**Vibrio cholerae** non O1, non O139 septicemia in a 19-year-old woman with β-thalassemia/hemoglobin E disease

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**ABSTRACT**

*Vibrio cholerae* bacteremia is rare and mainly reported in liver cirrhotic patients. All cases previously reported in the English language literature occurred in the setting of immune deficiency. We herein reported a case of 19-year-old woman with β-thalassemia/hemoglobin E disease who had developed *V. cholerae* non O1, non O139 septicemia and resulted in mortality. (*J Infect Dis Antimicrob Agents* 2011;29:33-5.)

Note: This case had been presented and discussed in the Interhospital Case Conference on Infectious Disease (ICCID), 18 August 2011, Bangkok, Thailand.

**INTRODUCTION**

Bacteremia due to non-O1 *Vibrio cholerae* is rare and mainly reported in immunocompromised patients, particularly those with hematologic malignancy or cirrhosis.1 The presenting symptoms of serogroup non O1, non O139 *V. cholerae* bacteremia were fever with abdominal pain. Some cases had peritonitis, cellulitis or cerebritis but diarrhea was not necessarily found in the presenting symptoms.2,3 The degrees of infection were ranging from severe bacteremia to fatal disease. The mortality rate was varying in each report, but still high, up to 50 percent3 and most of the patients were immunocompromised or cirrhotic. We reported a case of a 19-year-old woman with β-thalassemia/hemoglobin E disease who developed acute renal failure and severe metabolic acidosis from non O1, non O139 *V. cholerae* septicemia and rapidly deteriorated until death.

**CASE REPORT**

A 19-year-old Thai woman with β-thalassemia/hemoglobin E disease was hospitalized at Rajavithi Hospital, Bangkok, Thailand on June 28, 2011 because of high fever and watery diarrhea for 3 days. She had undergone splenectomy at 4-year-old of age and received iron chelator for 1 year. On examination,
the temperature was 38.7°C, respiratory rate 40 per minute, pulse rate 140 beat per minute, blood pressure 119/82 mmHg and moderate dehydration. Abdomen was soft with generalized tenderness, bowel sounds were hypoactive. She was drowsy, the neck was supple and the remaining examinations were normal. Oxygen saturation was 84 percent before intubation. Laboratory investigations showed as the following; hematocrit 20.1 percent, WBC 21,500/mm³ (N 60%, L 36%, M 4%), and platelet count 379,000/mm³, blood urea nitrogen 102 mg/dL and creatinine 5 mg/dL, respectively. Stool exam revealed a WBC of 30/HPF and no parasites. Liver function tests revealed albumin 4.2 g/dl, globulin 3.9 g/dl, alkaline phosphatase 88 U/L, aspartate aminotransferase 62 U/L, alanine transaminase 70 U/L, total bilirubin 19.2 mg/dl, and direct bilirubin 12.2 mg/dl. Electrolytes revealed sodium 128, potassium 7.7, chloride 86 and bicarbonate 7 mmol/L. An electrocardiogram showed accelerated junction tachycardia with tall peak T. Chest X-ray was normal.

The patient was hospitalized at an intensive care unit (ICU), stool and 2 sets of aerobic blood cultures were sent to the Microbiology Unit. The chosen antimicrobials on the first day were ceftriaxone and ciprofloxacin, with adjusted dosages to creatinine clearance. The patient was intubated with ventilation support due to acute respiratory failure. Hemodialysis was performed immediately due to hyperkalemia and severe metabolic acidosis from acute renal failure. Meropenem was prescribed instead of ceftriaxone by the attending physician, and ciprofloxacin was continued. On the second day of hospitalization, she still had a high fever and developed hypotension. Dopamine and norepinephrine were started to maintain her blood pressure. The second hemodialysis was performed due to unimproved metabolic acidosis, but it was not completely done because of unstable hemodynamic. She still had diarrhea and unconsciousness. Even though the maximal dose of inotropic drugs were prescribed, her vital signs were not stable and developed ventricular arrhythmia and died on the second day of hospitalization. Ten hours later, two specimens of her blood cultures revealed the growth of *V. cholerae* non O1, non O139 which were susceptible to ciprofloxacin.

**DISCUSSION**

The patient had septic shock from non O1, non O139 *V. cholerae* bacteremia. In addition, she also had acute renal failure and severe metabolic acidosis. Although the combined - broad spectrum antimicrobials were promptly administered and hemodialysis was performed, the patient expired rapidly because of the severity of disease.

Most previously reported cases of non O1, non O139 *V. cholerae* bacteremia were found in immunodeficiency or cirrhotic patients. Most cirrhotic patients came to the hospital with presenting sepsis and abdominal pains. The first reported case of β-thalassemia/hemoglobin E disease with non O1, non O139 *V. cholerae* bacteremia in Thailand, reported by Thisyakorn U et al, was a 15-year-old girl who had undergone a splenectomy 3 years before the admission. She presented with peritonitis and bacteremia. She underwent exploratory laparotomy to rule out secondary peritonitis on the first day. Post operative course was uneventful and she became afebrile on the fifth day of hospitalization. In this report, her condition was similar to our patient, but the clinical presentation was not as severe as our patient. However, our patient had no peritonitis, and we did not evaluate for a surgical condition.

Even though our patient received proper antimicrobials as fast as possible, she still had uncontrolled septic shock and died. This may
be from an underlying disease of the patient and/or the complications of acute renal failure which were so severe collectively. She may have had a surgical condition such as bowel perforation as discussed above. Our case was an example of severe non O1, non O139 *V. cholerae* bacteremia in β-thalassemia/hemoglobin E disease not in a cirrhotic patient, which should be considered in differential diagnosis in such patients that come to the hospital with diarrhea and sepsis.

References
