Pseudomonas putrefaciens as a Cause of Septicemia in Humans

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Septicemia due to Pseudomonas putrefaciens was found in an elderly man with chronic leg ulcers. This organism is rarely cultured from human material and has been reported to cause skin and ear infections in only a few patients. Its potential for invasiveness is documented in this case for the fourth time.

In recent years, several unusual Pseudomonas species have been found to cause opportunistic infections (26). One of them, Pseudomonas putrefaciens, has been associated with spoilage of fish, meat, and butter (2, 11, 14, 25, 26) and has been isolated for the most part from dairy products, soil, sewage, freshwater (8, 17), seawater (21), and oil brines (16). Human isolates have been reported since 1963 (18); in most instances, these represented colonization in the absence of clinical disease (8, 29). However, in a small number of cases, P. putrefaciens has been etiologically linked to ear infections (7, 10, 12, 20) and skin ulcers (4, 7). In two very recent papers (23, 28), three patients with bloodstream invasion have been described. This is another report of sepsis due to this organism.

Case report. A 63-year-old black man was hospitalized for fever and leg ulcers. In the preceding 4 years he had suffered from mild congestive heart failure. At 7 months before admission leg ulcers developed without obvious trauma, and these increased gradually in size. In recent weeks the legs had become painful, forcing him to stop working as a bartender.

He had been an alcoholic until 10 years ago, when he was hospitalized for intestinal bleeding and clinical evidence of liver cirrhosis. He had also experienced several attacks of gouty arthritis in the last 10 years.

Physical examination revealed an obese man with a temperature of 40.4°C. Fine crackling rales were audible at both lung bases. Hepatomegaly (20 by 8 cm) was present, but no ascites or skin changes suggestive of cirrhosis were noted. The lower legs showed moderate pitting edema, with atrophied and hyperpigmented skin. Multiple superficial ulcers, 2 to 6 cm in diameter and covered with greyish eschar, were present above both ankles; a sero-purulent exudate oozed from three small areas. The wound edges were tender, but not raised. Debridement revealed several superficial pockets of yellowish pus without foul odor.

Laboratory data showed a white blood count of 18,200/mm³ with a left shift, a hematocrit of 30%, and a hemoglobin of 9.6 g/100 ml; the erythrocytes were normocytic and normochromic. The lactate dehydrogenase was moderately elevated (214 U/100 ml), as was the alkaline phosphatase (115 U/100 ml); transaminases were in the normal range. Urinalysis was normal, and urine cultures showed no significant growth. Gram stain of the sputum revealed a few polymorphonuclear leukocytes and some gram-positive diplococci, and the sputum culture showed a mixed flora. A chest X ray disclosed moderate cardiomegaly and bilateral perihilar infiltrates consistent with mild congestive failure. X rays of the lower legs showed no osseous involvement.

Bacterial cultures taken from the leg ulcers grew coagulase-positive staphylococci, Enteroxacter agglomerans, and Pseudomonas fluorescens.

Blood cultures from both day 1 and day 4 of hospitalization grew P. putrefaciens in pure culture.

The patient was treated initially with gentamicin parenterally (1 mg/kg every 8 h) and debridement of the wounds. He continued to have a spiking temperature up to 39°C, and chloramphenicol (750 mg every 6 h) was added to the antibiotic regimen. The white blood count remained high, up to 19,000/mm³. Bone (technetium) and gallium scans showed no abnormalities. Because of deteriorating renal function the antibiotics were stopped on day 16, and trimethoprim-sulfamethoxazole was administered intravenously. Two days later the patient became afebrile; recovery thereafter was uneventful, and the leg ulcers healed after 4 weeks of hospitalization.

Bacteriological studies of P. putrefaciens.
*P. putrefaciens* was isolated from the blood culture bottles (*Brucella* broth with 0.05% sodium polyanethol sulfate; Pfizer EVAC) in the Clinical Microbiology Laboratory. The aerobic bottle subcultures became turbid in 24 h and were plated on blood agar, chocolate agar, and MacConkey agar. A gram-negative motile rod grew on all three plates and was identified by using the API 20E system (Analytab Products, Plainview, N.Y.), which incorporates 23 biochemical tests. The plates were positive for ornithine decarboxylase, citrate utilization, hydrogen sulfide production, gelatinase, cytochrome oxidase, and reduction of nitrate to nitrite and negative for β-galactosidase, arginine dihydrolase, lysine decarboxylase, urease, tryptophan deaminase, indole reaction, Voges-Proskauer reaction, fermentation of glucose, reduction of nitrate to N₂, and oxidation of mannitol, inositol, sorbitol, rhamnose, sucrose, melibiose, amygdalin, and arabinose. Additional tests revealed good growth in nutrient broth containing 6% NaCl and on *Salmonella-Shigella* agar. This, together with lack of acid production from sucrose and arabinose, puts this strain into group 2 of Riley et al. (20). Antibiotic susceptibility was performed with the Kirby-Bauer disk method. It showed sensitivity to ampicillin, carbenicillin, chloramphenicol, kanamycin, gentamicin, tobramycin, tetracycline, and trimethoprim-sulfamethoxazole; the organism was resistant to cephalothin and polymyxin. By the tube dilution technique, it was resistant to 350 U of polymyxin B per ml.

The identity of this organism was confirmed by the Special Bacteriology Unit, Division of Laboratories, New Jersey Department of Health.

*P. putrefaciens* is only rarely isolated in hospital laboratories (8, 29). Its identification is relatively easy: it is the only nonfermentative gram-negative bacillus that produces hydrogen sulfide (in the API 20E system, triple sugar in agar); the colonies form a characteristic reddish-pink or pink water-soluble pigment, and the organism decarboxylates ornithine. Other positive tests are deoxyribonuclease, gelatinase, nitrate reduction, and oxidase (7, 8, 11, 12, 25). It belongs to the group of relatively sensitive *Pseudomonas* species; multiple antibiotic resistance has not been reported (8, 21, 22, 29). Our strain is the first one described with resistance to polymyxin B.

*P. putrefaciens* has been cultured from different human sources (Table 1). Most often it has been found in ear discharge, sputum, skin ulcers, feces, and urine (1, 3, 5, 6, 7, 9, 10, 12, 13, 15, 18, 19, 21–23, 27). In most instances it was regarded as a saprophyte, often in company with other bacteria.

In only a few patients did this organism appear to be of pathogenic importance, causing otitis media in some (4, 7, 10) and in one case a phlegmon at the base of a leg ulcer (4). Its role in a skin ulcer overlying a compound fracture of the tibia (7) or in recurrent otitis externa (12) is less clear (Table 2).

The case presented here resembles very much a patient in a recent report (23). It is of particular interest because of the demonstration that this organism may invade the bloodstream and cause systemic disease. This patient had septicemia

### Table 1. Isolation of *P. putrefaciens* from human sources

<table>
<thead>
<tr>
<th>Source</th>
<th>No. of isolates reported in the following references:</th>
<th>Schmidt et al.*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>1 3 6 7 9 10 12 13 15 18 19 21 22 23 27 28</td>
</tr>
<tr>
<td>Skin infection</td>
<td></td>
<td>1 1 1 3 4 S° 2° 1 1</td>
</tr>
<tr>
<td>Sputum</td>
<td></td>
<td>4 2 3 1 3 1 1</td>
</tr>
<tr>
<td>Ear discharge</td>
<td></td>
<td>1 2 5 2 2 2</td>
</tr>
<tr>
<td>Feces</td>
<td></td>
<td>2 1 1 3 —d 2</td>
</tr>
<tr>
<td>Urine</td>
<td></td>
<td>4 1 1 1 1 5</td>
</tr>
<tr>
<td>Stored blood</td>
<td></td>
<td>1 1 1 2</td>
</tr>
<tr>
<td>Blood (sepsis)</td>
<td></td>
<td>1 1 2</td>
</tr>
<tr>
<td>Spinal fluid</td>
<td></td>
<td>1 1</td>
</tr>
<tr>
<td>Bile</td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>Conjunctiva</td>
<td></td>
<td>1 1</td>
</tr>
<tr>
<td>Pleural fluid</td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>Ascitic fluid</td>
<td></td>
<td>1</td>
</tr>
</tbody>
</table>

* Data of U. Schmidt, R. Kapila, Z. Kaminski, and D. Louria from two additional isolates (one from chronic leg ulcer, one from bile in patient with biliary-colonic fistula).
° Six additional isolates from "miscellaneous infections," probably from skin infections.
°° There were five additional isolates from "infections" which are probably listed under references 6 and 27.
°°° — Unable to obtain exact data.
with fever and leukocytosis, and his blood cultures grew *P. putrefaciens* as the only organism on two occasions 3 days apart. It originated most likely from the infected leg ulcers, although the wound culture revealed three different bacteria. Skin ulcers, especially venous stasis ulcers, have been reported to be colonized or infected with *P. putrefaciens* (1, 3, 4, 7, 10, 19), and they were the port of entry in one septic patient (23). In this case there was no other apparent source from which bloodstream dissemination might have occurred and no evidence for a generalized defect in host defense mechanisms, except possibly for the liver disease (24, 30). Two additional compromised patients with positive blood cultures have just been reported (28). One was a 60-year-old diabetic with gangrene of the foot; the other one was a 90-year-old man with congestive heart failure, prostatic carcinoma, and pneumonia with empyema (which also grew *P. putrefaciens*).

**LITERATURE CITED**


