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## Native Valve Endocarditis due to *Corynebacterium striatum*

*Corynebacterium* species are nonsporulating, nonbranching, non-acid-fast, gram-positive bacilli that are part of the normal flora of the skin and mucous membranes. When isolated from clinical specimens, they are most often considered to represent contaminants, although they are capable of causing significant infection, particularly in the presence of intravenous catheters or prosthetic devices. Prosthetic valve endocarditis due to corynebacteria is a well-recognized entity, most often caused by *Corynebacterium jeikeium* (1). Native valve endocarditis due to *Corynebacterium* species has also been described, although less frequently (2). We report a case of native valve endocarditis due to *Corynebacterium striatum*.

A 68-year-old man was admitted to hospital with a three-day history of anorexia, fatigue, and confusion. He had not noted any fever, sweats, or chills. His past medical history included non-insulin dependent diabetes mellitus, hypertension, and congestive heart failure. He was a cigarette smoker and had a history of chronic obstructive pulmonary disease. He had not had any recent dental work, and was not taking any antibiotics.

Physical examination revealed moderate respiratory distress, with a temperature of 38.4°C and a blood pressure of 100/55 mm Hg. He had atrial fibrillation with a heart rate of 100/min. The central venous pressure was elevated and there were signs of left ventricular failure. There was a loud pansystolic murmur at the apex, radiating into the left axilla. The abdominal examination was normal. He was intermittently confused, but the neurological examination was otherwise unremarkable. There were no skin lesions and no peripheral stigmata of endocarditis. The hemoglobin level was 125 g/l, the leukocyte count 8800/mm$^3$, and the platelet count 640,000/mm$^3$. A chest radiograph demonstrated changes consistent with left ventricular failure but no evidence of pulmonary consolidation.

Three sets of blood cultures obtained on admission grew *Corynebacterium striatum*. Treatment with intravenous vancomycin was started and the patient became afebrile. A transthoracic echocardiogram revealed moderate left ventricular dysfunction and mitral valve regurgitation (unchanged from an echocardiogram done two years earlier). In the absence of any clinical or echocardiographic evidence of infective endocarditis, or of other foci of infection, antimicrobial therapy was discontinued after six days. However, one week after discontinuing antibiotics, the patient again became febrile. Two additional blood cultures were obtained, and both grew *Corynebacterium striatum*. At this time, a transesophageal echocardiogram was done which demonstrated severe mitral regurgitation and a vegetation on the atrial aspect of the anterior mitral valve leaflet. Treatment with vancomycin was restarted; this was

switched to intravenous penicillin when it was determined that the isolate was susceptible to penicillin. Antimicrobial therapy was continued for a total of six weeks.

*Corynebacterium striatum* was identified on the basis of Gram stain appearance, colonial morphology, and standard biochemical test reactions (3). The identity was further confirmed by cellular fatty acid analysis by gas-liquid chromatography, using the Microbial Identification System (Microbial ID, USA) and CLIN version 3.9 library database (4). Susceptibility to penicillin was determined by an agar dilution method. The isolate was found to be susceptible to penicillin with a minimum inhibitory concentration of 0.5 μg/ml.

Most nondiphtherial corynebacteria colonize skin and mucous membranes. They are uncommon causes of human infection, and when isolated from blood cultures, they are most often assumed to represent contaminants. Infective endocarditis due to *Corynebacterium* species is infrequently reported, and the species most often identified are *Corynebacterium jeikeium* (5, 6) and *Corynebacterium pseudodiphtheriticum* (6, 7). *Corynebacterium striatum* has rarely been reported to be pathogenic although bacteremia, pleuropulmonary infections and chorioamnionitis have been reported (2, 8). Two previous cases of endocarditis due to *Corynebacterium striatum* have been described (6, 9), and there has been one other case of endocarditis due to an organism resembling *Corynebacterium striatum* (10) (Table 1). Although pathologic confirmation was not available, the present case fulfils clinical diagnostic criteria for infective endocarditis (11). These four cases of *Corynebacterium striatum* endocarditis all involved native valves, and, with the exception of the present case, there was no known underlying valvular disease. In one case (9), endocarditis was associated with an infected endocardial pacemaker wire. No source of infection could be determined for the other cases (including the current case), although bacteremia presumably arose from a site of cutaneous or mucosal colonization with *Corynebacterium striatum*.

In summary, *Corynebacterium striatum* is now recognized as a pathogenic microorganism capable of causing a variety of human infections, including infective endocarditis. Therefore, *Corynebacterium* species isolated repeatedly from blood cultures should not automatically be assumed to represent contaminants, even in patients without prior structural damage to their heart valves. Early recognition of infection and institution of appropriate antimicrobial therapy should reduce the risk of subsequent complications.

*Note added in proof:* A fifth case of endocarditis due to *Corynebacterium striatum* has recently been published (reference 12).

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**Table 1:** Four cases of infective endocarditis due to *Corynebacterium striatum*.

| Reference no. | Year | Age/Sex | Valve | Previous valvular disease | Underlying disease | Therapy | Outcome |
|---|---|---|---|---|---|---|---|
| 10* | 1990 | 76/M | aortic | no | none | ampicillin, gentamicin | died |
| 6 | 1994 | 54/M | aortic | no | hypertension | ampicillin, gentamicin, vancomycin | survived; aortic valve replaced |
| 9 | 1996 | 73/M | tricuspid | no | infected permanent pacemaker electrode wire | vancomycin | survived |
| Present case | 1996 | 68/M | mitral | yes | diabetes mellitus, congestive heart failure | vancomycin, penicillin | survived |

* Organism most closely resembled *Corynebacterium striatum*

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## Catalase-Negative *Listeria monocytogenes* Causing Lethal Sepsis and Meningitis in an Adult Hematologic Patient

*Listeria monocytogenes* is an ubiquitous gram-positive, beta-hemolytic, facultative intracellular bacterium that is almost always catalase positive (1). Infections due to *Listeria monocytogenes*, particularly those of the central nervous system, are characterized by a high mortality rate (20–50%) (1). Whether catalase activity is a virulence factor of *Listeria monocytogenes* is a matter of controversy (2–5); however, catalase activity is used to identify *Listeria monocytogenes* in differentiation schemes.

We describe a fatal case of sepsis and meningitis in an adult hematologic patient caused by a catalase-negative isolate of *Listeria monocytogenes*.

A 69-year-old woman was hospitalized in the Department of Dermatology at the University Hospital Hamburg-Eppendorf, Germany, because of erythrodermia due to cutaneous T-cell lymphoma. After three infusions of methotrexate, 20 mg once weekly, progressive leukopenia developed. The patient complained of fever with chills, progressive somnolence, and nuchal rigidity. Her fever rose to 39.5°C despite empiric antibiotic therapy with ciprofloxacin 200 mg b.i.d., administered intravenously. Upon admission to the intensive care unit, pancytopenia was noted, with a total leukocyte count of 600/μl (normal, 4000–11,000/μl), hemoglobin of 9.3 g/dl (normal, 11.5–16.5 g/dl), and platelet count of 80,000/μl (normal, 150,000–450,000/μl). The analysis of the cerebrospinal fluid (CSF) showed 80 cells/μl (normal, 0–5), glucose of 20 mg/dl (normal, > 50% of serum glucose with 90 mg/dl), and total protein of 500 mg/l (normal, < 500 mg/l).

Microscopically, Gram stains of the CSF showed a few gram-positive rods and leukocytes. Gram-positive rods were cultured from CSF and blood; the colonies displayed beta-hemolysis on sheep blood agar (5% v/v). Catalase activity was tested by transferring a loopful of growth to a microscope slide and observing the evolution of bubbles after addition of a drop of 3% $H_2O_2$ (6). The isolates from both CSF and blood displayed negative catalase activity after primary isolation on sheep blood agar and were reproducibly catalase negative after subculture on trypticase soy agar (Oxoid, UK), Mueller-Hinton agar (Oxoid), and sheep blood agar, whereas reference strains of *Staphylococcus aureus* and *Listeria monocytogenes* were consistently catalase positive. Additionally, neither isolate displayed catalase activity using cells from log- and early stationary-phase growth after aerobic incubation in trypticase soy broth (Oxoid). Despite the lack of catalase activity, the isolates were identified as *Listeria monocytogenes* by the API 20 Strep, the API Coryne, and the API Listeria (bioMérieux, France) as well as by positive motility after incubation in trypticase soy broth at 30°C.

The biochemical identifications were confirmed by both *Listeria monocytogenes*-specific polymerase chain reaction (PCR) and Western blot analyses. For PCR, genus- and species-specific primer pairs derived from the *Listeria monocytogenes iap* gene were used (7). The amplification products ob-