not analyze changes in the outer membrane proteins responsible for alteration of permeability.

Continual monitoring of drug resistance patterns is imperative. Antimicrobial drug susceptibility testing should be conducted for clinical isolates, and empirical antimicrobial drug therapy should be changed accordingly. AmpC β-lactamase genes will eventually be transferred to typhoidal salmonellae, which may pose a threat to public health. Spread of broad-spectrum β-lactamases would greatly limit therapeutic options and leave only carbapenems and tigecycline as secondary antimicrobial drugs.

Bindiganavile N. Gokul, Godfred A. Menezes, and Belgode N. Harish

Author affiliations: Sri Devaraj Urs Medical College, Kolar, India (B.N. Gokul, G.A. Menezes); Fortis Hospitals, Bangalore, India (B.N. Gokul); and Jawaharlal Institute of Postgraduate Medical Education and Research, Pondicherry, India (G.A. Menezes, B.N. Harish)

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Address for correspondence: Belgode N. Harish, Department of Microbiology, Jawaharlal Institute of Postgraduate Medical Education and Research, Pondicherry 605006, India; email: drbnharish@yahoo.com

Endocarditis Caused by Actinobaculum schaali, Austria

To the Editor: In May 2009, a 52-year-old man was hospitalized with middle cerebral artery stroke and fever of unknown origin. He had a complicated medical history of middle cerebral artery stroke and mechanical valve replacement of the aortic valve 2 years earlier and gastric-duodenal angiodysplasia. Two months before the most recent hospitalization, he had been hospitalized because of fever and anemia; blood cultures were positive; Gram stain identified coryneform rods that did not grow in culture. Antimicrobial drug therapy with levofloxacin (400 mg 1×/d) was initiated, and the patient was discharged.

At the most recent admission, laboratory testing showed a leukocyte count of 5.92 × 10⁹ cells/µL, with 81% neutrophils, 7% lymphocytes, and 9% monocytes; thrombocyte count was 338 × 10³ cells/µL. C-reactive protein level was 62.6 mg/L (reference value <8 mg/L). Basic serum and urine chemical profiles and urine culture were unremarkable. Empiric antimicrobial drug therapy with piperacillin–tazobactam (4.5 g 3×/d) was initiated and discontinued after 5 days because of clinical improvement. The next day, the patient’s condition deteriorated, C-reactive protein level increased from 15 mg/L to 32 mg/L, and blood was collected for culture on the day after piperacillin-tazobactam discontinuation and the next 2 days. After 4 days of incubation, bacterial growth was detected in 1 aerobic and 3 anerobic samples. Gram stain showed positive coryneform rods. Within 48–72 hours, the isolate yielded growth on blood, chocolate, and Schaedler agar; colonies were 1–2 mm in diameter and gray. The specificity of the organism was unsatisfactory with the system we used (API Coryne sys-
A 16S rRNA gene analysis was performed by using eubacterial universal primers. Subsequently, a BLAST search (www.ncbi.nlm.nih.gov/BLAST) of the partial 16S rRNA gene sequence (730 bp) was performed by using the taxonomy browser of the National Center for Biotechnology Information (www.ncbi.nlm.nih.gov). Homology of 99.7% (728/730 bp) was detected for *Actinobaculum schaalii*. The isolate was deposited in GenBank under accession no. GQ355962. MICs were obtained for various antimicrobial drugs, including amoxicillin–clavulanic acid (0.25 mg/L), piperacillin–tazobactam (0.125 mg/L), and levofloxacin (1 mg/L).

Infectious disease specialists were consulted. On physical examination, the patient exhibited Janeway lesions on hands and feet and a temperature of 38.4°C. Transesophageal echocardiogram showed filiform vegetation on the aortic valve, which was not consistent with echocardiographic major criteria. According to the modified Duke criteria (5), the patient’s condition fulfilled 1 major clinical criterion (at least 2 positive cultures of blood samples collected 12 hours apart) and 3 minor clinical criteria (prosthetic aortic valve, temperature >38°C, Janeway lesions). Accordingly, definite infective prosthetic valve endocarditis was diagnosed. Intravenous antimicrobial drug therapy with piperacillin-tazobactam (4.5 g 3×/d) was initiated, followed by oral therapy with amoxicillin–clavulanate acid (1 g 3×/d) for 8 weeks. Because a repeated transesophageal echocardiogram 10 days after initiation of antimicrobial drug therapy showed no infective endocarditis, heart surgeons declined to replace the prosthetic valve. The patient’s condition improved, and 2 weeks later he was discharged in good clinical condition.

Four species within the genus *Actinobaculum* have been described: *A. massiliae* (causing urinary tract infection [UTI] and superficial skin infection), *A. urinale* (isolated from human urine), *A. suis*, and *A. schaalii* (1,3,4,6). *A. schaalii*, which is difficult to identify by culture, has been reported to cause UTI in elderly patients with underlying urologic conditions; a few studies have reported subsequent urosepsis, abscess formations, and osteomyelitis (1,3,6–9). Recently, Bank et al. (7) reported development of a TaqMan real-time quantitative PCR for *A. schaalii* and consecutive detection of the organism in 22% of 252 routine urine samples of patients.

| Isolate that caused endocarditis | Reaction of (reference)* |
|----------------------------------|--------------------------|
| *Actinobaculum schaalii* (1,2) | *Actinobaculum massiliae* (3) | *Actinobaculum urinale* (4) | Arcanobacterium pyogenes | Actinomyces turicensis (1,2) |
| Catalase reaction | – | – | – | – | – |
| β-hemolysis on sheep blood agar | – | – | – | – | – |
| Nitrate reduction | – | – | – | – | – |
| Pyrazinamidase | – | – | – | – | – |
| Pyrrolidonyl arylamidase | + | + | – | – | – |
| Alkaline phosphatase | – | – | – | – | – |
| β-glucuronidase | – | – | – | – | – |
| β-galactosidase | – | – | – | – | – |
| α-glucosidase | – | – | – | – | – |
| N-acetyl-β-glucosaminidase | – | – | – | – | – |
| Esculin hydrolysis | – | – | – | – | – |
| Urease activity | – | – | – | – | – |
| Gelatin hydrolysis | – | – | – | – | – |

### Table. Comparison of isolated *Actinobaculum schaalii* with related human pathogens reported in the literature

- **Catalase reaction**: – or + indicates presence or absence, respectively. 
- **β-hemolysis on sheep blood agar**: –, >90% strains negative; +, >90% strains positive. 
- **Nitrate reduction**, **Alkaline phosphatase**, **β-glucuronidase**, **β-galactosidase**, **α-glucosidase**, **N-acetyl-β-glucosaminidase**, **Esclusin hydrolysis**, **Urease activity**, **Gelatin hydrolysis**: –, negative; +, positive. 
- **Acid from**: –, negative; +, positive. 

*API Coryne system (bioMérieux, Craponne, France) profile for our isolate was compared with those described in the references (14 strains of *Actinobaculum schaalii*, 1 strain of *A. massiliae*, 1 strain of *A. urinale*, 43 strains of *A. turicensis* and those in the manufacturer’s database for *Arcanobacterium pyogenes*, +, >90% of strains positive; –, >90% strains negative; V, variable; w, weak. 
†In API Coryne, the strain gave the profile number 4110621 (unacceptable profile because of lack of specificity). 
‡*A. schaalii* was described as nonhemolytic for 5 patients (1) and as showing weak β-hemolysis only after 2–5 d in 9 cases (2). 
§Reported as positive for 1 of 14 strains.
>60 years of age (8). Those findings suggest that A. schaalii is a common undetected pathogen, especially in elderly patients with unexplained chronic UTI.

We report infective endocarditis caused by A. schaalii. To our knowledge, infective endocarditis caused by Actinobaculum spp. has not been reported. However, several reports have documented endocarditis caused by Arcanobacterium spp. and Actinomyces spp., which are phylogenetically related to Actinobaculum spp. (10).

Characteristics of the patient reported here differed from those of patients in previous reports. He had no underlying urologic condition and could not recall any symptoms usually associated with UTI during the year before hospital admission. Urine culture remained negative for Actinobaculum spp. despite prolonged incubation for 5 days on chocolate agar in an atmosphere of 5% CO2 and on Schaedler agar under anaerobic conditions.

This report highlights the usefulness of the recent development of a specific real-time PCR by Bank et al. (7), which may prove effective not only for patients typically at risk for A. schaalii but also for patients with a wider spectrum of infection. More studies are needed to identify the real prevalence of disease caused by this difficult-to-cultivate organism because it may occur in many other groups of patients.

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Author affiliation: Medical University of Graz, Graz, Austria

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Address for correspondence: Andrea J. Grisold, Institute of Hygiene, Microbiology and Environmental Medicine, Medical University of Graz, Universitaetsplatz 4, A-8010 Graz, Austria; email: andrea.grisold@medunigraz.at

Mycobacterium chelonae Wound Infection after Liposuction

To the Editor: We recently investigated a case of Mycobacterium chelonae abdominal wound infection after liposuction performed under local anesthesia at an outpatient medical office. Our aim was to determine whether other cases of atypical mycobacterial infections had previously occurred after liposuction. M. chelonae is widely distributed in soil and water, including tap water. Atypical mycobacterial infections have been associated with skin and soft tissue infections, including infections after cosmetic surgeries, and outbreaks have been documented (1–4). Previously reported potential sources of liposuction equipment contamination have been inadequate disinfection or sterilization after rinsing of liposuction equipment with tap water, tap water used in cleaning liposuction cannulae, or the quaternary ammonium solution used to disinfect liposuction equipment (2,4). Increased numbers of procedures performed in freestanding medical centers (not connected with hospitals) that are not routinely monitored by infection control committees or equivalent oversight bodies may contribute to atypical mycobacterial infection (1).

Our investigation showed that proper cleaning, disinfection, and sterilization of liposuction equipment and other infection control issues at this medical office were concerns.