Fatal Pneumonia Due to *Serratia proteamaculans* subsp. *quinovora*

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*Serratia proteamaculans* subsp. *quinovora* was isolated from several samples (blood cultures, tracheal aspirates, pleural effusion) from a patient with pneumonia. This is the first clinical isolate and the first documented human infection caused by this organism.

*Serratia liquefaciens* has been well known for years in clinical microbiology laboratories, first as *Aerobacter liquefaciens*, then as *Enterobacter liquefaciens*, and in the last 15 years as *S. liquefaciens* (6). Following a DNA hybridization study in 1982, Grimont et al. (7) reported the existence of three groups within the *S. liquefaciens* group. They proposed two new species, *Serratia proteamaculans* and *Serratia grimesii*, the former being divided into two subspecies: *Serratia proteamaculans* subsp. *proteamaculans* and *Serratia proteamaculans* subsp. *quinovora* (8).

*S. liquefaciens* sensu stricto and *S. grimesii* have been isolated from clinical samples, most often without clinical significance (4).

*S. proteamaculans* subsp. *proteamaculans* and *S. proteamaculans* subsp. *quinovora* have been isolated from insects, soil, rodents, and plants (4, 5). We report here the first isolation of *S. proteamaculans* subsp. *quinovora* from a human clinical specimen.

A 43-year-old homeless male alcoholic from the south of France was admitted on 14 June 1990 to an intensive care unit in a Marseille hospital for a large and obstructing abscess of the floor of the mouth following a dental abscess. Treatment with piperacillin (12 g/day) and vancomycin (2 g/day) was started. On 18 June, the patient had a tracheotomy because of his suffocating abscesses. The abscess site was then incised (no samples for culture were taken). In the immediate postoperative period, the patient showed signs of respiratory distress which was perhaps due to the inhalation of pus. On 24 June, he presented with a purulent left pleural effusion that required the insertion of a drain. He also had digestive hemorrhaging because of a peptic ulcer esophagitis. On 28 June, a computed tomography scan revealed a pneumonia in the right lung with a septated right pleural effusion and extension to the right superior mediastinum. On the same day, a gram-negative bacterium was isolated in pure culture and in great quantity from a bronchial aspirate and a pleural effusion sample. This bacterium was identified in our laboratory as *S. proteamaculans*. On 2 July, the same bacterium was isolated from two drains (left and right sides) and from two sets of blood cultures. Approximately 1 liter of pus was drained. The numerous false membranes required the positioning of four thoracic and two mediastinal Delbey drains. *S. proteamaculans* was again isolated from bronchial aspirates on 9 and 12 July. On 13 July, the patient presented with a cholecystitis requiring exploratory laparotomy. At that time, a chronic calcifying pancreatitis was observed. A cholecystectomy was performed. A hepatic biopsy revealed a cirrhosis with steatosis. On the next day, the hemodynamic and respiratory conditions of the patient worsened. On 16 July, *Klebsiella pneumoniae* and *Pseudomonas aeruginosa* were isolated from bronchial aspirates. On the same day, *S. proteamaculans* was again isolated from a central venous catheter (10⁶ CFU/cm of catheter). On July 17, a cervicothoracic computed tomography scan revealed a bilateral pneumonia without mediastinal gathering. On the same day, acute renal failure was observed. Despite renal dialysis, the patient died on 18 July of multiple-organ failure.

After 24 h, a culture of the isolated organism showed smooth colonies on Mueller-Hinton agar (bioMérieux, Marcy-l’Étoile, France). The strain had a biochemical profile number of S 307 563 when tested by the API 20E system (bioMérieux). The 1992 edition of the API Index gives for this profile “excellent identification to the genus *Serratia*,” but the data matrix of the API 20E identification system did not contain the species of the *S. liquefaciens* group. The organism was identified as *S. proteamaculans* subsp. *quinovora* by the TAXIDEN numerical identification program (Intelligence Artificielle Applications, Clapiers, France), which computes the probability products and standardized Lapaline identification score (1). Multiple biochemical tests were subsequently performed, as described earlier (2), to confirm the identification of the organism. Confirmation was confirmed by using carbon source utilization tests as described earlier (3). All biochemical test cultures were incubated for 72 h at 30°C. The organism was positive for catalase, lysine decarboxylase (Moeller), ornithine decarboxylase (Moeller), Voges-Proskauer reaction, DNase, gelatin liquefaction, and Tween 80 esterase but was negative for oxidase, indole, urease, and arginine hydrolysis. The organism utilized the following compounds as sole sources of carbon and energy: N-acetyl-d-glucosamine, L-arabinose, cellobiose, d-lyxose, raffinose, d-sorbitol, sucrose, d-turanose, L-tyranoine, and D-xylose. The organism did not utilize adonitol, i-erythritol, D-glucosamine, D-quinate, L-hamnose, trigonelline, or xylitol.

The MICs of 17 antibiotics for the organism were determined by the agar dilution method, as described by the National Committee for Clinical Laboratory Standards, by using cation-supplemented Mueller-Hinton medium (bioMérieux). The isolate was found to be susceptible to cefotaxime (MIC, 0.025 µg/ml), cefotiam (MIC, 2 µg/ml), ceftri-
axone (MIC, 0.12 μg/ml), piperacillin (MIC, 0.12 μg/ml), imipenem (MIC, 0.12 μg/ml), tobramycin (MIC, 0.25 μg/ml), amikacin (MIC, 0.5 μg/ml), netilmicin (MIC, 0.12 μg/ml), ofloxacin (MIC, 0.12 μg/ml), pefloxacin (MIC, 0.25 μg/ml), and ciprofloxacin (MIC, 0.012 μg/ml). The isolate was resistant to ampicillin (MIC, 64 μg/ml), ampicillin-clavulanic acid (MIC, 32 μg/ml), cefoxitin (MIC, 256 μg/ml), erythromycin (MIC, 64 μg/ml), doxycycline (MIC, 256 μg/ml), and fosfomycin (MIC, 32 μg/ml). Because no other causative agent was found, the pneumonia described here seems to have been caused by the isolated S. proteamaculans strain. No other S. liquefaciens group infection was noted in the same ward or the entire hospital during this period. Given the patient's life-style, this Serratia strain may well have been acquired while he slept on the ground outside.

To our knowledge, this is the first documented human infection caused by S. proteamaculans subsp. quinovora.

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REFERENCES