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Septic Shock Caused by *Aeromonas veronii* in a Patient with Chronic Liver Disease: a Case Report

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Abstract: *Aeromonas* species are gram-negative bacilli found in freshwater or brackish water. This infection is acquired in the community through the consumption of contaminated water and food, as well as through traumatic wounds in contact with contaminated soil or water. A 44-year-old man, with a history of chronic liver disease and significant alcohol consumption, was brought to the Emergency Department (ED) with complaints of nausea and food-related vomiting, associated with jaundice, fever, and night sweats for 5-6 days, as well as epistaxis. He denied other complaints. A few days prior, he had gone fishing and, on physical examination, presented with multiple cuts on both feet. Laboratory tests revealed hematological, renal, and hepatic dysfunction. Microbiological screening showed positive blood and urine cultures for *Aeromonas veronii*. The patient was admitted to the Intensive Care Unit (ICU) with septic shock and multiorgan dysfunction, progressing to refractory septic shock, and eventually passed away. *Aeromonas veronii* bacteremia was associated with gastrointestinal symptoms, such as abdominal pain, presented by the patient. Sepsis caused by this bacterium is more frequent in immunocompromised patients, such as those with chronic liver disease, and is associated with a higher mortality rate.

Keywords: Bacteremia; Septic shock; Chronic liver disease.

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1. Introduction

Aeromonas species are flagellated, Gram-negative bacilli that are oxidase-positive, fermentative, and facultative anaerobes, whose natural habitat is freshwater or brackish water. They have also been isolated from bottled water, public water supplies, including hospital water systems, and soil [1, 2]. These species are not typically found in the gastrointestinal flora of healthy humans, although there are reports of isolating these organisms from fecal samples of healthy, asymptomatic individuals [1]. This infection is commonly acquired through the consumption of contaminated water or food, or through traumatic skin wounds that meet contaminated soil or water [3]. Nosocomial infections are rarer but are presumed to be transient gastrointestinal colonization following procedures such as pancreatic or biliary instrumentation [1].

Microorganisms commonly found alongside *Aeromonas* species include aerobic Gram-negative bacilli such as *Escherichia coli, Klebsiella pneumoniae, Enterobacter species*, and *Pseudomonas species*, as well as Gram-positive cocci such as *Enterococcus* and *Staphylococcus* species [1]. In tropical and subtropical countries, the asymptomatic colonization rate by Aeromonas can reach up to 30%. In Asian countries, there is a high number of Aeromonas infections, which may be linked to increased participation in water-related activities and frequent contamination of uncooked foods [3].

Aeromonas species are primarily associated with gastrointestinal infections, but they can also cause extraintestinal infections such as bacteremia, pneumonia complicated by empyema, arthritis, endocarditis, meningitis, urinary tract and biliary infections, secondary peritonitis, spontaneous bacterial peritonitis, and skin and soft tissue infections [3, 4]. These bacteria are recognized as opportunistic pathogens, causing more severe infections and septic shock in immunocompromised patients, such as those with cirrhosis, hematological malignancies, diabetes mellitus, severe biliary tract diseases, renal failure, and individuals undergoing immunosuppressive treatment, such as chemotherapy or corticosteroids [2, 4, 5, 6].

Infections caused by Aeromonas species are mostly attributed to *A. hydrophila, A. veronii* (sobria variant), and *A. caviae*. Some studies indicate that bacteremia caused by *A. hydrophila* and *A. veronii* (sobria variant) is associated with higher severity and mortality rates compared to infections caused by *A. caviae*. These studies also reported that *A. caviae* is less frequently associated with infections in cirrhotic patients than *A. hydrophila* or *A. veronii* (sobria variant) [4, 7]. Clinically relevant *Aeromonas* species are uniformly resistant to penicillin and ampicillin and are often resistant to first- and second-generation cephalosporins. However, they are invariably susceptible to third-generation cephalosporins, aztreonam, and carbapenems [1, 2, 4].

2. Case Report

A 44-year-old male, self-employed and working as a truck driver, with a medical history of hypertension and chronic liver disease (CLD), as well as a surgical history of herniorrhaphy for a strangulated umbilical hernia in 2014. He had alcohol consumption habits exceeding 35g of alcohol per day and a smoking history of approximately 20 pack-years. He denied regular medication use.

The patient was admitted to the Emergency Department (ED) with complaints of nausea and food-related vomiting (6-7 episodes), associated with jaundice, fever, and night sweats for approximately 5-6 days. He also reported epistaxis but denied abdominal pain, diarrhea, dark urine, pale stools, ingestion of unpasteurized dairy products, consumption of well water or other potentially contaminated sources, and contact with animals, despite living in a rural area. He had no other complaints related to other systems. The patient mentioned that he had been fishing a few days prior and sustained multiple cuts on his feet and legs.

Upon admission, the patient was conscious and cooperative, although disoriented, with asterixis (flapping tremor), febrile, and eupneic on room air, without signs of respiratory distress, with an oxygen saturation of 97% on room air. Lung auscultation was unremarkable. He presented with hypotension and tachycardia, without abnormalities on cardiac auscultation and no peripheral edema. The patient was jaundiced, and inspection of the lower limbs revealed multiple healing abrasions and petechial lesions on his legs. His abdomen was tympanic on percussion, soft, and depressible on palpation, with mild hepatomegaly. He was catheterized, with dark urine output. During observation in the ED, the patient experienced an episode of epistaxis.

Laboratory tests showed thrombocytopenia, elevated INR, acute kidney injury, mixed hyperbilirubinemia, liver enzyme alterations consistent with a cytolytic pattern, elevated CK, and hyperammonemia (Table 1). Arterial blood gas analysis on room air showed compensated metabolic acidosis with hyperlactatemia.

Table 1. Blood tests at admission showing abnormalities relative to reference values.

	Results	**Reference Values**
Hematology		
Platelets	25 10^9/L	150 – 400 x10^9/L
Prothrombin Time	29,20seg	9,0 – 13,0seg

Prothrombin Activity	27,0%	70,0 – 120,0%
INR	2,90	
Activated Partial Thromboplastin Time (aPTT)	50,8seg	24,7 – 39,0seg
aPTT Ratio	1,58	
Factor V	22,5%	62 – 139%
Fibrinogen	1,2g/L	2,4 – 5,0g/L
Biochemistry		
Creatinine	2.0 mg/dL	0.8 – 1.2 mg/dL
Total Bilirubin	10.39 mg/dL	0.30 – 1.20 mg/dL
Direct Bilirubin	4.65 mg/dL	
Indirect Bilirubin	5.74 mg/dL	
Alkaline Phosphatase	270 IU/L	30 – 120 IU/L
Gamma-Glutamyl Transferase	73 IU/L	0 – 55 IU/L
Aspartate Aminotransferase (AST)	161 IU/L	0 – 50 IU/L
Alanine Aminotransferase (ALT)	61 IU/L	0 – 50 IU/L
Ammonia	127.0 μmol/L	16.0 – 53.0 μmol/L
Creatine Kinase (CK)	9639 IU/L	0 – 171 IU/L
CK-MB	139.0 IU/L	<24 IU/L
Myoglobin	11377.9 ng/mL	<105.7 ng/mL

Based on the analytical alterations of hyperbilirubinemia and hepatic cytolysis, associated with hepatomegaly identified in the physical examination, a thoraco-abdominopelvic computed tomography (CT) scan was performed. The exam revealed the presence of consolidated foci, nodules, ground-glass areas, hepatosplenomegaly, and mesenteric hypertension, without other significant alterations.

Given the neurological, cardiovascular, hepatic, renal, and hematological impairments presented by the patient, admission to the Intensive Care Unit (ICU) was suggested and subsequently confirmed. Initially, a probable case of Acute on Chronic Liver Failure (ACLF) was considered, resulting in grade 2 hepatic encephalopathy (West-Haven classification), severe thrombocytopenia with signs of hemorrhagic diathesis, and hepatorenal syndrome (HRS), with infection being identified as the probable triggering factor. From this point, blood and urine cultures were collected, and empirical antibiotic therapy with Ceftriaxone and Azithromycin was initiated.

As part of the differential diagnosis, Weil's syndrome was considered, as thrombocytopenia, acute kidney injury, hyperbilirubinemia, and the cytolysis pattern are also found in this syndrome [8, 9]. Imaging-wise, the presence of consolidated foci, nodules, and ground-glass areas aligns with what is observed in patients diagnosed with Weil's syndrome. However, a negative Weil-Felix reaction excluded this diagnosis.

Leptospirosis was another differential diagnosis considered, due to the clinical presentation consistent with the 2nd phase (immune phase) of the infection, characterized by fever, hepatic alterations with hyperbilirubinemia and hepatic cytolysis, acute kidney injury in the context of HRS, and rhabdomyolysis. Leptospira transmission to humans can occur directly through contact with infected animals, or indirectly through water or soil contaminated by infected urine. However, the patient had no history of risk contacts that could suggest this infection, and the immunological tests were negative for leptospirosis [8]. Several studies show that most patients with leptospirosis have a history of CLD, which makes them immunocompromised and, therefore, more susceptible to this type of

infection. Moreover, in patients infected with Leptospira, the presence of cirrhosis, hepatorenal involvement, and the need for artificial ventilation are predictors of poor prognosis [10].

The initially collected blood and urine cultures were positive for *Aeromonas veronii* (Table 2), with antimicrobial sensitivity testing (AST) revealing bacterial resistance to ceftazidime and sensitivity to ciprofloxacin and trimethoprim/sulfamethoxazole. Antibiotic therapy was then directed to levofloxacin.

Table 2. Altered immunological and microbiological test results.

	Results
Imunology	
Weil-Felix Reaction	
Weil-Felix – OX2 antigen	Positive with a titer of 1/80
Weil-Felix – OX19 antigen	Negative
Weil-Felix – OXK antigen	Negative
Total Leptospira antibodies	Non-reactive
Leptospira IgG antibodies	Non-reactive
Leptospira IgM antibodies	Non-reactive
Microbiology	
Blood culture (1st and 2nd sample)	*Aeromonas veronii*
Urine culture	*Aeromonas veronii*

Despite fluid resuscitation and vasopressor support, the patient remained in metabolic acidosis with hyperlactatemia, which led to the initiation of renal replacement therapy, resulting in a slight improvement in acidosis. However, there was a worsening of hypoxemia, with a progressive increase in respiratory effort, requiring orotracheal intubation and invasive mechanical ventilation. The clinical condition worsened, characterized by shock, necessitating increased vasopressor doses, including norepinephrine and terlipressin. Evidence of acute liver failure (ALF) with coagulation abnormalities was observed, and, in this context, hemorrhage was noted through the nasogastric tube. A thromboelastogram was performed, and transfusion of a pool of platelets, 1 g of fibrinogen, 1 unit of fresh plasma, and 1 g of tranexamic acid was initiated, but without clinical improvement. The patient, with ALF and septic shock refractory to all instituted measures, ultimately passed away.

3. Discussion

The most likely diagnosis for the clinical presentation of the patient is Acute on Chronic Liver Failure (ACLF) – with a Child-Pugh score of 12 and a CLIF-SOFA score of 50 – associated with multi-organ dysfunction (grade 2 encephalopathy, cardiovascular, hepatic, renal, and hematological dysfunction), with bacteremia caused by *Aeromonas veronii* as the probable triggering factor. Indeed, the patient had an epidemiological context for Aeromonas infection, with scattered abrasions on the lower limbs that may have served as an entry point, related to contact with potentially contaminated water while fishing.

Patients with liver cirrhosis are at higher risk of bacteremia and sepsis caused by *Aeromonas* [8], due to several factors, such as the hypoactivity of phagocytic cells in the hepatic reticuloendothelial system, reduced complement production, bacterial translocation (due to increased intestinal permeability), and the flow of bacteria into the systemic circulation through porto-caval shunts [11,12]. Since bacterial infections are a significant

cause of mortality in cirrhotic patients, it is crucial to be aware of the microorganisms that pose particular risks to these patients. Aeromonas should be considered one of these microorganisms in septic patients with chronic liver disease (CLD) and a history of infection or injury related to exposure to lakes or ponds that may be contaminated.

Empirical antibiotic therapy should be initiated promptly in patients showing signs of sepsis, especially in immunocompromised individuals. *Aeromonas* species produce beta-lactamases and are generally resistant to penicillin, ampicillin, amoxicillin, and clavulanic acid, as well as first- and second-generation cephalosporins [13,14,15]. Patients with CLD presenting with skin infections related to contact with potentially contaminated waters should receive antibiotics with appropriate coverage for *Aeromonas*. Knowledge of the resistance profiles of these bacteria is crucial. Third-generation cephalosporins, fluoroquinolones, and trimethoprim/sulfamethoxazole have been shown to be the most effective antibiotics and should, therefore, be considered the first choice for empirical therapy [13, 15].

A detailed medical history is fundamental for considering the group of microorganisms responsible for the clinical scenario, which helps in choosing the most appropriate empirical therapy. The association between skin lesions and contact with aquatic environments, especially freshwater, should raise a high level of clinical suspicion for *Aeromonas* infection, as in this patient's case. The collection of blood cultures is crucial due to the increased risk of bacteremia and sepsis in CLD patients, allowing for early identification of the infectious agent and timely adjustment of antibiotic therapy.

Recent studies suggest that the main challenges in managing bacteremia in cirrhotic patients are the lack of validated criteria to predict morbidity and mortality, as classical sepsis criteria have proven imprecise in these patients. The CLIF-SOFA and SOFA scores have better predicted mortality at 7 and 30 days after positive blood cultures compared to the MELD score [16, 17].

4. Conclusion

The collection of a comprehensive medical history is essential in all fields of medicine. However, in immunocompromised patients with an increased risk of infection, a detailed clinical history is particularly important to raise clinical suspicion of potential diagnoses. This can lead to early treatment and a significantly reduced morbidity and mortality rate in these patients.

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