Letter to the Editor

Plastic Expander-Related *Gordonia Sputi* Infection: Case Report and Literature Review



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Gordonia sputi causes rare bacterial infections resulting from a contaminated indwelling medical device. We report the case of a postoperative plastic expander abscess in a woman, with *G. sputi* identification by 16S ribosomal RNA sequencing. This report indicates that *Gordonia spp.* should be included in the list of organisms causing plastic implant infections.

Gordonia sputi is a ubiquitous, gram-positive coryneform bacterium found in environmental soil and water^[1]. G. sputi is usually not considered a common pathogen of human diseases. However, reports of G. sputi as a causative agent of human opportunistic infections have been increasing in recent years^[2-4]. Although reports on G. sputi infection in clinical patients are scarce, severe diseases, immunodeficiency, underlying and bacteremia heighten the risk factors and clinical symptoms. Here we report a localized abscess caused by G. sputi in a patient who received a breast expander implant in the plastic surgery department without any detectable classic risk factors and clinical presentation. The emerging different epidemiology and clinical characteristics may be helpful in furthering an understanding of rare pathogen infections in healthcare.

A 31-year-old woman was admitted to Peking Union Medical College Hospital on March 6, 2012 for successive expander removal and augmentation mammoplasty operations using a dual-breast silicone prosthesis implantation. Eight months earlier, the patient had been diagnosed with a ductal carcinoma of the left breast without lymph node metastasis and underwent a simple mastectomy and phase I breast reconstruction with a latissimus dorsi musculocutaneous flap. After the operations, a breast expander implanted in the was

retropectoralis major space. The patient recovered well and was discharged, with advice for regular follow-up.

Routine physical examination before the phase II augmentation mammoplasty operation was normal except for a localized swelling with itching in the lower outer quadrant of her left breast. The patient's temperature was normal (36.8 °C) and she had no chest pain or indisposition. The swelling, measuring approximately 3 cm × 2 cm, was red, slightly tender, without any detectable increase in temperature, and exhibited diabrosis, fluctuation, and secretion from the nipple. An electrocardiogram and routine laboratory examinations were within the normal limits. Blood, urine, liver and renal function, and coagulation function tests, conducted to determine the possibility of a systemic infection, showed no signs of a generalized infection. Therefore, the patient was suspected to have a localized postoperative infection and was scheduled to undergo expander removal followed by surgical debridement. During expander removal the operation, a copious amount of pus as well as some inflamed granulation tissue was found surrounding the expander. A culture sample of the pus was collected and sent to the laboratory for further testing. After flushing with copious amounts of saline, oxydol, and dilute iodine, tissue debridement was performed, and the wound was sutured with two surgical drains in situ. At the time of the infection, the patient was not receiving any chemotherapy (or radiotherapy).

The pus from the abscess was first identified to contain oxacillin-resistant *Staphylococcus epidermidis*. However, when the sample culture was rechecked, there were dry, raised, white-to-orange colonies observed in brain-heart infusion agar plates

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after more than 48 h of incubation at 37 °C. The bacterium was identified as a gram-positive bacillus and was tentatively characterized as an Actinomyces spp. on the basis of colony morphology and conventional biochemical reactions (bioMérieux, Marcy-l'Etoile, France). Thereafter, 16S ribosomal RNA (rRNA) gene sequencing was conducted using a pair of universal primers to confirm the results. The obtained product sequences (1403 bp) were compared with published sequences in the GenBank database (http://www.ncbi.nlm.nih.gov/blast). The results showed evidence of G. sputi infection (with GenBank DSM 43896 X80634, ATCC 29627, 99% similarity). The sequence has attained a GenBank accession number, seq.sgn seg KT122843. The antibiotic susceptibility of this organism to 16 antimicrobial agents was then determined via an E-test. For Gordonia spp., there are no interpretive minimal inhibitory concentration (MIC) breakpoints of antimicrobial agents recommended by the Clinical and Laboratory Standards Institute. The interpretation of the MICs used in our study is based on the proximity to bacterial colonies as well as the limited literature on Gordonia spp. (Table 1).

Table 1. MICs of Isolated Gordonia sputiDetermined by an E-test

Antimicrobial Agent	MIC (μg/mL)	Interpretation
Meropenem	0.016	S
Cefoxitin	24	R
Oxacillin	1.5	I
Vancomycin	0.25	S
Ampicillin	0.38	S
Tetracycline	1.5	N [*]
Rifampicin	0.094	S
Clindamycin	0.25	S
Moxifloxacin	0.094	S
Streptomycin	>256	R
Azithromycin	1	S
Metronidazole	>256	R
Erythromycin	0.5	S
Levofloxacin	0.125	S
Chloromycetin	1.5	I
Teicoplanin	0.38	S

Note. MICs, minimum inhibitory concentrations; S, susceptible; R, resistant; I, intermediate; ^{*}N, not available.

A member of the mycolic acid-containing group (Corynebacterium, Dietzia, Gordonia, Mycobacterium, Nocardia, Rhodococcus, and Tsukamurella), the genus Gordonia includes 31 species of actinomycetes^[5]. However, because of the frequently inconclusive biochemical results and time-consuming culture process for Gordonia spp., these bacteria are not generally included in routine detection procedures in clinical microbiology departments. Moreover, some strains belong to the genus Rhodococcus or Nocardia as well as to thus increasing the difficulty of Gordonia, determining the taxonomy of this group. Since 1971, the Gordonia spp. has been increasingly recognized as a pathogen, particularly in immunocompromised and immunocompetent individuals. The major pathogenic Gordonia spp.^[6] are G. sputi, G. bronchialis, and G. terrae. These species cause neurologic and vascular infections as well as cutaneous and respiratory infections, otitis externa, osteitis, and arthritis. The limited literature available implies that G. sputi plays a more dominant role in clinical infections caused by Gordonia spp. Formerly classified as Rhodococci, G. sputi was initially named Rhodococcus sputic (1975) and then renamed Gordona sputi (1989). Subsequently, the name Gordona was changed to Gordonia in 1997^[7]. From 1996 to 2015, 16 cases of infections caused by G. sputi in patients aged 13 days to 78 years were reported worldwide. Of these cases, 9 presented with systemic bacteremia, 3 with continuous dialysis (CAPD)-related ambulatory peritoneal peritonitis, and 4 with different types of infection. Eight of the nine bacteremia cases were caused by the use of contaminated central venous catheters (CVC), including Hickman and Groshong catheters, whereas one was due to cutaneous lesions. In the other 4 cases of G. sputi infections, 2 presented with symptoms of keratitis/conjunctivitis, 1 emerged after coronary artery bypass grafting (CABG) using the left internal mammary artery, and 1 may have been caused by a contaminated chest tube but did not present detectable symptoms. Thus, 15 of the 16 aforementioned cases had undergone classic medical device implantation operations, which are generally known to be major risk factors for G. sputi infections. The underlying conditions included diabetes, cancer, and antiretroviral therapy (Table 2).

Medical devices can be considered to be a double-edged sword in healthcare because they can increase the risk of infection. In previous reports of *G. sputi* infection, most cases were due to CVC usage

No.	Age/ Gender	Region	Type of Infection	Underlying Conditions	Antimicrobial Therapy/Outcome	Authors
L L	34	1996, France	Bacteremia, (due to cutaneous lesions)	Metastatic melanoma, (IL-2 treatment)	Penicillins and aminoglycosides	Riegel P et al. (J Clin Microbiol, 1996)
2	62F	1997, Taiwan	Kera titi s /conjunctivitis	Diabetes mellitus	Topical sulfamethoxazole/improved	Lai CC et al. (Clin Microbiol Infect, 2010)
m	54	1999, Japan	Mediastinitis	Surgery	Antibiotics/improved	Kuwabara M et al. (J Cardiovasc Surg, 1999)
4	49/M	1999, Taiwan	Bacteremia, (CVC)	Diabetes mellitus, gastric cancer	Cefoxitin/improved	Lai CC et al. (Clin Microbiol Infect, 2010)
ъ	13d/M	2000, Taiwan	Keratitis /conjunctivitis	Gastroschisis with colon atrial receiving operation	Topical sulfamethoxazole/improved	Lai CC et al. (Clin Microbiol Infect, 2010)
9	31/F	2000, France	Bacteremia, (CVC)	Double heterozygous hemoglobinopathy (hepatitis C with cirrhosis)	Amoxicillin, netilmicin, ceftriaxone/ improved	Lesens O et al. (Emerg Infect Dis, 2000)
7	14/F	2006, Taiwan	Bacteremia, (CVC)	Congenital Hirschsprung's disease, short bowl syndrome, total parenteral nutrition	Imipenem and amikacin lock therapy/ improved	Lai CC et al. (Clin Microbiol Infect, 2010)
ø	69/M	2009, France	Bacteremia, (CVC)	Diabetes, high blood pressure, and alcohol and tobacco addictions	Ticarcillin-clavulanate and ciprofloxacin/ improved	Renvoise A et al. (Emerg Infect Dis, 2009)
6	43/F	2009, USA	Bacteremia, (CVC)	Systemic lupus erythematosus	Vancomycin and imipenem/improved	Brust JC et al. (J Med Microbiol, 2009)
10	46/F	2009, USA	Bacteremia, (CVC)	HIV antiretroviral therapy	vancomycin and imipenem/improved/ died	Brust JC et al. (J Med Microbiol, 2009)
11	78/F	2009, USA	Bacteremia, (CVC)	COPD (chronic obstructive pulmonary disease)	Linezolid	Brust JC et al. (J Med Microbiol, 2009)
12	60/M	2009, USA	Contaminated chest tube?	Pneumonia	Repeat blood cultures remained negative (treated with levofloxacin)	Brust JC et al. (J Med Microbiol, 2009)
13	70/F	2012, Greece	Bacteremia, (CVC)	Breast cancer (radiation therapy and adjuvant chemotherapy)	Teicoplanin and meropenem/improved	Kofteridis DP et al. (J Infect Dev Countries, 2012)
14	M/69	2013, Taiwan	Peritonitis (PD catheter)	ESRD	Cefazolin, gentamicin, vancomycin/ improved	Ou SM et al. (Perit Dial Int, 2013)
15	65/M	2015, Hong Kong	Peritonitis (Tenckhoff catheter)	ESRD	lmipenem–cilastatin, amikacin/improved	Lam JY et al. (J Clin Microbiol, 2015)
16	67/M	2015, Hong Kong	Peritonitis (Tenckhoff catheter)	ESRD	Imipenem—cilastatin, amikacin/improved	Lam JY et al. (J Clin Microbiol, 2015)

Note. CVC, central venous catheter; ESRD, end-stage renal disease; NA, not available.

among patients of internal medicine. To the best of our knowledge, Gordona has not been reported in plastic surgery expander infections. In the United expander/implant-based reconstruction States. remains the most common form of breast reconstruction for most breast cancer cases. In general, most patients are candidates for prosthetic breast reconstruction. The incidence of periprosthetic infection following breast reconstruction using tissue expanders ranges from 1% to 24%^[8]. However, periprosthetic infection poses a significant threat and complicates postoperative recovery. А diverse range of organisms, including Enterococcus faecalis, methicillin-resistant Staphylococcus aureus, Pseudomonas aeruginosa, and Candida parapsilosis, cultured from were such plastic surgical reconstructions. For instance. an aerobic Actinomyces spp. was found in a silicone tissue expander in 2006. However, the specific genus or species of the strain remains unknown^[9]. Moreover, in our case with G. sputi infection, the strain was misidentified as an oxacillin-resistant S. epidermidis at first. These two cases do indicate that infections with Gordonia spp. may be underreported, particularly in surgical departments that use medical devices. However, 16S rRNA gene sequencing has been verified as a simple and efficient identification technique for mycolic acid-containing genus and species.

A review of antimicrobial therapies shows that G. *sputi* has high susceptibility to several antimicrobial drugs, specifically to carbapenems, aminoglycosides, linezolid, and fluoroquinolones. Combinations of penicillins and aminoglycosides, carbapenem or fluoroquinolone, and aminoglycoside are often used to treat Gordonia-related bacteremia. Most patients with G. sputi infection improve with antibiotics therapy (Table 2). Some previous data from Asia revealed that amoxicillin/clavulanic acid. ciprofloxacin, and vancomycin were more favorable agents for treating patients. However, the resistance of Gordonia spp. appears to have increased recently. For example, Gordonia spp. exhibits 11% resistance to vancomycin based on data from 24 clinical isolates^[10]. Meanwhile, ampicillin, trimethoprimsulfamethoxazole, clarithromycin, and penicillin exhibit low activities, with 56% to 70% of the tested isolates showing higher MICs. In our study, we found that the Gordonia spp. exhibited resistance to cefoxitin, streptomycin, and metronidazole. Moreover, oxacillin and chloramphenicol had low

activity against G. sputi. Thus, the resistance of Gordonia spp. should be considered when selecting an appropriate therapy or treatment. The patient in the current study underwent expander removal, wide debridement, and empirical antibiotic treatment with cefmetazole and metronidazole before results of antibiotic sensitivity became available. The patient's condition improved after 1 day of treatment. The discordance of the antibiotic sensitivity reported and treatment result seem to highlight that implant removal and surgical debridement play a more important role than antibiotics in the treatment of localized G. sputi infections.

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