Fatal Wound Infection Caused by *Chromobacterium violaceum* in Ho Chi Minh City, Vietnam

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**CASE REPORT**

A 21-month-old human immunodeficiency virus-negative boy was admitted to the children’s ward at the Hospital for Tropical Diseases (HTD), Ho Chi Minh City. The child was distressed and had a fever of 38.5°C, which peaked 3 days later at 40.8°C. The initial clinical presentation and examination suggested viral encephalitis of unknown origin. During the next 4 days, the child’s condition rapidly deteriorated, and he was transferred to the Pediatric Intensive Care Unit at the HTD. A secondary examination identified a small red rash in the vicinity of his right nipple. The area had been scratched and had become inflamed, and the skin was broken; a presumptive diagnosis of sepsis of bacterial origin (*Staphylococcus aureus*) was made. By this time, the fever was slightly reduced (38°C), although he had developed respiratory distress and septic shock, characterized by a sudden drop in white-blood-cell (WBC) and platelet counts (Table 1) and cyanosis of the fingers. He was treated with high doses of intravenous oxacillin, vancomycin, and imipenem, placed on a ventilator, and monitored with intensive supportive measures.

Numerous tests were carried out upon transfer to the Pediatric Intensive Care Unit, including hematology and biochemistry lab tests, a cerebrospinal fluid investigation, a stool examination, and a blood sample test for microbiological blood culture. The blood sample was cultured in a Bactec bottle and incubated in an automated Bactec blood culture identification machine at 37°C. A positive result was recorded on the second day of incubation, and bacteria were isolated for identification. A Gram-stained film demonstrated a gram-negative bacillus. The bacteria were subcultured on blood agar and nutrient agar plates and incubated aerobically at 35°C overnight. The blood plates demonstrated numerous small colonies with a blue pigmentation, while a similar morphology was seen on the nutrient agar plates, although the colonies had a more metallic dark-violet sheen. This pigmentation is associated with *Chromobacterium violaceum* and is due to the production of a chemical called violacin (1). Identification was confirmed by using API 20NE, giving a score of 5152555 (99.9% identification, 0.72 T). The bacterium was named *C. violaceum* HTD1, and unlike the majority of previously reported cases of *C. violaceum* infection, the strain was mannitol positive (8).

*C. violaceum* is a gram-negative, facultative, anaerobic betaproteobacterium which can be routinely isolated from soil and water (10). It is associated in particular with slow-moving or stagnant water sources in tropical and subtropical regions. A swab from the rash on the boy’s chest yielded a growth of *C. violaceum*, suggesting that this was the original entry point of the bacteria, although we were unable to confirm contact with stagnant water. These results were reported to the treating physicians 3 days after blood culture and wound sampling.

*Chromobacterium violaceum* HTD1 was tested for antimicrobial sensitivity, and the results are presented in Table 2. The antimicrobial and MIC testing was performed on Mueller-Hinton agar according to the Clinical and Laboratory Standards Institute guidelines (3). The phenomenon of drug resistance in *C. violaceum* is well known, although the species is usually sensitive to aminoglycosides and chloramphenicol (5, 7).

### TABLE 1. Hematology lab results over the course of *C. violaceum* infection

<table>
<thead>
<tr>
<th>Day(s) postadmission</th>
<th>WBC count (10³/µl)</th>
<th>% Neutrophils</th>
<th>Hemoglobin count (g/dl)</th>
<th>Platelet count (10³/µl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>21</td>
<td>47.5</td>
<td>12.9</td>
<td>320</td>
</tr>
<tr>
<td>4</td>
<td>19.7</td>
<td>86.6</td>
<td>10.1</td>
<td>240</td>
</tr>
<tr>
<td>5</td>
<td>1.62</td>
<td>61.3</td>
<td>10.8</td>
<td>159</td>
</tr>
<tr>
<td>6</td>
<td>8.39</td>
<td>81.3</td>
<td>10.2</td>
<td>86.6</td>
</tr>
<tr>
<td>8</td>
<td>25.9</td>
<td>85.9</td>
<td>7.83</td>
<td>47.9</td>
</tr>
<tr>
<td>9</td>
<td>22.2</td>
<td>83.9</td>
<td>7.99</td>
<td>52.8</td>
</tr>
</tbody>
</table>

* The normal ranges for WBC count, percent neutrophils, hemoglobin count, and platelet count are 4.3 × 10³ to 10.8 × 10³/µl, 45 to 74%, 14 to 18 g/dl, and 150 × 10³ to 350 × 10³/µl, respectively.
This particular isolate demonstrated high-level resistance to all tested cephalosporins; however, it did not exhibit typical extended-spectrum beta-lactamase activity when the combination disc method was used. This suggests a more general efflux-mediated resistance mechanism. Notably, the bacteria were sensitive to imipenem, which was one of the antimicrobials administered to the patient in the treatment cocktail.

Hematology lab results (Table 1) suggested massive bacteremia and septic shock, as the WBC count was initially 21.0 × 10^9/μl and then dropped to 1.62 × 10^9/μl and the platelet count dropped from 240 × 10^9 to 47.9 × 10^9/μl. This occurred in a short time frame (within 4 days), signifying that the sepsis was so severe that it had caused suppression of the bone marrow. The C-reactive protein result of 47.8 mg/liter (normal range, 0.0 to 10.0) was indicative of an immune response stimulated by an infectious agent. Despite the administration of an appropriate antibiotic, the patient failed to respond to treatment and died 9 days after admission.

For further characterization of the fatal bacteria, we isolated DNA from C. violaceum HTD1 and hybridized the DNA using an active surveillance of pathogens (ASP) oligonucleotide microarray (R. A. Stabler, L. F. Dawson, P. C. F. Oyston, R. W. Titball, J. Wade, J. Hinds, A. A. Witney, and B. W. Wren, unpublished data), thus providing data that would have potentially high level of exposure of many humans living in wet tropical areas. Indeed, the bacterium should possibly be described as an accidental rather than an opportunistic pathogen, as infections, like those in this report, are associated with the entry of bacteria through an open wound rather than through consumption of water from a contaminated source.

This fatal case of C. violaceum infection points out the need for rapid diagnosis of wounds contaminated with soil and water in subtropical and tropical areas. Prompt bacteriological isolation, identification, and susceptibility testing, especially in the young, are essential to maximize the treatment of these wounds and to prevent life-threatening sepsis. In this case, the isolation was done promptly despite the possibility of a presumptive diagnosis of Staphylococcus aureus sepsis. Regrettably, the patient died despite treatment with an appropriate antibiotic. The ASP array results demonstrate the utility of this system for rapid molecular identification and will play a significant role in treatment regimens and in monitoring gene acquisition in bacterial species in the future.

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