Acute Diarrhea Followed by Fatal Aeromonas Hydrophila Infection of Soft Tissue in a Cirrhotic Patient

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Abstract

*Aeromonas hydrophila* can cause severe infections in immune-compromised patients. We present a 46-year-old cirrhotic man who suffered from acute diarrhea, he was discharged after symptomatic treatment. Ten hours after discharge, he revisited our emergency department (ED) with painful swelling of his left thigh and fever. Severe soft-tissue infections with emergence of hemorrhagic bullae over bilateral lower extremities followed. Even under aggressive treatment, the patient died of overwhelming sepsis 28 hours after his second visit. Cultures of blood and serosanguineous fluid of hemorrhagic bullae revealed *Aeromonas hydrophila*. We would like to emphasize that the physicians should consider *A. hydrophila* infection early in cirrhotic patients who developed soft-tissue infections after acute diarrhea.

Keywords: acute diarrhea, liver cirrhosis, soft-tissue infections, *Aeromonas hydrophila*

Introduction

Acute diarrhea, defined as an increased frequency of defecation (three or more times per day or at least 200 g of stool per day), is a common symptom encountered by emergency physicians. Because most diarrheal illnesses are self-limited or viral, and nearly half last less than 1 day, and workups are usually unnecessary for patients who present within 24 hours after the onset of diarrhea, unless such patients are dehydrated or febrile or have blood or pus in their stool. Rehydration and nonspecific symptomatic therapy are recommended for patients with acute diarrhea. In patients who have evidence of inflammatory diarrhea (fever, tenesmus, dysentery, or fecal leukocytes), microbiologic investigation and empirical antibiotics such as fluoroquinolone are reasonable. However, acute infectious diarrhea in immunocompromised patients might have unpredicted clinical courses for which the clinician may underestimate the severity of diarrhea-related septicemia. Herein,
we report fatal *Aeromonas hydrophila* (*A. hydrophila*) sepsis in a cirrhotic patient after acute diarrhea.

**Case Report**

A 46-year-old male had suffered from five episodes of watery diarrhea during the 6 hours prior to admission had a history of hepatitis B-related liver cirrhosis for 5 years. He also complained of mild abdominal discomfort and nausea. He had eaten raw fish before the symptoms occurred. He denied fever, vomiting, abdominal pain, and lower extremities pain. He also denied any history of trauma to his lower extremities.

His vital signs included temperature of 37.2°C, heart rate of 80 beats/min, blood pressure of 115/64 mmHg, and respiratory rate of 16 breaths/min. Physical examination revealed normal heart and chest examination results, spider angioma on his upper chest wall, his abdomen had mild distension with shifting dullness, and his extremities had no signs of infection or trauma on the skin. Laboratory data revealed white blood count (WBC) at 5900/µl with 68% of segment, hemoglobin at 12.2 g/dl, hematocrit at 32.4 %, platelets at 65000/µl, total bilirubin at 2.5 mg/dl, and prothrombin time at 14.3 seconds (international ratio 1.32). The reserved hepatic function was grade B by modified Child's criteria. Under the impression of acute gastroenteritis, symptomatic treatment was given. After hydration, he was discharged with some medications but no antibiotics.

Six hours after discharge, he revisited to our emergency department (ED) with complaints of spiking fever and progressive pain in his left thigh. Abdominal pain, diarrhea with mucous defaces and tenesmus were also reported. His vital signs included temperature of 39.5°C, heart rate of 115 beats/min, blood pressure of 95/50 mmHg, and respiratory rate 22 of breaths/min. Swelling and erythematic skin over his left thigh were found. Laboratory data revealed white blood count (WBC) at 12900/µl with 92% of segment, hemoglobin at 13.2 g/dl, hematocrit at 33.4 %, platelets at 35000/µl, and creatinine at 2.7 mg/dl. Studies of urine, chest radiography and ascites were negative for infection. He was admitted to our hospital under the impression of soft-tissue infection and amoxicillin-clavulanic acid was administered after blood culture. Unfortunately, progressive dusky blue discoloration of the skin and edematous soft tissue over his left thigh and intractable pain were noted. Ten hours after his second admission to the ED, hemorrhagic bullae emerged initially over his left thigh (Figure), and then expanded rapidly to his left leg and right lower extremity. He also suffered from hematemesis and tarry stool. His vital signs included temperature of 38.1°C, heart rate of 128 beats/min, blood pressure of 80/50 mmHg, and respiratory rate of 25 breaths/min. Cultures of serosanguineous fluid of hemorrhagic bullae were collected. Repeat results of laboratory data showed WBC at 4500/µl with 10% of band form, hemoglobin at 7.2 g/dl, hematocrit at 18.4 %, platelets at 35000/µl, and the coagulation studies revealed a pattern consistent with disseminated intravascular coagulation (DIC). Under the impression of septic shock with DIC and respiratory failure, the patient was admitted to our intensive care unit and antibiotics including ceftiraxone, amikacin and doxycycline were given. Owing to hematemesis, upper
gastrointestinal endoscopy was performed and
endoscopic injection sclerotherapy for
esophageal varices bleeding was done. In
addition, surgical intervention was suggested
under the impression of severe soft-tissue
infections or purpura fulminans, but his family
members refused. Even after aggressive
treatment, the patient died of overwhelming
sepsis and multiple organ failure 28 hours after
of his second visit to the ED. Cultures of his
blood and serosanguineous fluid of
hemorrhagic bullae grew gram-negative
bacillus, which was identified as *A. hydrophila*
with resistance to cefazolin, oxacillin,
ampicillin and amoxicillin-clavulanic acid on
susceptibility testing.

**Discussion**

The pathogenic mechanism for increased risk
of infection in cirrhotic patients has been
suggested to be reduced serum levels of
complements, depressed cell-mediated
immunity, increased serum ion concentration,
and shunting portal-systemic circulation. Increased risk of bacterial infection is generally
considered to be due to an invasive procedure,
increased intestinal translocation, and greater
suppression of the reticuloendothelial system.

In cirrhotic patients with acute diarrhea,
accurate medical history is very important to
identify the possibility of acute infectious
diarrhea that may cause bacterial translocation.
For early detection of acute infectious diarrhea
in cirrhotic patients, Wright’s stain for fecal
leukocytes had been used in the ED with the
sensitivity and specificity are around 0.82 and
0.83, respectively. Our patient presented with
Acute diarrhea for which symptomatic treatment was given initially, then he had suffered from fatal soft-tissue infection caused by *A. hydrophila* with the emergence of hemorrhagic bullae.

*A. hydrophila* is an anaerobic gram-negative bacillus with positive catalase and oxidase reactions. It may be acquired from soil, sewage, and fresh or brackish water and from clinical specimens such as feces, blood, ascites, wound discharge, sputum, and cerebrospinal fluid. In immunocompromised patients, *A. hydrophila* can cause infectious diarrhea, soft-tissue infections, meningitis, endocarditis, peritonitis, cholecystitis, and septicemia. As for soft-tissue infections caused by *A. hydrophila*, two mechanisms have been proposed. The first mechanism postulates that the bacterium invades through trauma and causes primary infection of the soft tissue and then sepsis develops. Second, sepsis is first induced by the pathogen and then develops metastatic lesions in the soft tissue. Picard et al. suggested that *A. hydrophila* was able to invade via the intestinal route. Due to the shunting portal-systemic circulation in cirrhotic patients, *A. hydrophila* can establish bacteremia by gaining access to the systemic vein from the gastrointestinal tract to escape phagocytosis by the Kupffer cells of the hepatic reticuloendothelial system. Our patient had neither infectious signs nor trauma over his left thigh at his first visit to the ED. Possibly, the infection was caused by *A. hydrophila* which invaded through the intestines into the bloodstream and induced septicemia that induced severe soft-tissue infection of the bilateral lower extremities.

In immunocompromised patients, the fatality rate of *A. hydrophila* infection with soft-tissue infections as well as sepsis is high. In this setting, early recognition of the surgical condition and adequate antimicrobial therapy are very important. However, more and more clinical studies have pointed out that the organism has become resistance to antibiotics resulting from inducible [beta]-lactamases, and that first-generation cephalosporins, penicillin, semi-synthetic penicillin and their combinations with [beta]-lactamase inhibitors are not recommended for empiric therapy in Taiwan. Third- and fourth-generation cephalosporins, carbapenems, monobactams, aminoglycosides, fluoroquinolones and tetracycline may be used as empiric treatment. In our case, although aggressive treatment with strong antibiotics after the initial administration of amoxicillin-clavulanic acid, the patient died 28 hours after his second admission to the ED due to overwhelming sepsis.

In conclusion, fatal septicemia can occur after acute diarrhea in cirrhotic patients and early detection is very important. We would like to emphasize that emergency physicians should consider *A. hydrophila* infection early in cirrhotic patients who developed soft-tissue infections after acute diarrhea. Appropriate empiric antimicrobial therapy and prompt surgical evaluation are recommended.

**References**


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肝硬化病人急性腹瀉後發生致命性親水性產氣單胞菌
的軟組織感染

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摘要

親水性產氣單胞菌(Aeromonas hydrophila)為厭氧革蘭氏陰性菌，常會造成免疫不全患者嚴重的感染。本文報告一位 46 歲有肝硬化病史的男性因急性腹瀉求診，經症狀治療後出院。因病患後續發生左下肢疼痛腫脹及發燒，於 6 小時後重返急診。最後由於左下肢軟組織的感染快速發展成雙下肢的嚴重軟組織發炎合併出血性水泡，而病患於重返急診 28 小時後死於嚴重的敗血症，血液及水泡液培養出親水性產氣單胞菌。在此提醒急診醫師，肝硬化病患在急性腹瀉後，若發生軟組織發炎應及早考慮親水性產氣單胞菌感染的可能性。

關鍵字：急性腹瀉，肝硬化，軟組織發炎，親水性產氣單胞菌