Isolation of Serratia ficaria from Human Clinical Specimens

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Serratia ficaria was isolated from sputum in a 62-year-old man and from tracheal aspiration fluid in a 62-year-old woman with acute respiratory distress. These strains are the second and third isolations of S. ficaria from human sources. The case reports are presented, together with the laboratory findings and the biochemical activities of the isolates.

Serratia ficaria was described and identified by Grimont et al. in 1976 (6). It was found in figs and in wasps and other insects that pollinate fig trees (6, 7). This new species of the genus Serratia was first isolated from a human clinical specimen by Gill et al. in the United States (3). We now report isolates of S. ficaria from two patients in an emergency care unit.

CASE REPORTS

Case report 1. In January 1982, a 62-year-old man with acute myocardial infarction was sent to the Grand Hornu Hospital. This coal miner had been retired on a pension for several years owing to pneumoconiosis, with a high degree of disablement. He suffered from a productive cough and breathlessness. Moreover, he was still a heavy smoker (50 cigarettes a day). His height and weight were 66 in. (167 cm) and 198 lb. (90 kg), respectively. His blood pressure was 180/100 mmHg.

This patient had been admitted previously, in July 1981 on our service, for an impending infarction, with no sign of necrosis. At the time, chest X-rays showed a known picture of progressive massive fibrosis or complicated pneumoconiosis of coal workers; large localized masses superimposed numerous tiny nodulations scattered throughout both lungs. Pulmonary function tests revealed a significant restrictive-obstructive respiratory disease with a slight obstructive predominate. Resistance to airflow was increased, and the diffusion capacity for carbon monoxide was slightly impaired; vital capacity, maximal breathing capacity, and diffusion capacity for carbon monoxide were lowered to 66, 52, and 79% of normal, respectively, whereas residual volume was increased to 135% of normal. No improvement of these values appeared after inhalation of a sympathomimetic drug (ociprenaline). During the first admission of this patient, several sputum cultures yielded normal pharyngeal microflora. Home treatment included clindamycin (75 mg per day) and isosorbide dinitrate (20 mg per day). As a prevention of infectious complication, he had been given oral doxycycline (200 mg per day), which was stopped on admission. In October 1981, the patient had been vaccinated against influenza. He received no corticosteroids.

On 5 January 1982, this patient was admitted to our emergency care unit because of constritive retrosternal pain, radiating to the left arm, with no relief by oral nitroglycerin. On examination, the patient was afebrile, with a blood pressure of 150/80 mmHg. The heart rate was regular (100/min), and there was no sign of cardiac failure. Cardiopulmonary auscultation and abdominal and neurological examinations were normal. Chest X-rays showed no change compared with the previous films. An electrocardiogram revealed an anterior infarction in the precordial derivations. Laboratory studies showed normal hematological values: red cell count was 5.1 × 1012/mm3, hemoglobin was 15.5 g/dl, hematocrit was 44%, and the leukocyte count was 10,000/mm3 with a normal differential. Chemistry tests were unremarkable except for those for enzymes: serum levels of creatine kinase and lactate dehydrogenase rose to 1,800 and to 1,200 IU/liter, respectively. The MB isoenzyme of creatine kinase rose to 284 IU/liter. Arterial blood gas analysis showed the following: pH, 7.4; pCO2, 38.6 mmHg (5.145.38 Pa); pO2, 68 mmHg (9.064.4 Pa); base excess, 0.1 mmol/liter; buffer base, 47.8 mmol/liter; actual bicarbonate, 23.7 mmol/liter; oxygen saturation of hemoglobin, 93%.

The day after admission, a routine sputum culture yielded a mixed growth of Enterobacter agglomerans (= Erwinia herbicola) and an unidentified gram-negative rod. Sputum Gram stain revealed a mixed flora with a predominance of gram-negative rods. The scoring for leukocytes and epithelial cells was not recorded. Cultures taken for Mycobacterium tuberculosis were negative. Clinical follow-up revealed no aggravation of the respiratory impairment. Further laboratory data and chest radiographs confirmed cardiac infarction and did not suggest any lung infection. Five days later, the patient developed an urinary tract infection due to Escherichia coli. He was given oral amoxicillin (2 g per day). A subclavian catheter culture was sterile. The patient improved and was sent home with the same treatment he had before his admission. At follow-up thereafter, the patient felt no pain and was well. An electrocardiogram showed sequelae of heart infarction.

Case report 2. A few hours after the admission of the above patient, a 62-year-old woman was admitted to the same service with acute respiratory distress. She was asthmatic, diabetic, and a resident in a psychiatric institution. Measurements of pulmonary function demonstrated the presence of increased airway resistance. Chest X-rays were normal. Home treatment included bronchial dilators and doxycycline. Just before admission, she had been given hydrocortisone succinate intravenously (2 × 250 mg). Laboratory investigation on admission revealed the following: red cell count, 5 × 1012/mm3; hemoglobin, 15.7 g/dl; hematocrit, 44.6%; leukocyte count, 14,100/mm3; blood glucose, 216 mg/
périmétrie bronchique. Nous pouvons donc conclure que la culture a permis la détection rapide de ce bactérium.

**RESULTS AND DISCUSSION**

Both strains were cultured on nutrient medium supplemented with 5% (vol/vol) horse blood at 37°C. We noted a very strong odor of the cultures on isolation, similar to that of _Serratia odorifera_ and related organisms reported by Grimont et al. (4, 5). Identification by the method of Edwards and Ewing (2) and additional tests proposed by the tables of Gill et al. (3) allowed us to conclude that both strains were _S. ficaria_.

Table 1 shows biochemical features of our two strains, which were identical. We noted no discrepancy between the results of Grimont et al. (6, 7) and ours, except for acid production from adonitol. On the API 20E, the cultures were identified by the code number 1 007 773, which was not listed in the Analytical Profile Index (Analytab Products) index but placed our strains in the tribe _Klebsiellaceae_. According to P. A. D. Grimont (personal communication), _S. ficaria_ comprises four different O antigens and one H antigen. The strain of patient 1 is O:2 serotype, whereas the American strain of Gill et al. is O:1. The strain of patient 2 was not tested.

Antibiotic susceptibilities were performed by the Kirby-Bauer method (1). Both isolates were susceptible to ampicillin, amoxicillin, cefamandole, co-trimoxazole, carbenicillin, minocycline, doxycycline, kanamycin, gentamicin, tobramycin, and chloramphenicol. They were also resistant to penicillin, cephalothin, and erythromycin and showed a double-zone phenomenon with colistin.

The clinical and biological evolution of our two patients attests to this. As with the case reports of Gill et al. (3), these two _S. ficaria_ strains did not play a pathogenic role, but they were apparently transient contaminants of the upper respiratory tract. However, since this species is rarely isolated from humans, we tend to believe that the two strains had a common origin. Unfortunately, delayed identification limited our investigations. We questioned the patients but did not find any evidence of fig ingestion during the days preceding hospitalization. Hand and respirator cultures were not performed at an appropriate time. Throat cultures were taken from the entire medical and nursing staff to detect a possible carrier, but all cultures were negative. A faster investigation might have uncovered the origin of these strains. We consider it important that _S. ficaria_ should be known to microbiological laboratories to allow its rapid identification.

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