

Septic Shock due to Unusual Pathogens, *Comamonas testosteroni* and *Acinetobacter guillouiae* in an Immune Competent Patient

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Comamonas testosteroni and *Acinetobacter guillouiae* are gram-negative bacilli of low virulence that are widely distributed in nature and normal flora. Despite their common occurrence in environments, they rarely cause infectious disease. We experienced a case of septic shock by *C. testosteroni* and *A. guillouiae*, and isolated them by 16S ribosomal RNA sequencing method from the blood cultures of a previously healthy female during postoperative supportive care. This is the first case of septic shock required ventilator care and continuous renal replacement therapy due to these organisms in Korea.

Key Words: *Acinetobacter*; bacteremia; *Comamonas testosteroni*; shock, septic.

Comamonas testosteroni (previously classified in the *Pseudomonas* genus) is found in a wide range of natural habitats, including soil, plants, and water saprophytes. It can also be isolated from some hospital devices, such as intravenous lines and the reservoir water in the humidifiers of respiratory therapy equipment. *Acinetobacter guillouiae* is a nonfermenting, aerobic, gram-negative bacillus that inhabits the normal flora of the oropharynx, skin, and peritoneum in approximately 25% of healthy individuals. Both organisms demonstrate low virulence and are rarely reported as a cause of sepsis in immunocompetent hosts. However, we encountered a case of septic shock caused by these low-virulence organisms. We identified these organisms from the blood cultures of a previously healthy female who has been recently hospitalized for postoperative supportive care. This is the first case of septic shock caused by *C. testosteroni* and *A. guillouiae* to be reported in Korea.

Case Report

A 42-year-old female patient visited our emergency department because of abdominal pain, fever, and chills. Six days previously, she had undergone right frontotemporal craniotomy at our hospital to remove meningioma and was discharged the

day before visiting the emergency room. During the prior admission period, her vital signs had been stable and she had no neurosurgery-related complications. After discharge, she was admitted to a primary clinic for postoperative care, and intravascular fluids were administered for nutritional support. On the day before visiting our emergency department, she developed severe abdominal pain and chills and then presented

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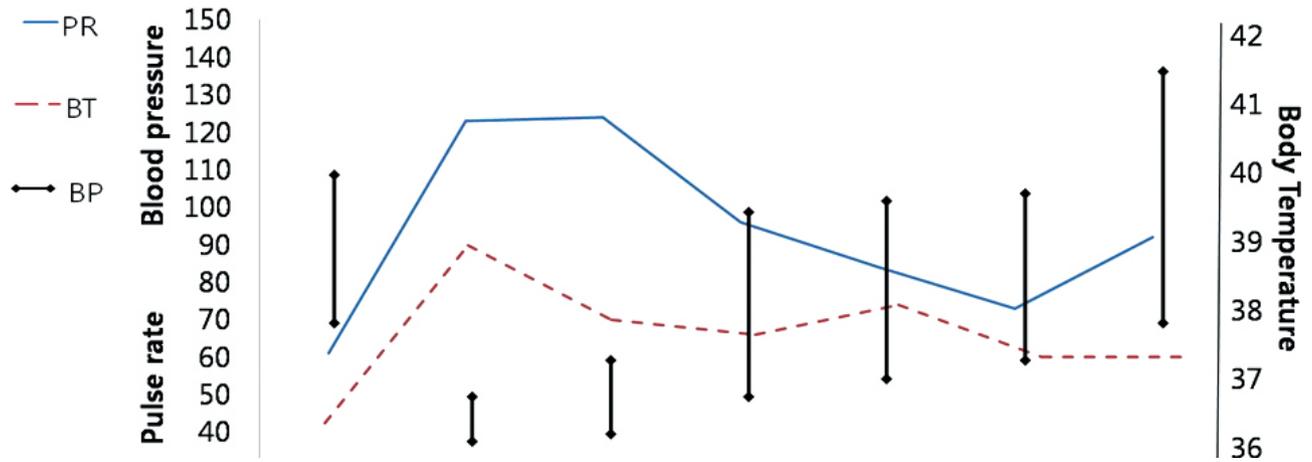
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Table 1. Timetable of clinical course

| Hospital day | POD #4 | ER | HD#1 | #4 | #5 | #9 | #29 |
|-----------------------------------|-----------|------|-------|-------|--------|------|-----------|
| Event | Discharge | | CRRT | Prone | Supine | | Discharge |
| Laboratory findings | | | | | | | |
| WBC ($\times 10^3/\mu\text{L}$) | 8.8 | 32.5 | 63.4 | 46.5 | 46.2 | 17.0 | 5.4 |
| Hb (g/dL) | 9.4 | 8.2 | 6.9 | 7.5 | 8.6 | 7.0 | 10.4 |
| PLT ($\times 10^3/\mu\text{L}$) | 262 | 36 | 36 | 42 | 47 | 75 | 411 |
| pH | 7.39 | 7.12 | 7.20 | 7.41 | 7.58 | 7.42 | |
| pCO ₂ (mmHg) | 41.4 | 25.0 | 29.0 | 37.0 | 25.0 | 43.0 | |
| PaO ₂ (mmHg) | 128.8 | 85.0 | 105.0 | 68.0 | 86.0 | 89.0 | |
| HCO ₃ (mmEq/L) | 25.2 | 8.0 | 11.0 | 24.0 | 23.0 | 28.0 | |
| FiO ₂ | 0.21 | 1.0 | 0.7 | 1.0 | 0.6 | 0.35 | |
| Lactate (mmol/L) | | 11.8 | 12.2 | 3.8 | 2.9 | 1.4 | |
| CRP (mg/dL) | 0.1 | 6.6 | | 19.0 | 18.8 | 10.0 | 0.9 |
| Procalcitonin (ng/mL) | | 61.2 | | | 14.1 | | |
| PT (%) | 94.2 | 43.5 | 22.5 | 60.4 | 75.7 | 87.6 | |
| AST (IU/L) | 13 | 567 | 1,245 | 440 | 283 | 94 | 26 |
| ALT (IU/L) | 11 | 312 | 595 | 359 | 252 | 102 | 15 |
| T-bil (mg/dL) | 0.3 | 0.7 | 2.7 | 8.7 | 13.3 | 9.4 | 1.5 |
| Albumin (g/dL) | 3.2 | 2.6 | 2.5 | 2.9 | 2.6 | 2.4 | 2.8 |
| Creatinine (mg/dL) | 0.5 | 1.6 | 3.9 | 2.0 | 1.9 | 1.8 | 1.6 |

PR: pulse rate; BT: body temperature; BP: blood pressure; POD: postoperative day; ER: emergency room; HD: hospital day; CRRT: continuous renal replacement treatment; WBC: white blood cell; Hb: hemoglobin; PLT: platelet; CRP: c-reactive protein; PT: prothrombin time; AST: aspartate transaminase; ALT: alanine transaminase; T-bil: total bilirubin.

with septic shock next day (Table 1). The initial chest radiography was normal (Fig. 1A). Blood and urine cultures were taken, and piperacillin/tazobactam, levofloxacin, and metronidazole were intravenously administered. A mechanical ventilator and intravascular vasopressors were applied to manage the septic shock. Although chest computed tomography (CT), abdominal CT, and brain CT were performed to

evaluate the infection focus, we could not find any evident cause of the septic shock. Continuous renal replacement therapy (CRRT) was applied due to acute kidney injury with oliguria that was not responsive to furosemide. The next day, her thrombocytopenia progressed and spontaneous hematuria developed, and then intravenous immunoglobulin was covered.

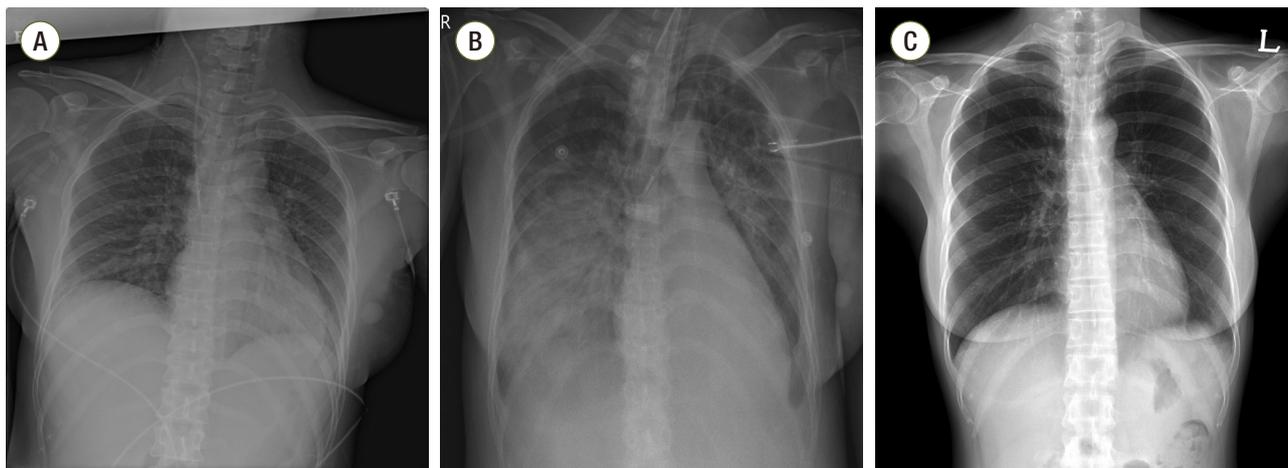


Fig. 1. (A) Chest radiograph on arriving at emergency department shows no evidence of pulmonary infiltration. (B) Chest radiograph on fourth hospital day shows that bilateral dense consolidation and intubated state. (C) On discharge, chest radiograph shows bilateral consolidation is resolved.

At 14 and 18 hours after incubating the blood cultures, positive growth signals were detected in 2 of the culture bottles: one signal was positive for the oxidase test but the other was negative. Meropenem and levofloxacin were administered because both positive growth signals were gram-negative, and the patient had a recent history of hospitalization. On hospital day 3, echocardiography revealed that 53% of the ejection fraction of the left ventricle had no vegetation or regional wall motion abnormalities. The next day, the bilateral dense opacity on chest radiography worsened, and we placed her in the prone position to achieve the proper oxygen level (Fig. 1B). On hospital day 5, one of the positive growth signals of blood culture results confirmed *Acinetobacter lwoffii*, but the other one was indiscernible. We used the 16S ribosomal RNA (rRNA) gene sequencing to identify the organisms. Finally, the results confirmed *Acinetobacter guillouiae* and *Comamonas testosteroni*. Due to the antibiograms of the isolated organisms, we changed the antibiotic regimen to ceftazidime with levofloxacin.

On hospital day 8, the patient complained of hearing loss. Sensorineural hearing loss was diagnosed by an otologist, and high-dose steroid treatment was started (1 mg/kg methylprednisolone). With continuous antibiotics and CRRT, the patient's condition improved and extubation was performed on hospital day 9 (Fig. 1C). Hemodialysis was discontinued on hospital day 20, and she was discharged on hospital day 29. Since discharge, her hearing has improved.

Discussion

C. testosteroni is a widely distributed aerobic gram-negative bacillus that has been identified in various hospital devices. Although *C. testosteroni* has traditionally been considered nonpathogenic, there have been some reports of aggressive clinical manifestations resulting from infection with this microorganism.[1-5] *A. guillouiae* is also a known low-virulence organism[6-8] but was positively detected in blood cultures of our current patient within 14 hours. This is the first case of septic shock caused by *C. testosteroni* and *A. guillouiae* to be reported in Korea.

Based on our patient's recent hospital admission history, treatment with broad spectrum antibiotics (including quinolone and carbapenem) could have been effective. The exact cause of the rapidly deteriorating course in our current case was not confirmed, despite the identification of a low-virulence causative organism and immunocompetent host. However, due to our patient's history and clinical manifestations, we suspected that the source was infusion-related sepsis. Infusate-related bloodstream infection is formally defined as the isolation of the same organism from the infusate and separate percutaneous blood cultures without any other identifiable source of infection.[9] Culture specimens from medical devices and the hospital environment would have been needed to confirm the cause of our patient's septic shock. However, following infusing fluids, the patient's systemic inflammatory response rapidly progressed and we

could not find the infection focus despite evaluating brain, chest, and abdominal CT. Even though *C. testosteroni* and *A. guillouiae* are low-virulence organisms, rapid deterioration might occur if they directly permeate the blood. There is a previous case report of *C. testosteroni* bacteremia-related perforated acute appendicitis in an immunocompetent host. [10,11] Our patient did have abdominal pain, but there was no evidence of bowel perforation or inflammation on abdominal CT. Septic shock from intraabdominal infection could not be ruled out, but follow-up abdominal CT could not find any focus in the abdomen and, except for abdominal pain, other symptoms such as diarrhea, direct tenderness, or ileus did not present.

With advances in nonculture-based microbiology methods,[7,12] a variety of different pathogens that are difficult to culture can now be more readily identified as the cause of disease in an increasing number of immunocompromised hosts. In our present case, we identified the unusual causative pathogens using 16S rRNA sequencing.

Critically ill patients are often exposed to risk factors for hearing loss, including noise, ototoxic medications, sepsis with organ dysfunction, electrolyte abnormalities, dehydration, and malnutrition. Even though furosemide was administered to our patient on hospital day 1, the total dose was only 200 mg and aminoglycoside antibiotics were not administered.[13,14] We could not find any evidence that sepsis or the administered drugs affected sensorineural hearing loss. It has not been confirmed previously if sensorineural hearing loss is a clinical manifestation of septic shock due to unique microorganisms or another independent phenomenon.

This is the first case report of septic shock due to *C. testosteroni* and *A. guillouiae*, with the 16S RNA sequencing method used to identify these causative pathogens. The rapid and progressive clinical manifestation of septic shock due to these microorganisms is indicative of their potential pathogenicity and ability to sometimes cause life-threatening infections.

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