Fatal Case of Community-Acquired Bacteremia and Necrotizing Fasciitis Caused by \textit{Chryseobacterium meningosepticum}: Case Report and Review of the Literature

Ching-Chi Lee, Po-Lin Chen, Li-Rong Wang, Hsin-Chun Lee, Chia-Ming Chang, Nan-Yao Lee, Chi-Jung Wu, Hsin-I Shih, and Wen-Chien Ko

Departments of Internal Medicine and Pathology, National Cheng Kung University Hospital, and Department of Medicine, College of Medicine, National Cheng Kung University, Tainan, Taiwan

Received 23 August 2005/Returned for modification 2 October 2005/Accepted 4 December 2005

A diabetic patient with chronic heart failure developed necrotizing fasciitis and bacteremia caused by \textit{Chryseobacterium meningosepticum}, which rapidly evolved into death, even with fasciotomy and intensive care. A review of the English literature found 10 cases of soft tissue infection caused by \textit{C. meningosepticum}, which is rarely acquired in the community.

CASE REPORT

A 62-year-old diabetic man with coronary artery and rheumatic heart disease received a mitral valve replacement by mechanical valve and coronary artery bypass in October 2000. He took regular medications, including oral warfarin, glibenclamide, metformin hydrochloride (Glucophage), digoxin, and furosemide. In January 2005, he developed bilateral lower-leg edema, progressive dyspnea, orthopnea, and a decline in urine output for 3 days until erythema and exquisite pain of the left lower leg made him visit the emergency department of the hospital. He denied fever, chills, and a history of trauma and contact with water. On initial physical examination, he had a blood pressure of 66/48 mm Hg, a pulse rate of 78/min, a body temperature of 37.8°C, and a respiratory rate of 22/min. He had feeling in his left lower limb. Some bullae with clear content and erythematous skin were observed. Initial laboratory tests showed leukocytosis with a left shift (17,200 cells/mm$^3$ with 30% band form). The serum level of C-reactive protein was 39.1 mg/liter. Acute renal failure (serum creatinine, 0.38 mg/dl) and an elevated aspartate aminotransferase level (64 U/liter) were found. Chest X-ray film showed marked cardiomegaly and bilateral pulmonary congestion. Cardiac sonography revealed four-chamber dilation and global hypo-kinesis with impaired performance of the left ventricle. Medical therapy for cardiogenic shock was instituted, and empirical antibiotic with intravenous cefpirome was administrated. Color duplex sonography of the left lower leg revealed no evidence of deep vein thrombosis.

On the next day, more tenderness and hemorrhagic bullae were noted for his left lower leg (Fig. 1). Intravenous ciprofloxacin was initiated based on initial susceptibility results by the disk diffusion technique. On the third day, fasciotomy was undergone, and he was then admitted into the intensive care unit. However, clinical evolution was rapid, and a loss of consciousness, anuria, and multiple organ failure developed. He died on the fourth day.

Nonfermentative, catalase- and oxidase-positive gram-negative rods were discovered in the blood and wound, and both yielded slightly yellow-pigmented colonies on blood agar after 24 h of incubation. They hydrolyzed esculin and could produce acid from mannitol (13, 15). They were identified as \textit{Chryseobacterium meningosepticum} by means of the API 20NE system (BioMerieux, Marcy l’Etoile, France) and the GNI Plus system (Vitek Systems, Marcy l’Etoile, France). They hydrolyzed esculin and could produce acid from mannitol (13, 15). They were identified as \textit{Chryseobacterium meningosepticum} by means of the API 20NE system (BioMerieux, Marcy l’Etoile, France) and the GNI Plus system (Vitek Systems, Marcy l’Etoile, France). The MICs of selected antimicrobial agents measured by E-test strips (AB Biodisk, Solona, Sweden) were 0.38 mg/ml for levofloxacin, 0.25 mg/ml for ciprofloxacin, 0.06-0.32 mg/ml for trimethoprim-sulfamethoxazole, 0.5 mg/ml for minocycline, 1.0 mg/ml for rifampin, 24 mg/ml for vancomycin, 24 mg/ml for cefpirome, and >32 mg/ml for imipenen.

Discussion. The genus \textit{Chryseobacterium} (formerly known as \textit{Flavobacterium}) is a group of non-glucose-fermenting gram-negative rods (15). These bacteria are typically found in fresh-water, saltwater, or soil and are not normally present in the human microflora (2, 15). \textit{C. meningosepticum} is the species most commonly reported as a human pathogen within the genus \textit{Chryseobacterium} and was initially described for a case of neonatal meningitis in 1959 (9). This bacterium has rarely been reported to cause infections among adults, with most cases involving nosocomial pneumonia among intubated patients in intensive care units (11). Other reported infections in adults include bacteremia, subacute bacterial endocarditis, endophthalmitis, and abdominal infections (2).

A MEDLINE search was conducted by using the keywords “\textit{Chryseobacterium meningosepticum}” and “\textit{Flavobacterium meningosepticum}” in combination with “necrotizing fasciitis” or “soft tissue infection” for the time between January 1966 and May 2005. Cases of community-acquired necrotizing fasciitis and bacteremia caused by \textit{C. meningosepticum} have not been described before. Ten cases of soft tissue infections secondary to \textit{C. meningosepticum} infection were reported in the English literature (Table 1). There was an unexpected finding that at
least five patients acquired soft tissue infections in the community, which is contradictory to the notion that *C. meningosepticum* is a nosocomial and opportunistic pathogen. All except two patients had certain chronic underlying diseases, such as liver cirrhosis, diabetes mellitus, chronic heart disease, or malignancy, in accordance with previous reports indicating that *C. meningosepticum* infections often occur in immunocompromised adults (2). Four patients had flame burns involving large body surface areas, and *C. meningosepticum* bacteremia was regarded to result from the disrupted integument. The mortality rate for eight cases of soft tissue infection with reported outcomes was 25%, in contrast to the previous literature showing that higher mortality rates were noted for the postneonatal population with *C. meningosepticum* pneumonia (53%), meningitis (50%), and abdominal infections (100%) (2).

Although published data about the antimicrobial susceptibilities of clinical *Chryseobacterium* isolates are limited and the MIC breakpoints have not been established by the Clinical and Laboratory Standards Institute (5), *Chryseobacterium* isolates are regarded as resistant to many antimicrobial agents commonly prescribed for gram-negative infections, including expanded-spectrum and broad-spectrum cephalosporins, carbapenems, and aminoglycosides (6, 7, 15). However, in vitro susceptibilities determined by the disk diffusion method have been shown to poorly correlate with those by the broth microdilution method, which is the preferred methodology (6). The E-test can be considered an alternative for testing of the susceptibilities of *Chryseobacterium* species to commonly prescribed drugs, except for piperacillin (6, 7). The in vitro susceptibilities of our clinical isolate, as studied by the E-test, showed that minocycline, rifampin, trimethoprim-sulfamethoxazole, and levofloxacin were active in vitro, in accordance with earlier reports (2, 6, 15). Vancomycin had been recommended for *C. meningosepticum* infections (2, 12), but such a notion is increasingly being questioned due to in vitro resistance (8). As for fluoroquinolones, they were usually active against *C. meningosepticum* (8, 16) and resulted in favorable clinical outcomes for cases 2, 9, and 10 in Table 1. Therefore, they are a

![FIG. 1. Bullous skin lesions (arrows) and swelling of left lower leg 24 h after admission.](image)

### Table 1. Summary of 11 cases of soft tissue infections caused by *Chryseobacterium meningosepticum*

<table>
<thead>
<tr>
<th>Patient no./age (yr)/gender</th>
<th>Year of case (reference)</th>
<th>Underlying disease(s)</th>
<th>Infection site</th>
<th>Type of infection</th>
<th>Antimicrobial therapy</th>
<th>Place of acquisition</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/57/F 2/74/M</td>
<td>1989 (3)</td>
<td>None</td>
<td>Left leg</td>
<td>Cellulitis</td>
<td>AMK + CLI</td>
<td>Community</td>
<td>Recovered</td>
</tr>
<tr>
<td></td>
<td>1993 (1)</td>
<td>Congestive heart failure</td>
<td>Bilateral lower legs</td>
<td>Cellulitis, bacteremia</td>
<td>TIM, then CIP</td>
<td>Community</td>
<td>Recovered</td>
</tr>
<tr>
<td>3/14/M</td>
<td>1993 (12)</td>
<td>Burn</td>
<td>Burn wound (85% BSA)</td>
<td>Wound infection, bacteremia</td>
<td>NAF + GEN, then AMK + MED + TIC + CAZ</td>
<td>Hospital</td>
<td>Died</td>
</tr>
<tr>
<td>4/3/M</td>
<td>1993 (12)</td>
<td>Burn</td>
<td>Burn wound (95% BSA)</td>
<td>Wound infection, bacteremia</td>
<td>NAF + GEN, then CLI + SXT + MEZ + CAZ</td>
<td>Hospital</td>
<td>Recovered</td>
</tr>
<tr>
<td>5/84/</td>
<td>2000 (4)</td>
<td>None</td>
<td>NA</td>
<td>Cellulitis, bacteremia</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>6/49/</td>
<td>2000 (4)</td>
<td>Burn</td>
<td>Burn wound</td>
<td>Wound infection, bacteremia</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>7/37/</td>
<td>2004 (10)</td>
<td>Burn</td>
<td>Burn wound</td>
<td>Wound infection, bacteremia</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>8/48/M</td>
<td>2004 (10)</td>
<td>Liver cirrhosis, valvular heart disease</td>
<td>NA</td>
<td>Wound infection, bacteremia</td>
<td>IPM + AMK</td>
<td>Hospital</td>
<td>Recovered</td>
</tr>
<tr>
<td>9/68/M 10/62/M</td>
<td>2004 (10)</td>
<td>Diabetes mellitus</td>
<td>NA</td>
<td>Wound infection, bacteremia</td>
<td>CIP</td>
<td>Community</td>
<td>Recovered</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Nasopharyngeal cancer</td>
<td>NA</td>
<td>Cellulitis, bacteremia</td>
<td>CIP</td>
<td>Community</td>
<td>Recovered</td>
</tr>
<tr>
<td>11/62/M</td>
<td>2005 (present case)</td>
<td>Diabetes mellitus, valvular heart disease</td>
<td>Left lower leg</td>
<td>Necrotizing fasciitis, bacteremia</td>
<td>CPO, then CIP</td>
<td>Community</td>
<td>Died</td>
</tr>
</tbody>
</table>

*AMK, amikacin; BSA, body surface area; CAZ, ceftazidime; CIP, ciprofloxacin; CLI, clindamycin; CPO, cefpirome; GEN, gentamicin; IPM, imipenem; MEZ, mezlocillin; MED, metronidazole; NA, data not available; NAF, nafcillin; SXT, trimethoprim-sulfamethoxazole; TIC, ticarcillin; TIM, ticarcillin-clavulanate.*
promising option, but more clinical experiences are mandatory before their general recommendation for *Chryseobacterium* infections.

In conclusion, *C. meningosepticum* is not only a nosocomial pathogen but is also a causative agent of community-acquired soft tissue infections in immunocompetent individuals.

REFERENCES