

Mycetomalike Skin Infection Due to *Gordonia bronchialis* in an Immunocompetent Patient

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PRACTICE POINTS

- *Gordonia bronchialis* is an emerging cause of human skin and soft tissue infection, typically occurring after trauma, inoculation, or surgery.
- *Gordonia* species can cause a mycetomalike skin infection.
- Increasing use of molecular methods to identify bacteria has improved identification of clinically relevant actinomycetes, such as *Gordonia*, and increases the likelihood that clinicians will see these organisms on culture results.

Gordonia bronchialis is a partially acid-fast, gram-positive rod that has been found in a variety of nosocomial infections, most frequently sternal wound infection following coronary artery bypass surgery. We report a case of a mycetomalike infection due to *G bronchialis* in an immunocompetent patient with complete resolution after 3 months of oral antibiotics.

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Mycetoma is a chronic subcutaneous infection due to fungal (eumycetoma) or aerobic actinomycetes (actinomycetoma) organisms. Clinical lesions develop from a granulomatous infiltrate organizing

around the infectious organism. Patients can present with extensive subcutaneous nodularity and draining sinuses that can lead to deformation of the affected extremity. These infections are rare in developed countries, and the prevalence and incidence remain unknown. It has been reported that actinomycetes represent 60% of mycetoma cases worldwide, with the majority of cases in Central America from *Nocardia* (86%) and *Actinomadura madurae* (10%).¹ *Gordonia* species are aerobic, partially acid-fast, gram-positive actinobacteria that may comprise a notable minority of actinomycete isolates.² The species *Gordonia bronchialis* is of particular interest as a human pathogen because of increasing reports of nosocomial infections.^{3,4} We describe a case of a mycetomalike infection due to *G bronchialis* in an immunocompetent patient with complete resolution after 3 months of antibiotics.

Case Report

An 86-year-old man presented to the emergency department with a pruritic rash on the right forearm. He had a history of chronic kidney disease, hypertension, and inverse psoriasis complicated by steroid atrophy. He reported trauma to the right antecubital fossa approximately 1 to 2 months prior from a car door; he received wound care over several weeks at an outside hospital. The initial wound healed completely, but he subsequently noticed erythema spreading down the forearm. At the current presentation, he was empirically treated

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with mid-potency topical steroids and cefuroxime for 7 days. Initial laboratory results were notable for a white blood cell count of 5.7×10^3 cells/ μL (reference range, $3.7\text{--}8.4 \times 10^3$ cells/ μL) and a creatinine level of 1.5 mg/dL (reference range, 0.57–1.25 mg/dL). The patient returned to the emergency department 2 weeks later with spreading of the initial rash and worsening pruritus. Dermatologic evaluation revealed the patient was afebrile and had violaceous papules and nodules that coalesced into plaques on the right arm, with the largest measuring approximately 15 cm. Areas of superficial erosion and crusting were noted (Figure 1A). The patient denied constitutional symptoms and had no axillary or cervical lymphadenopathy. The differential initially included an atypical infection vs a neoplasm. Two 5-mm punch biopsies were performed, which demonstrated a suppurative granulomatous infiltrate in the dermis with extension into the subcutis (Figure 2A). Focal vacuolations within the dermis demonstrated aggregates of gram-positive pseudofilamentous organisms (Figures 2B and 2C). Aerobic tissue cultures grew *G bronchialis* that was susceptible to all antibiotics tested and *Staphylococcus epidermidis*. Fungal and mycobacterial cultures were negative. The patient was placed on amoxicillin 875 mg–clavulanate 125 mg twice daily for 3 weeks. However, he demonstrated progression of the rash, with increased induration and confluence of plaques on the forearm (Figure 1B). A repeat excisional biopsy was performed, and a tissue sample was sent for 16S ribosomal RNA sequencing identification. However, neither conventional cultures nor sequencing demonstrated evidence of *G bronchialis* or any other pathogen. Additionally, bacterial, fungal, and mycobacterial blood cultures were negative. Amoxicillin-clavulanate was stopped, and he was placed on trimethoprim-sulfamethoxazole for 2 weeks, then changed to linezolid (600 mg twice daily) due to continued lack of improvement of the rash. After 2 weeks of linezolid, the rash was slightly improved, but the patient had notable side effects (eg, nausea, mucositis). Therefore, he was switched back to trimethoprim-sulfamethoxazole for another 6 weeks. Antibiotic therapy was discontinued after there was notable regression of indurated plaques (Figure 1C); he received more than 3 months of antibiotics in all. At 1 month after completion of antibiotic therapy, the patient had no evidence of recurrence.

Comment

Microbiology of *Gordonia Species*—*Gordonia bronchialis* originally was isolated in 1971 by Tsukamura et al⁵ from the sputum of patients with cavitary tuberculosis and bronchiectasis in Japan. Other *Gordonia* species (formerly *Rhodococcus* or *Gordona*) later were identified in soil, seawater, sediment, and wastewater. *Gordonia bronchialis* is a gram-positive aerobic actinomycete short rod that organizes in cordlike compact groups. It is weakly acid fast, nonmotile, and nonsporulating. Colonies exhibit pinkish-brown pigmentation. Our understanding of the



FIGURE 1. A, Initial presentation with a massive purple to violaceous nodular plaque measuring 15 cm in greatest diameter. B, Numerous areas with serosanguineous drainage and crusting. There was interim progression with an increase in confluent indurated plaques after 3 weeks of amoxicillin 875 mg–clavulanate 125 mg twice daily. C, Minimal scarring and postinflammatory hyperpigmentation was seen 1 month after completion of antibiotic therapy.

clinical significance of this organism continues to evolve, and it is not always clearly pathogenic. Because *Gordonia* isolates may be dismissed as commensals or misidentified as *Nocardia* or *Rhodococcus* by routine biochemical tests, it is possible that infections may go undetected. Speciation requires gene sequencing; as our utilization of molecular methods has increased, the identification of clinically relevant aerobic actinomycetes, including *Gordonia*, has improved,⁶ and the following species have been recognized as pathogens: *Gordonia araii*, *G bronchialis*, *Gordonia effusa*, *Gordonia otitidis*,

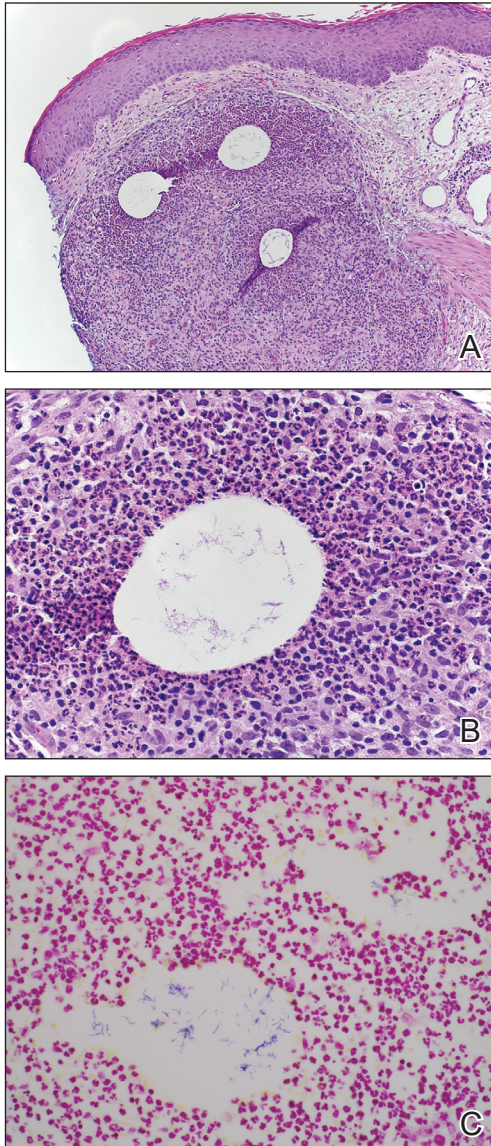


FIGURE 2. A, A 5-mm punch biopsy of the right forearm nodularity demonstrated a robust neutrophilic and histiocytic inflammatory infiltrate surrounding vacuolations within the papillary dermis (H&E, original magnification $\times 100$). B, Clumped pseudofilamentous organisms within vacuolated spaces were seen on higher magnification (H&E, original magnification $\times 400$). C, Gram-positive rods were seen (Gram, original magnification $\times 600$).

Gordonia polyisoprenivorans, *Gordonia rubripertincta*, *Gordonia sputi*, and *Gordonia terrae*.⁷

Cases Reported in the Literature—A PubMed search of articles indexed for MEDLINE using the term *Gordonia bronchialis* yielded 35 previously reported human cases of *G bronchialis* infection, most often associated with medical devices or procedures.⁸⁻³¹ Eighteen of these cases were sternal surgical site infections in patients with a history of cardiac surgery,^{3,4,12-16,30} including 2 outbreaks following coronary artery bypass grafting that were thought to be related to intraoperative transmission from a nurse.^{3,4}

Of the remaining cases, 12 were linked to a procedure or an indwelling catheter: 4 cases of peritonitis in the setting of continuous ambulatory peritoneal dialysis^{17,18,26,27}; 3 cases of skin and soft tissue infection (1 at the site of a prior needle injection,¹⁰ 1 after acupuncture,¹¹ and 1 after breast reduction surgery²⁹); 1 case of ventriculitis in a premature neonate with an underlying intraventricular shunt¹⁹; 2 cases of pacemaker-induced endocarditis^{20,28}; 1 case of tibial osteomyelitis related to a bioresorbable polymer screw²¹; and 1 case of chronic endophthalmitis with underlying intraocular lens implants.²² The Table lists all cases of *G bronchialis* skin or surgical site infections encountered in our literature search as well as the treatment provided in each case.

Only 4 of these 35 cases of *G bronchialis* infections were skin and soft tissue infections. All 4 occurred in immunocompetent hosts, and 3 were associated with needle punctures or surgery. The fourth case involved a recurrent breast abscess that occurred in a patient without known risk factors or recent procedures.²³ Other *Gordonia* species have been associated with cutaneous infections, including *Gordonia amicalis*, *G terrae*, and recently *Gordonia westfalica*, with the latter 2 demonstrating actinomycetoma formation.³²⁻³⁴ Our case is remarkable in that it represents actinomycetoma due to *G bronchialis*. Of note, our patient was immunocompetent and did not have any radiation or chronic lymphedema involving the affected extremity. However, his history of steroid-induced skin atrophy may have predisposed him to this rare infection.

Clinical Presentation—Classic mycetoma demonstrate organismal granules within the dermis, surrounded by a neutrophilic infiltrate, which is in turn surrounded by histiocytes and multinucleated giant cells. Periodic acid-Schiff and silver stains can identify fungal organisms, while Gram stain helps to elucidate bacterial etiologies.¹ In our patient, a biopsy revealed several dermal aggregates of pseudofilamentous gram-positive organisms surrounded by a neutrophilic and histiocytic infiltrate.⁸ Because this case presented over weeks to months rather than months to years, it progressed more rapidly than a classic mycetoma. However, the dermatologic and histologic features were consistent with mycetoma.

Management—General treatment of actinomycetoma requires identification of the causative organism and prolonged administration of antibiotics, typically in combination.³⁵⁻³⁷ Most *G bronchialis* infections associated with surgical intervention or implants in the literature required surgical debridement and removal of contaminated material for clinical cure, with the exception of 3 cases of sternal wound infection and 1 case of peritonitis that recovered with antimicrobial therapy alone.^{3,17} Combination therapy often was used, but monotherapy, particularly with a fluoroquinolone, has been reported. Susceptibility data are limited, but in general, *Gordonia* species appear susceptible to imipenem, ciprofloxacin, amikacin, gentamicin, and linezolid, with variable susceptibility to vancomycin

Reported Cases of *Gordonia bronchialis* Causing Skin or Surgical Site Infections

Reference (year)	No. of cases	Age, y	Sex	Comorbid conditions	Infection type	Surgical therapy	Antimicrobial therapy
Richet et al ⁹ (1991)	7	51–68	M	CABG	Sternal wound infection	Surgical debridement in 4 patients; 2 of 4 had sternum removed and flap grafts	3 patients treated with antibiotics alone: ciprofloxacin × 74 d, trimethoprim-sulfamethoxazole × 122 d, ceftriaxone × 38 d + ciprofloxacin × 108 d; 4 patients treated with antibiotics plus surgical debridement (details not described)
Werno et al ²³ (2005)	1	43	F	Pituitary adenoma	Recurrent breast abscess	Incision and drainage × 3	Penicillin + flucloxacillin × 3 d, amoxicillin-clavulanate + metronidazole × 3 d, doxycycline + clindamycin × 12 d, doxycycline × 5 mo
Wright et al ⁴ (2012)	3	56–80	M	CABG	Sternal wound infection	Surgical debridement and flap grafts	All 3 patients required prolonged IV antibiotics, including imipenem 41–77 d (mean, 60 d); 1 patient received an additional 8 wk of PO antibiotics (moxifloxacin, linezolid, minocycline) for persistently elevated inflammatory markers
Nguyen et al ¹² (2014)	3	47–68	M/F	CABG	Sternal wound infection	Wound debridement, negative pressure therapy	Antimicrobial therapy not described
Chang et al ¹³ (2014)	1	69	F	CABG	Sternal osteomyelitis	Surgical debridement	Imipenem × 8 wk
Bartolomé-Álvarez et al ¹⁰ (2016)	1	50	F	Prior needle injection	Cutaneous abscess	Abscess drainage	Amoxicillin-clavulanate × 10 d
Rodríguez-Lozano et al ¹⁴ (2016)	1	64	F	Mitral valve replacement for rheumatic mitral stenosis	Sternal wound infection	Surgical debridement	Imipenem + ciprofloxacin × 3 wk, teicoplanin + ciprofloxacin + rifampin × 2 wk, ciprofloxacin + rifampin × 6 wk
Akrami et al ¹⁵ (2017)	1	69	M	CABG	Sternal osteomyelitis	Surgical debridement	Ceftaroline × 8 wk
Ambesh et al ¹⁶ (2019)	1	74	M	CABG	Sternal osteomyelitis	Surgical debridement	Vancomycin + meropenem (duration not reported)
Choi et al ¹¹ (2019)	1	61	F	Developed after acupuncture and cupping therapy	Infected cutaneous nodule	Punch biopsy	Cefpodoxime × 6 wk

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TABLE. (continued)

Reference (year)	No. of cases	Age, y	Sex	Comorbid conditions	Infection type	Surgical therapy	Antimicrobial therapy
Davidson et al ²³ (2022)	1	26	F	Breast reduction surgery	Delayed, recurrent infection at the surgical site	Incision and drainage	Amoxicillin-clavulanate ×6 wk
Nwaedozie et al ³⁰ (2022)	1	81	M	CABG	Sternal osteomyelitis	Surgical debridement	Ampicillin-sulbactam ×6 wk, then amoxicillin-clavulanate ×6 wk
Current report	1	86	M	Inverse psoriasis complicate by steroid atrophy	Mycetomalike cellulitis	None	Various antibiotics for >3 mo

Abbreviations: CABG, coronary artery bypass graft; F, female; IV, intravenous; M, male; PO, by mouth.

(89% of isolates), third-generation cephalosporins (80%–90% of isolates), tetracyclines ($\leq 85\%$ of isolates), penicillin ($\leq 70\%$ of isolates), and trimethoprim-sulfamethoxazole ($\leq 65\%$ of isolates).^{7,10,19,38–40} Although there are no standardized recommendations for the treatment of these infections, the most commonly used drugs to treat *Gordonia* are carbapenems and fluoroquinolones, with or without an aminoglycoside, followed by third-generation cephalosporins and vancomycin, depending on susceptibilities. Additional antibiotics (alone or in combination) that have previously been used with favorable outcomes include amoxicillin or amoxicillin-clavulanate, piperacillin-tazobactam, rifampicin, trimethoprim-sulfamethoxazole, minocycline, doxycycline, and daptomycin.

Our patient received amoxicillin-clavulanate, trimethoprim-sulfamethoxazole, and linezolid. We considered combination therapy but decided against it due to concern for toxicity, given his age and poor renal function. The antibiotic that was most important to his recovery was unclear; the patient insisted that his body, not antibiotics, deserved most of the credit for healing his arm. Although cultures and polymerase chain reaction assays were negative after 3 weeks of amoxicillin-clavulanate, the patient did not show clinical improvement—reasons could be because the antibiotic reduced but did not eliminate the bacterial burden, sampling error of the biopsy, or it takes much longer for the body to heal than it takes to kill the bacteria. Most likely a combination of factors was at play.

Conclusion

Gordonia bronchialis is an emerging cause of human infections typically occurring after trauma, inoculation, or surgery. Most infections are localized; however, the present case highlights the ability of this species to form a massive cutaneous infection. Treatment should be tailored to susceptibility, with close follow-up to ensure improvement and resolution. For clinicians encountering a similar case, we encourage biopsy prior to empiric antibiotics, as antibiotic therapy can decrease the yield of subsequent testing. Treatment should be guided by the clinical course and may need to last weeks to months. Combination therapy for *Gordonia* infections should be considered in severe cases, in cases presenting as actinomycetoma, in those not responding to therapy, or when the susceptibility profile is unknown or unreliable.

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