Liver Abscess and *Aeromonas* Bacteremia with Septic Pulmonary Embolism

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**Abstract**

A 68-year-old healthy man was admitted because of liver abscess and *aeromonas* bacteremia accompanied by pulmonary embolism. *Aeromonas sobria* was isolated from blood. Coagulation parameters were normal and a lower limb venogram showed no evidence of thrombosis. Septic embolism originating from the liver abscess was considered as the possible cause of pulmonary embolism. The patient was successfully treated with antibiotics. (Internal Medicine 42: 1047-1049, 2003)

**Key words:** *Aeromonas sobria*, liver abscess, septic pulmonary embolism

**Introduction**

*Aeromonas* infections have been often reported in the compromised hosts with liver cirrhosis and malignancies (1). Bacteremia of *aeromonas* infection takes a fulminant course and is sometimes fatal. The mortality rate for *aeromonas* sepsis is estimated to be 30–50% (2). *Aeromonas* bacteremia in healthy adults has been occasionally reported (1). We report a case of healthy adult with liver abscess and bacteremia complicated with pulmonary embolism due to *Aeromonas sobria*.

**Case Report**

A 68-year-old man noticed general fatigue in June 2000. Later he also noticed epigastralgia and left sided chest back pain. Twenty days later he developed fever and was referred to our hospital. He had no significant past history including liver disease and malignant process. He has been living in Kozu Island as a fisherman, but no recent cuts of the skin or soft tissue infections were noted.

On admission, his temperature was 37.5°C with normal blood pressure. Lungs were clear and the heart was normal. Tenderness over the right abdomen was elicited. Murphy’s sign was not observed. The liver and spleen were not palpable. There were no skin lesions. Laboratory data revealed that WBC count was 7,100/μl, RBC count was 398x10^4/μl, hemoglobin was 12.8 g/dl, hematocrit was 38.2% and a platelet count was 182x10^4/μl. Aspartate aminotransferase (AST) and alanine aminotransferase (ALT) were elevated to 75 IU and 55 IU, respectively. CRP was 14.9 mg/dl and fibrinogen also was increased to 489 mg/dl. Prothrombin time was 13.9 seconds. INR was 1.25, APTT was 33.3 seconds. D-dimer was elevated to 1.0 g/ml. Arterial blood gas analysis revealed pH 7.519, PaO\(_2\) 57.9 mmHg, PaCO\(_2\) 33.5 mmHg. However, protein C, protein S and anti-cardiolipin antibody were normal. The radiograph of the chest on admission was not revealing (Fig. 1). Lung perfusion scintigram showed multiple perfusion defects in the bilateral lung fields (Fig. 2). The computed tomographic (CT) scan of the abdomen demonstrated a 3x3 cm low density area in the right posterior hepatic segment (Fig. 3). The pelvic CT scan showed no findings of venous thrombosis. Abdominal ultrasonography 5 days after admission revealed a cystic lesion in the right lobe of the liver, which was compatible with liver abscess. A diverticulum was demonstrated at Vater’s papilla (7 mmx7 mm) with upper gastrointestinal endoscopy. Deep vein thrombosis was not observed by venogram of the lower limbs. Intravenous cholangiography showed no abnormal findings suggesting biliary calculus or malignancies.

He had a high fever of up to 41°C after admission (Fig. 4). Intravenous cefmetazole sodium (CMZ 6 g/day) was started for suspected liver abscess. Oxygen saturation level was 88–90% and 2L of oxygen was administered. Heparin (25,000 U/day) was also administered continuously because pulmonary embolism was considered. Later, *Aeromonas sobria* was isolated from blood cultures, which was sensitive to piperacillin sodium, cefazolin sodium, ceftazidime, cefmetazole sodium, imipenem, aztreonam, gentamycin sulfate, amikacin.
Figure 1. Chest radiograph on admission. There were no abnormal finding in the lungs.

Figure 2. Lung perfusion scintgram showed multiple perfusion defects in the bilateral lung fields.

Figure 3. The computed tomographic (CT) scan of the abdomen demonstrated a 3x3 cm low density area in the right posterior hepatic segment.

Figure 4. Clinical course after admission.

Discussion

*Aeromonas* species are gram-negative bacilli, which have been isolated from fresh water, brackish water, tap water, soil, and non-fecal organic materials. The spectrum of *aeromonas* infection in humans includes acute gastroenteritis, cholecystitis, cholangiitis, liver abscess, pneumonia, empyema, meningitis, septic arthritis, osteomyelitis, endocarditis, myonecrosis, and necrotizing fasciitis. Most patients have underlying illnesses, such as chronic liver disease, hematological neoplasms, solid tumors, or acquired immunodeficiency syndrome (1). However, even healthy persons can rarely acquire bacteremic infection due to an *aeromonas* species.

Four species, *Aeromonas hydrophila*, *A. sobria*, *A. caviae*, and *A. salmonicida*, were recognized with antibiotic

sulfate, ofloxacin and trimethoprim-sulfamethoxazole.

The antibiotic was changed to meropenem (MEPM 2 g/day), due to fever and possible breakthrough bacteremia, and was continued for 3 weeks. Six days after admission, the oxygen saturation level recovered to 96% without oxygen administration. His condition gradually improved without drainage of the abscess and he was discharged 1 month later. Nine months later, an abdominal ultrasonography showed disappearance of the abscess in the liver.

KAMANO et al
susceptibility to earlier-generation cephalosporins, gentamicin, tobramycin, trimethoprim-sulfamethoxazole and fluoroquinolones. However, some *aeromonas* species have multi-drug resistant plasmids and produce beta-lactamase (2).

In 1995, 59 *aeromonas* bacteremias were reported in Taiwan. *A. hydrophila, A. veronii* subtype sobria, and *A. caviae* were isolated in 95% of these episodes, with *A. hydrophila* accounting for two-thirds of the cases. Most patients were immunocompromised, because of chronic liver disease and underlying malignancies. Most cases of *aeromonas* bacteremia seen in immunocompromised hosts are frequently caused by drug-resistant bacteria resulting in a high mortality rate of 30 to 50%. Several reports were available on the *aeromonas* bacteremia in healthy persons (1). In 1990, in Israel, Golik et al reported a case of *aeromonas* bacteremia in a non-immunocompromised host. The primary source of *aeromonas* is abdominal and soft tissue infections (3). Immunocompetent cases with *aeromonas* infection can be saved with adequate antibiotic treatment and possible surgical intervention.

We think that the *Aeromonas sobria* bacteremia in the current case was caused by the liver abscess, since he had no cutaneous infections nor other infection sites to cause bacteremia. It is known that *aeromonas* species are found in seafood, and have been isolated from the feces of healthy persons who ate raw seafood contaminated with *aeromonas* species (4). Because the present patient has been living on an island and eating seafood, it is possible that he might have *aeromonas* species in his intestine as one flora. A diverticulum at Vater's papilla was documented with upper gastrointestinal endoscopy, which might be colonized with *aeromonas* and could be the source of the liver abscess. Pulmonary embolism was thought as a possible consequence of septic embolism from liver abscess because there was no evidence of deep venous thrombosis in the CT scan or in the venogram, or hypercoagulable state. Cheng and colleagues reported ten cases of septic pulmonary abscesses and/or emboli among 23 cases with pyogenic liver abscess (5). Hepatic abscesses may be complicated with pulmonary embolism (9).

Six cases of liver abscess accompanied by septic embolism were reported in the past 20 years (Table 1). To date, no case of *Aeromonas sobria* bacteremia with septic pulmonary embolism has been reported.

One choice of treatment for liver abscess, is using antimicrobial agents and drainage. The present patient was successfully treated without drainage of the abscess in order to avoid bleeding because heparin was employed for the pulmonary embolism.

### References