nitrate reductase, and susceptibility to ethambutol. On the basis of tobramycin resistance (MIC, 32 μg/mL), the isolate was identified as Mycobacterium wolinskyi. Isolates were susceptible to ciprofloxacin, ofloxacin, doxycycline, amikacin, and ethambutol and resistant to clarithromycin, sulfamethoxazole-trimethoprim, rifampicin, isoniazid, capreomycin, cycloserine, tobramycin, streptomycin, cefoxitin, and imipenem.

**Environmental studies.** Various species of NTM, such as Mycobacterium kansasii, Mycobacterium fortuitum, and Mycobacterium gordonae, were isolated from tap water and the cooling tower water. Air samples obtained in the operating room were sterile. The only specimens that grew the mycobacteria that caused the outbreak were those obtained from the regions of surgeon A’s body that grow hair—namely, his eyebrows, scalp, face, nose, ears, and groin. The surgeon was healthy and did not have skin rashes or paronychia, and his chest radiograph was normal. Cultures of samples obtained from surgeons B and C and from other surgical personnel did not grow the mycobacteria. The outbreak-causing mycobacteria were subsequently also isolated from surgeon A’s bed linen, pillows, towels, bath-
Mycobacteria and Breast Implants

Figure 1. Products of randomly amplified polymorphic DNA–PCR of 15 Mycobacterium jacuzzii isolates. A primer (5′-TGGTCGCGGC-3′) was used in the amplification. Isolates were obtained from 11 patients (indicated by the letter “P” and the corresponding patient number), from the hair on surgeon A’s head (HH), from surgeon A’s eyebrows (EB1 and EB2), and from the whirlpool (JS). Control isolates include Mycobacterium smegmatis ATCC 607, M. smegmatis MC²-6, and a Mycobacterium fortuitum clinical isolate. The dendrogram was constructed by computer analysis with the Phoretix1D software package (Nonlinear, Dynamics) using the weighted pair group method of arithmetic averages.

Molecular comparison and identification of isolates. All 15 outbreak isolates (11 from case patients, 3 from the head hair and the eyebrows of surgeon A, and 1 from the whirlpool) were morphologically and biochemically identical. All had the same antimicrobial susceptibility pattern. randomly amplified polymorphic DNA–PCR and PFGE demonstrated 2 different strains (figure 1), but they were indistinguishable by their HSP65, RPOB (figure 2), 16S rRNA, SODA, and RECA sequences (data not shown).

Genotypic comparison demonstrated that the outbreak mycobacteria were most related to *M. wolinskyi*. Comparison of the HSP65, RPOB, 16S rRNA, SODA, and RECA sequences of the outbreak mycobacteria (accession numbers DQ137415, DQ137416, DQ137412, DQ137414, and DQ137413) using the Basic Local Alignment Search Tool program for known sequences in the GenBank database demonstrated 98% identity (381 of 387 bp) in the HSP65 sequence with *M. wolinskyi* strains CIP 106348 and ATCC 700010 (accession numbers AF547890 and AF548064, respectively). The 16S rRNA sequence differed by 1 bp from that of *M. wolinskyi* strain ATCC 700010 (accession number AF547083). The RPOB sequence was most identical (94%; 698 of 738 bp) to that of *Mycobacterium farcinogenes* strain 3753 (accession number AF547083) and was 91% identical to RPOB gene of *M. wolinskyi* strain ATCC 700010 (accession number AF547083) (figure 2). The SODA sequence had 99% identity to that of *M. wolinskyi* strain ATCC 700010 (499 of 501 bp) and CIP 106348 (439 of 442 bp); (accession numbers AF547890 and AF548064, respectively). RECA was 97.5% identical (928 of 951 bp) to that of *M. wolinskyi* strain ATCC 700010 (accession number AF547083).

Outbreak control measures. Surgeon A cleaned himself daily with a body scrub and shampoo containing triclosan (Dermax; Fischer), and he stopped using the whirlpool bath. During surgery, he wore a hood-style cap that covered all exposed facial areas. Weekly cultures of samples from the hair-growing areas of his body were negative for 2 years of follow-up. With these

robe, and car air conditioning system, as well as from the water of his outdoor home whirlpool. Another whirlpool user in his family had colonization with the same mycobacteria.

**Molecular comparison and identification of isolates.** All 15 outbreak isolates (11 from case patients, 3 from the head hair and the eyebrows of surgeon A, and 1 from the whirlpool) were morphologically and biochemically identical. All had the same antimicrobial susceptibility pattern. randomly amplified polymorphic DNA–PCR and PFGE demonstrated 2 different strains (figure 1), but they were indistinguishable by their HSP65, RPOB (figure 2), 16S rRNA, SODA, and RECA sequences (data not shown).

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interventions, the colonized mycobacteria were eradicated, and the outbreak was terminated.

DISCUSSION

Our investigation identified an outbreak of *M. jacuzzii*-associated SSI following the insertion of breast implants. The same mycobacterium was recovered from the hair-growing areas of a surgeon who was asymptomatic; he had acquired the mycobacterium from water in his outdoor whirlpool. Presumably, scales colonized with *M. jacuzzii* were shed from the surgeon during surgery, even though he wore a standard paper cap. The Israeli guidelines for surgical attire include gown, mask, and hair coverings extending over the sideburns and neckline. Surgical personnel with beards must use full masks and head attire. SSI outbreaks have occasionally been traced to organisms isolated from the hair or scalp of surgical personnel, even when caps were worn [18, 19]. Surgeon A had no beard; thus, dandruff shed from his eyebrows was probably the source of the contamination.

The presence of a foreign body, such as a breast implant, appears critical for development of this infection, because infection did not occur in patients who had other operations performed by the same surgeon. Perhaps surgeon A used the whirlpool before operations in which the patients developed infection. Although surgeon A had no contact with patient 10, whose surgery was performed by surgeon B in a different suite, cross-contamination between the surgeons could have occurred in the scrub-in area.

Asymptomatic health care workers who are colonized with pathogens may be involved in the transmission of microorganisms, such as methicillin-resistant *S. aureus* [20], group A streptococci [19, 21], and *Rhodococcus bronchiialis* [22], resulting in hospital outbreaks. The noses, vaginas, rectums, hands, and scalps of health care workers were the source of the col-
onizing bacteria in these outbreaks, and unnoticed skin lesions, such as eczema, psoriasis, and seborrhea, were found on their skin. In most instances, the health care worker who was colonized with a microorganism had direct patient-care responsibilities in the operating theater, but airborne dissemination of bacteria in the operating room via aerosolized droplets from health care workers has been reported [21, 22].

NTM exist widely in soil and water and can colonize and cause disease in many animal species [23]; NTM can also colonize and cause disease in the respiratory tract of persons with chronic lung disease. However, colonization of skin by environmental mycobacteria and intrasurgical transmission have never been described.

Outbreaks of illness caused by NTM—especially RGM—have become relatively common [24–31]. In most outbreaks, the source of the organism has not been identified, but some outbreaks have been attributed to contamination of solutions used for skin marking [24], the ice machine in the operating room [25], and recently, whirlpool foot baths at nail salons [31]. The frequency of wound infection—especially by M. chelonae, M. fortuitum, and M. abscessus—following cosmetic surgery procedures (mainly AM) has increased [24, 27, 29, 30, 32–36]. Clinicians should have a high index of suspicion of mycobacterial infection in a patient with delayed symptoms of wound infection in the absence of systemic signs following insertion of breast implants. Specimens should be obtained, and the laboratory should be informed about the suspected organism, because the small gray colonies of mycobacteria can be mistaken for nonpathogenic coagulase-negative staphylococci or diphtheroids.

Risk factors for infection associated with breast implants have not been carefully assessed in the literature. In the current outbreak, increased risk of infection occurred among women undergoing surgery for reconstruction and asymmetry correction of the breast or receiving adjustable implants. However, these patients were also directly associated with surgeon A, who performed these operations.

For all case patients, the implant had to be removed, as has been suggested elsewhere [32, 36]. Antimicrobial therapy should be guided by in vitro susceptibility testing, and its duration—from 6 weeks to many months—should be guided by the patient’s clinical response. A range of 1–4 antimicrobial agents can be administered parenterally or orally, depending on the severity of infection [25, 26, 28, 29, 32, 33, 35, 36]. The antibiotic susceptibility pattern of M. jacuzzii differed from that of other common RGM, because it was resistant to clarithromycin, cefoxitin, and imipenem. Resistance to sulfamethoxazole-trimethoprim refuted the initial identification of the strain as M. smegmatis or M. wolinski. In our experience, the most effective treatment was removal of the implants, total capsulectomy whenever possible, and excision of all granulated tissue.

Conventional laboratory methods, including cell-wall fatty acid and mycolic acid composition analysis, cannot discriminate among emerging RGM [37, 38]. During the past 10 years, 16S rRNA gene sequencing has contributed to the establishment of >45 novel NTM species. Cumulative experience has indicated that RPOB gene sequencing improves the recognition of emerging RGM and that a >3% sequence divergence delineates a novel species [37, 38]. We found significant gene sequence differences for 5 different genes, conforming to the proposition of the ad hoc committee for reevaluation of the species definition that novel species should be described according to differences in the sequences of 5 housekeeping genes, instead of DNA–DNA hybridization [39]. Antibiotic susceptibility patterns and 5 gene-sequence differences are sufficient to define this outbreak Microbacterium strain as a novel species. Although randomly amplified polymorphic DNA–PCR and PFGE demonstrated 2 different strains, their 16S rRNA, HSP65, RPOB, SODA, and RECA genes were 100% similar, confirming that both belonged to the same species.

Municipal water supplies are recognized as major reservoirs for mycobacteria and as the source of most nosocomial outbreaks [23]. Supported by biofilms in pipes, these mycobacteria can grow at high temperatures and in distilled water, and they can resist common disinfectants and the low chlorine concentrations of tap water.

In summary, an outbreak of SSI due to M. jacuzzii following breast implantation surgery was traced to mycobacteria shed during surgery by an asymptomatic surgeon who had acquired M. jacuzzii from his whirlpool. Our article raises the possibility that human-to-human transmission of environmental mycobacteria may occur. Meanwhile, we recommend delineating guidelines for appropriate cleaning and disinfection of private whirlpool baths, specifically those owned by surgical personnel or by patients planning to undergo surgery.

Acknowledgments

Potential conflicts of interest. All authors: no conflicts.

References