Case Report

GMSMJ

Greater Mekong Subregion Medical Journal

# Acute Fever with Painful Swelling of Leg in Alcoholic Cirrhosis Patient

Sarut Lerwiwattaworn, M.D.[1]

[1]School of Medicine, Mae Fah Luang University, Chiang Rai 57100, Thailand

Received 24 October 2024 • Revised 12 November 2024 • Accepted 14 November 2024 • Published online 1 January 2025

**Abstract:**

Patients with liver cirrhosis are susceptible to bacterial infection. The common infection sites are spontaneous bacteremia, urinary tract infection, pneumonia, soft tissue infection and spontaneous bacterial peritonitis. Gram-negative bacteria such as *Escherichia coli and Klebsiella* spp. were the most common microorganisms causing bacteremia in cirrhotic patients. *Vibrio, Aeromonas*, and *Campylobacter* spp. occasionally caused bacteremia in cirrhotic patients, but these individuals are at great risks for invasive infections. The vibrios are found in marine and surface waters. Aeromonads are inhabitants of aquatic ecosystems, worldwide, and are found in fresh and brackish waters. The campylobacters are found in many species of animals, including many domesticated animals. Here we report a case of soft tissue infection from *Aeromonas hydrophila* in liver cirrhosis patient which caused rapidly progressive septicemia and shock.

**Keywords:** Liver cirrhosis, Aeromonas infection, Sepsis, Septic shock

## Introduction

*Aeromonas hydrophila*, is a facultative anaerobic Gram-negative rod present in fresh and brackish waters which causes opportunistic infections, as well as occasional cases of food- and water-borne illness. *A. hydrophila* is an opportunistic pathogen in humans causing extraintestinal and intestinal infections and is an animal pathogen leading to infections in fish, amphibians, and mammals.[1] It causes a broad spectrum of infections (septicemia, meningitis, endocarditis) in humans, often in immunocompromised hosts, and *Aeromonas* spp. have been associated epidemiologically with travelers' diarrhea.[2] The pathogenic potential of Aeromonas is considered multifactorial and the presence of several virulence factors allows these bacteria to adhere, invade, and destroy the host cells, overcoming the immune host response.[3] Wound and soft tissue infection especially at lower extremities is another manifestation of *A. hydrophila*. Patients usually had history of wading through water. *A. hydrophila* can cause severe localized skin infection. It may present as: folliculitis (pustules) and abscesses, impetigo-like rash with crusting and erosions that leads to ecthyma gangrenosum, cellulitis - redness and swelling involving deeper skin and

Corresponding author: Sarut Lerwiwattaworn, M.D.
[1]School of Medicine, Mae Fah Luang University, Chiang Rai 57100, Thailand
E-mail: sarut.ler@mfu.ac.th

GMSMJ 2025; 5 (1): 32-34

necrotizing fasciitis - a rapidly destructive deep soft tissue and muscle infection. *A. hydrophila* skin infection is treated with antibiotics such as tetracyclines, sulfonamides, trimethoprim + sulphamethoxazole or ciprofloxacin. It is usually resistant to penicillin. An Aeromonas abscess is surgically drained. Recovery is followed by scarring with temporary or permanent hair loss.

**Case Presentation**

A 63-year-old man, underlying chronic HBV infection and alcoholic liver cirrhosis, Child Class C, living in Chiang Rai, presented with acute onset of fever, swelling and painful of left leg one day. Two hours prior to admission, he developed increasing pain of left calf. He had no diarrhea or abdominal pain. Without improvement after taking paracetamol, he went to the hospital for medical attention. The previous admission was one week before with diagnosis of hepatic encephalopathy. Physical examination at emergency service: BT 39.7°C, BP 88/42 mmHg, PR 136/min, RR 28/min. oxygen saturation on room air was 91%. General appearance: An old man, acutely ill, febrile with tachypnea, good consciousness and orientation. He had moderate dehydration, no anemia but moderate jaundice. He had normal breath sound, regular heart sound and tachycardia. Abdomen exam was unremarkable with negative fluid thrill. Extremities revealed swelling and tenderness at left calf, no erythematous, no discoloration, no crepitus, Homan's sign positive at left leg, left femoral artery 2+, popliteal artery 2+, dorsalis pedis 2+.

| Leg circumference | |
|---|---|
| Right midthigh | Left midthigh |
| 36 cm. | 35 cm. |
| Right calf | Left calf |
| 30.5 cm. | 35 cm. |

**Problem list:** Acute febrile with painful swelling of left calf.
Differential diagnosis was 1) deep venous thrombosis 2) Cellulitis

**Investigation**

Bedside ultrasound deep vein of left leg revealed no thrombosis on left femoral vein, compressible venous, not seen cobble stone appearance or fluid collection at left leg

Complete blood counts: Hct 41.8%, Hb 14.5 g/dL, WBC count 5.81 x $10^9$/L, Neutrophil 90 %, Lymphocyte 6 %, Monocyte 3.5%, platelets 100 x $10^9$/L

PT 18.4 sec, INR 1.7

BUN 33 mg/dL, Cr 1.47 mg/dL, GFR 50 mL/min/1.73 $m^2$

Electrolyte: Na 129 mmol/L, K 3.6 mmol/L, Cl 92 mmol/L, HCO3 23 mmol/L, Mg 1.7 mg/dL, Phosphorus 1.7 mg/dL

Liver function test: total protein 5.9 g/dL, albumin 2.7 g/dL, globulin 3.2 g/dL, ALT 79 U/L, AST 106 U/L, ALP 308 U/L, total bilirubin 8.4 mg/dL, direct bilirubin 7.1 mg/dL

**Hemoculture:** *Aeromonas hydrophila* x 2 specimens sensitive to ciprofloxacin, levofloxacin and tigecycline

**Final Diagnosis:** Necrotizing fasciitis of left leg caused by *Aeromonas hydrophila* with septicemia and rapidly progressive septic shock

**Clinical course**

Patient received piperacillin/tazobactam 4 gm intravenously every 4 hours and hydration. After 3 hours of admission, patient developed progressive hypotension and profound shock with alteration of

consciousness and peripheral vasoconstriction. Blood pressure was down to 60/40 mmHg, $SpO_2$ 80%. Abdomen: distended, not tender. Neurological: E1V1M1, pupil 2 mm react to light both eyes. Extremities: Hemorrhagic bleb and ecchymosis was developed on left calf (Figure 1). Then, he had cardiac arrest and received CPR without success and was pronounced death after 24 hours of admission.

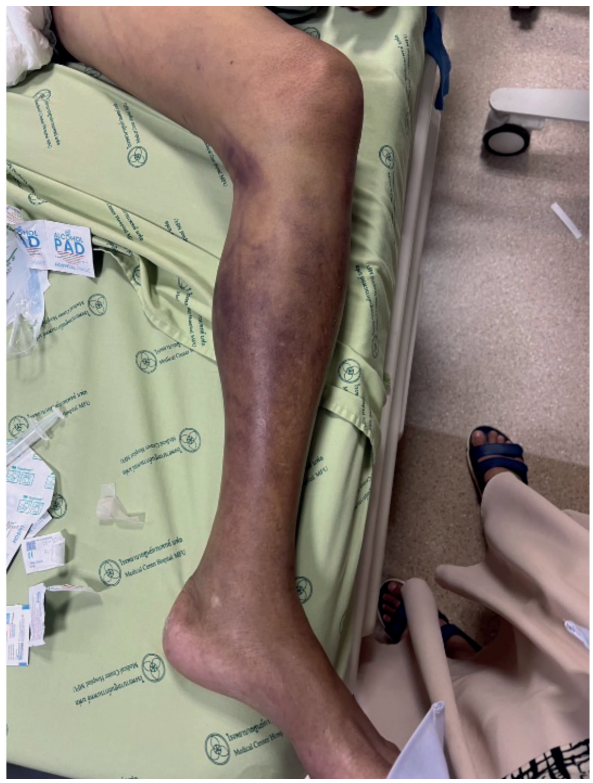

**Figure 1** Left leg after hospitalization

## Discussion

Cirrhosis is associated with several abnormalities in innate and adaptive components of the immune system's response to microbial challenge, leading to a state of acquired immunodeficiency.[4] Cirrhosis can cause sinusoidal fibrosis and capillarization, septal fibrosis with portal-systemic shunts, and Kupffer cell loss.[5] This structural derangement reduces the clearance of endotoxin and bacteria from the blood, leading to bacteremia, metastatic organ infection, and persistent immune system stimulation. A lack of Kupffer cells or of their complement receptors results in uncontrolled bacteremia and increased host death in experimental models.[6] In agreement with these experimental findings, diminished reticuloendothelial system function in cirrhosis has been associated with a greater risk of bacterial infection and lower survival.[7] In cirrhosis, there is coexistence of acquired immunodeficiency and systemic inflammation. The latter results from the persistent stimulation of immune cells and is defined by increased production and enhanced serum levels of pro-inflammatory cytokines and the upregulated expression of cell activation markers.[4] The clinical spectrum of cirrhosis associated immune dysfunction (CAID) varies from a poor response to the bacterial challenge, with

increased susceptibility to bacterial infection accompanied by high mortality, to multi-organ inflammatory damage. The clinical expression of bacteria-dependent events during cirrhosis includes both chronic systemic and organ-specific damage and intercurrent acute insults (i.e. acute-on-chronic).[8,9] It has been demonstrated that the greater the intensity of the cellular and molecular CAID, the greater the risk of severe bacterial infection. This patient presented with acute fever and painful leg swelling. Then, he had rapidly progressive of *Aeromonas hydrophila* septicemia, the possible transmission route might be skin and soft tissue of left leg which resulted in septic shock and multiorgan failure within 24 hours of admission. Although a history of exposure to fresh water is crucial, we did not get the definite history of this. We did not perform Gram stain of the hemorrhagic bleb because the patient was in critical condition. So, early recognition, detection and early intervention especially prompt broad-spectrum antibiotics with hemodynamic resuscitation is mandatory in cirrhosis patients who presented with acute fever and had signs of inflammation in any organs.

**References**


1. Smith J, Fratamico PM, Uhlich G. Molecular mechanisms involved in biofilm formation by food-associated bacteria in Biofilms in the Food and Beverage Industries. A volume in Woodhead Publishing Series in Food Science, Technology and Nutrition. Fratamico PM, Annous BA and Nereus W. Gunther NW, IV. Eds. Woodhead Publishing. 2009, pp 42-98.
2. O'Beirne D, Francis GA. Reducing pathogen risks in MAP-prepared produce in Novel Food Packaging Techniques. Woodhead Publishing Series in Food Science, Technology and Nutrition. Ahvenainen R, ed. Woodhead Publishing. 2003, pp 231-75.
3. Fernández-Bravo A, Figueras MJ. An Update on the Genus Aeromonas: Taxonomy, Epidemiology, and Pathogenicity. Microorganisms. 2020; 8 (1):129.
4. Albillos A, Lario M, Álvarez-Mon M. Cirrhosis-associated immune dysfunction: Distinctive features and clinical relevance. Journal of Hepatology. 2014; 61: 1385-96.
5. Jenne CN, Kubes P. Immune surveillance by the liver. Nat Immunol. 2013; 14: 996–1006.
6. Helmy KY, Katschke Jr KJ, Gorgani NN, Kljavin NM, Elliott JM, Diehl L, et al. CRIg: a macrophage complement receptor required for phagocytosis of circulating pathogens. Cell. 2006; 124: 915-27.
7. Rimola A, Soto R, Bory F, Arroyo V, Piera C, Rodes J. Reticuloendothelial system phagocytic activity in cirrhosis and its relation to bacterial infections and prognosis. Hepatology. 1984; 4: 53-8.
8. Arvaniti V, D'Amico G, Fede G, Manousou P, Tsochatzis E, Pleguezuelo M, et al. Infections in patients with cirrhosis increase mortality four-fold and should be used in determining prognosis. Gastroenterology. 2010; 139: 1246-56.
9. Moreau R, Jalan R, Gines P, Pavesi M, Angeli P, Cordoba J, et alCANONIC Study Investigators of the EASL–CLIF Consortium. Acute-on-chronic liver failure is a distinct syndrome that develops in patients with acute decompensation of cirrhosis. Gastroenterology. 2013; 144: 1426-37.