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VIBRIO ALBENSIS BACTERAEMIA IN A DECOMPENSATED CIRRHOTIC PATIENT WITH AN ATYPICAL TRANSMISSION SOURCE

Thi Thu Huong Dinh[1], Quoc Phuong Nguyen[2], Thi Thuy Bui[1], Kim Nam Nguyen[1]

[1] National Hospital of Tropical Diseases, Hanoi, Vietnam
[2] Hanoi Medical University, Hanoi, Vietnam

Corresponding author's e-mail: quocphuong@hmu.edu.vn

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ABSTRACT

Introduction: Vibrio albensis is a rare Gram-negative halophilic bacterium belonging to the *Vibrio cholerae* non-O1/non-O139 (NOVC) group, which rarely causes infections in humans. To date, only approximately 5 cases of *V. albensis* infection in humans have been reported in the global medical literature. This bacterium is typically found in marine and brackish water environments (estuaries), with primary transmission routes including exposure to seawater through open wounds or the consumption of raw seafood.

Case description: We report a 49-year-old male with decompensated cirrhosis secondary to alcohol abuse and hepatitis C virus infection who presented with acute liver failure. On day 5 of hospitalization, the patient developed fever (39.5°C) and rigors. Two sets of blood cultures were positive for *V. albensis*. Notably, the patient denied any recent exposure to seawater, brackish water, or raw seafood consumption. The isolate demonstrated full susceptibility to all tested antimicrobial agents. The patient was successfully treated with ertapenem combined with levofloxacin for 7 days and discharged in stable condition after 11 days of hospitalization.

Conclusion: This is a rare case with distinctive features: (1) *V. albensis* bacteraemia without typical environmental exposure, (2) to the best of our knowledge the first reported case of hospital-acquired *V. albensis* infection, and (3) the case supports the hypothesis of opportunistic pathogenic bacteria originating from the gastrointestinal tract in a cirrhotic patient. This case emphasizes the importance of microbiological diagnosis and careful monitoring of infectious complications in immunocompromised patients.

KEYWORDS

Vibrio albensis, bacteraemia, liver cirrhosis, nosocomial infection, non-O1/non-O139 *Vibrio cholerae*

LEARNING POINTS

- *Vibrio albensis* is an extremely rare pathogenic organism in humans, with only 5 previously reported cases worldwide.
- Liver cirrhosis is a significant risk factor for invasive *Vibrio albensis* infection due to compromised immune function.
- Hospital-acquired infection with an unknown source suggests alternative transmission routes that warrant further investigation.

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INTRODUCTION

Vibrio albensis is a Gram-negative, halophilic bacterium belonging to the non-O1/non-O139 *Vibrio cholerae* (NOVC) group, commonly isolated from marine and brackish water environments. In contrast to *V. cholerae* O1 and O139 strains, which cause pandemic cholera, *V. albensis* is rarely documented as a human pathogen[1]. According to a systematic review by Ahmed (2022), only 4 cases of *V. albensis* infection in humans have been published globally, all representing community-acquired infections with clinical manifestations including bacteraemia, urinary tract infection, gastroenteritis, and endophthalmitis, associated with exposure to seawater or consumption of canned seafood[1,2].

Cirrhotic patients have a substantially elevated infection risk due to multiple immune deficiencies, including impaired macrophage function, diminished complement activity, and bacterial translocation from the gastrointestinal tract[3]. Other Vibrio species, such as *V. vulnificus* and *V. alginolyticus*, are well-documented in causing high mortality rates in cirrhotic patients, with mortality rates reaching 50% in primary bacteraemia[4,5].

We report a case of *V. albensis* bacteraemia in a decompensated cirrhotic patient that occurred on day 5 after hospitalization without any documented history of seawater or fresh seafood consumption. This case raises important questions about the transmission routes and opportunistic pathogenic mechanisms of this bacterium in cirrhotic and immunocompromised patients.

CASE DESCRIPTION

A 49-year-old male with a 2-year history of cirrhosis secondary to alcohol abuse and hepatitis C virus (HCV) infection was referred to our hospital. The patient had previously received hepatitis C treatment with sustained virological response (HCV ribonucleic acid negative). The patient explicitly denied any recent exposure to seawater, brackish water, or consumption of raw seafood.

Approximately 10 days before hospitalization, the patient developed progressive fatigue, anorexia, abdominal distention, bilateral lower extremity oedema, and dark urine without fever. He was admitted to our hospital with a diagnosis of decompensated cirrhosis and acute liver failure.

On physical examination at admission, the patient was alert (Glasgow Coma Scale score: 15), with icteric skin and sclera. Bilateral lower extremity oedema was present without any petechiae or purpura. Vital signs were: heart rate 80 BPM, blood pressure 110/70 mmHg, oxygen saturation 98% on room air. Abdominal examination revealed mild distention with hepatomegaly and splenomegaly, without collateral circulation. Cardiovascular and pulmonary examinations were unremarkable.

Initial laboratory investigations revealed: white blood cell count 5.5 $\times 10^9$/l, haemoglobin 110 g/l, mean corpuscular volume 114 fl, platelet count 101 $\times 10^9$/l, aspartate aminotransferase (AST) 80 U/l, alanine aminotransferase (ALT) 40 U/l, gamma-glutamyl transferase (GGT) 58 U/l, alkaline phosphatase 141 U/l, creatinine 86 µmol/l, albumin 22 g/l, total bilirubin 66 µmol/l, indirect bilirubin 26 µmol/l, prothrombin time 32%, alpha-fetoprotein 7 ng/ml. Abdominal ultrasonography demonstrated cirrhotic liver morphology, splenomegaly, thickened gallbladder wall, and minimal ascites. Transient elastography (Fibroscan) revealed S3F4 fibrosis stage. Upper endoscopy showed grade II esophageal varices, gastric mucosal inflammation, and bile reflux. The chest radiograph was unremarkable.

The patient was diagnosed with decompensated cirrhosis and acute liver failure. Initial management included: furosemide 40 mg/day combined with spironolactone 150 mg/day, albumin infusion, propranolol 40 mg/day, and thiamine (Vitamin B1) 100 mg/day.

On hospital day 5, the patient developed high-grade fever (39.5°C) with rigors, without cough, dyspnoea, abdominal pain, or gastrointestinal symptoms. Infection workup revealed: C-reactive protein 26.5 mg/l (elevated), white blood cell count 14 $\times 10^9$/l (elevated). The dengue NS1 rapid test and the influenza rapid test were negative. Chest radiography showed bilateral basal pneumonia. Two sets of blood cultures were obtained, and empirical antibiotic therapy was initiated with ertapenem 1 g/day and levofloxacin 500 mg/day.

After 4 days of incubation, both blood culture bottles yielded bacterial growth, identified as *Vibrio albensis* by matrix-assisted laser desorption ionization–time-of-flight mass spectrometry (MALDI-TOF-MS; Bruker, Germany). Antibiotic susceptibility testing demonstrated full susceptibility to all tested antimicrobial agents, including piperacillin-tazobactam, amoxicillin-clavulanate, amikacin, ampicillin, ceftazidime, ciprofloxacin, cefotaxime, cefepime, gentamicin, imipenem, meropenem, and trimethoprim-sulfamethoxazole.

The patient demonstrated marked clinical improvement within 48 hours of antimicrobial therapy, with defervescence and normalization of white blood cell count (6.5 $\times 10^9$/l). Antibiotic therapy was continued for 7 days. The patient was discharged after 11 days of hospitalization in stable condition, afebrile with improved jaundice. Discharge laboratory values: AST 66 U/l, ALT 36 U/l, GGT 50 U/l, albumin 23 g/l, total bilirubin 46 µmol/l, indirect bilirubin 21 µmol/l, prothrombin time 27%. The patient was counselled on continued outpatient management of chronic liver disease at a local facility.

DISCUSSION

Vibrio albensis is a rare bacterium belonging to the NOVC group, typically inhabiting marine and brackish water environments (estuaries). According to the systematic review by Ahmed and colleagues (2022), only 4 cases of *V. albensis* infection have been reported in the medical literature, in patients aged 27–60 years, predominantly males, and all cases were community-acquired. Clinical

manifestations of *V. albensis* infection include bacteraemia, endophthalmitis, and urinary tract infection[1]. Our case is distinguished by its hospital-acquired nature, occurring 5 days after hospitalization without a documented history of seawater or seafood exposure. The case opens new perspectives on understanding the pathogenic potential and transmission routes of this rare organism.

The absence of environmental exposure in our patient suggests several hypotheses regarding transmission mechanisms:

1) *Prolonged incubation period:* The patient may have acquired *V. albensis* from the community prior to hospitalization, with symptom manifestation during the hospital stay attributable to prolonged incubation in the setting of severe immunosuppression.
2) *Hospital-acquired transmission:* Possible exposure through contaminated medical equipment, central venous catheter colonization, or contaminated hospital food supply.
3) *Bacterial translocation from the gastrointestinal tract:* The organism may have been present as asymptomatic colonization within the gastrointestinal flora, subsequently causing bacteraemia due to compromised intestinal barrier function and bacterial translocation, mechanisms well-recognized in cirrhotic patients.
4) *An environmental source within the hospital:* Vibrio species may persist in hospital water systems or moist surfaces, although this has not been previously documented for *V. albensis*.

Recent literature suggests that NOVC species, including *V. albensis*, may have broader ecological habitats than previously recognized, with documented cases in non-coastal regions, suggesting alternative bacterial reservoirs (e.g., freshwater, processed foods)[6,7].

According to the World Health Organization and The Centers for Disease Control and Prevention definitions, a nosocomial infection is an infection that occurs 48–72 hours after hospitalization, without prior clinical manifestations, or within the incubation period at hospital admission. Our patient developed symptoms on day 5 of hospitalization, meeting these criteria. To the best of our knowledge, if this is indeed a hospital-acquired infection, it is the first documented case of nosocomial *V. albensis* bacteraemia and the sixth case of *V. albensis* infection in humans worldwide *(Table 1)*.

The clinical presentation of high-grade fever, rigors, and bacteraemia with pulmonary involvement is consistent with previous reports of Vibrio bacteraemia in cirrhotic patients[1]. Modern identification methods, particularly MALDI-TOF-MS, have substantially improved the accuracy and speed of NOVC species identification compared to traditional biochemical methods[8]. This is clinically relevant

Author/year	Age (years)/ sex	Diagnoses	Risk factors	Possible mode of transmission	Specimen	Treatment	Duration of treatment	Outcome
Tendolkar et al., 1990[10]	60/F	Endophthalmitis	Unknown	Unknown	Vitreous fluid	IO injection of chloramphenicol only	Unknown	Reduced vision
Engel et al., 2016[9]	50/M	Septic shock with deep skin infection	COPD, depression, marihuana, and alcohol up use	Ready-made tuna ingestion three days prior presentation	Blood culture and wound (Bullae)	Empirical antibiotics for septic shock (ciprofloxacin, cefotaxime, polymyxin E, tobramycin, and amphotericin B)	Unknown	Died
Araj et al., 2019[6]	27/M	Urinary tract infection	Healthy patient (immunocompetent)	Exposure to brackish well water at home	Urine, household water at home, brackish well water	Ciprofloxacin 7 days	14 days	Recovered
Ahmed et al., 2022[1]	64/F	Bacteraemia and possible pneumonia	Liver cirrhosis, History of endometrial malignancy	Unknown	Blood	Meropenem for 4 days then ertapenem, 14 days total	14 days	Recovered
Stolaj et al., 2023[2]	47/F	Acute gastroenteritis	Borderline seromucinous tumour of the ovary and obesity	Unknown	Stool sample	Ceftriaxone + azithromycin, followed by ceftriaxone monotherapy	14 days	Recovered
Our case, 2026	49/M	Septicaemia, pneumonia	Decompensated cirrhosis Child-Pugh C (due to alcohol + treated hepatitis C)	No history of seawater, brackish water exposure, or seafood consumption	Blood (2 blood culture bottles)	Ertapenem 1g/day + levofloxacin 500 mg/day	7 days	Recovered

Table 1. *Reported cases of Vibrio albensis infection.*

Abbreviations: IO, intraosseous; COPD, chronic obstructive pulmonary disease.

because different Vibrio species have varying pathogenic potential and antimicrobial susceptibility profiles.

In Vietnam, microbiological diagnostic capacity has significantly advanced with the implementation of automated systems and molecular techniques, enabling the detection of rare pathogens such as *V. albensis* as demonstrated in this case.

Currently, there are no standardized treatment guidelines for *V. albensis* infection. Based on previous case reports and recommendations for NOVC infections in general, treatment options include 3rd-generation cephalosporins, fluoroquinolones, tetracyclines, and carbapenems for severe infections, typically for 7–14 days[1,2].

Although our patient had severe cirrhosis (Child-Pugh Class C with albumin 22 g/l, prothrombin time 32%, and total bilirubin 66 µmol/l), favourable outcomes were achieved through early detection of infection, timely initiation of appropriate empirical antimicrobial therapy, absence of septic shock, and full antimicrobial susceptibility of the isolate. The patient became afebrile within 48 hours with normalization of white blood cell count, experienced no complications, and was discharged after 10 days with improved hepatic synthetic function on the background of chronic liver disease. This contrasts sharply with mortality rates exceeding 50% documented for *V. vulnificus* bacteraemia in cirrhotic patients[7,9].

Vietnam has an extensive coastline and a well-developed aquaculture industry; therefore, the risk of infection with Vibrio species from environmental sources is a relevant public health concern. However, this case demonstrates that *V. albensis* can cause life-threatening infection in immunocompromised patients even without documented environmental exposure.

This study has several limitations: (1) molecular testing was not performed to identify bacterial virulence factors, (2) environmental cultures from the hospital environment were not conducted to trace the source of infection, (3) no identification data of Vibrio species in the patient's gastrointestinal tract were obtained, and (4) the exact transmission route remains undetermined. These limitations highlight the need for in-depth research on the ecology, pathogenic mechanisms, and molecular epidemiology of *V. albensis*.

CONCLUSION

We report the first globally documented case of hospital-acquired *Vibrio albensis* bacteraemia in a decompensated cirrhotic patient without documented exposure to saline water environments. This case expands the clinical spectrum of *V. albensis* infection and underscores the importance of atypical Vibrio species as opportunistic pathogens in immunocompromised hosts.

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