

Case Report

Vibrio cholerae non-O1/non-O139 Serotype O27 septicemia in a patient with decompensated chronic liver disease - A Case Report

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A B S T R A C T

Vibrio cholerae serotypes that belong to the non-O1 and non-O139 category, especially O27, are emerging opportunistic pathogens being increasingly associated with severe septicemia, particularly in immunocompromised individuals. Most non-O1/non-O139 serogroups cause sporadic infections, but certain strains of *Vibrio cholerae*, including the O27 serotype, have acquired additional virulence factors, raising concerns about their potential for more invasive disease. We report a case of septic shock due to NOVC (serotype O27) in a patient suffering from decompensated chronic liver disease (DCLD). Despite the efforts of intensive resuscitation, the patient died within 36 hours after admission, reflecting the highly aggressive nature of this infection amongst high-risk individuals. As patients with DCLD are particularly prone to immune dysfunction and increased gut permeability, in addition to increased susceptibility to bloodstream infection, they may be particularly vulnerable. Since there is limited information on *Vibrio cholerae* O27, this study will describe the clinical manifestation, associated risk factors, disease trajectory, and patient outcomes. We also review existing, but sparse, literature on the epidemiology and emerging therapeutic considerations of this infection and emphasize the role of early recognition and identification as well as the application of targeted management strategies. Expanding research on *Vibrio cholerae* O27 serotype is crucial to understanding its true public health significance and improving clinical outcomes.

Keywords: *Vibrio Cholerae*, Bacteremia, Liver Cirrhosis, Virulence Factors, Septic Shock

Introduction

Vibrio cholerae is an aerobic, Gram-negative, curved rod-shaped bacillus found in marine, estuarine, and brackish water. It is a member of the Vibrionaceae family that causes cholera. Although *Vibrio cholerae* has over 200 serogroups, only the O1/O139 serogroups are linked to epidemic outbreaks, with the Classical as well as El Tor biotypes differing in epidemiological traits.¹ While *Vibrio cholerae* is best known for causing cholera outbreaks, non-epidemic strains have emerged as important pathogens in diverse clinical settings. Unlike their toxigenic counterparts, these strains are often associated with invasive infections such as septicemia, wound infection, and gastrointestinal disease.^{2,3,4} Of these, the O27 serotype has featured more and more in rare cases of severe illness, raising questions about its virulence potential. The ability of certain non-O1/non-O139 strains to acquire important virulence factors, including cholera toxin and toxin-coregulated pilus, further complicates their clinical impact.^{5,6,7} Given the increasing prevalence of non-O1/non-O139 infections in immunocompromised populations, a deeper understanding of their pathogenic mechanisms, antibiotic resistance patterns, and epidemiological significance is essential for improving diagnosis and management.^{2,4,8}

Case Summary

A 39-year-old male had presented to our hospital's emergency department, accompanied by a one-day history of abdominal distension, abdominal pain, and dyspnea (grade I to III) on and off. Patient was apparently normal before the day of admission, after which he developed abdominal distension, which was sudden in onset and was associated with constant abdominal pain. Patient reported decreased urine output, non-passage of stools, nausea, and fever. He had developed giddiness and slurring of speech on the day of presentation. Patient also complained of swelling of legs for the past two months, which was insidious in onset and gradually progressive.

He was a known case of decompensated chronic liver disease, previously treated six months ago, with a history of jaundice. Four months prior, he had also received Ayurvedic treatment, though specific details were unavailable. The patient gave a past surgical history of EVL (esophageal variceal ligation) and an orthopedic spinal plating. Personal history elicited from the patient revealed that he had been regularly consuming alcohol for the past 20 years but had abstained for the past two months. He was a non-smoker, on a mixed diet, with normal bowel and bladder

habits. There was no history of consumption of seafood or contaminated water exposure. However, the patient resided in Thaiyur, a coastal area in Chennai.

On examination, the patient was conscious, oriented, and alert with pallor, icterus, pedal edema, and dehydration. There were no signs of cyanosis and lymphadenopathy. On admission, his vitals were PR - 110 bpm, BP - 130/80 mmHg, RR - 28 breaths/minute, T - 37°C, SPO₂ - 90% on room air, CBG - 82 mg/dl, CRT <3 sec, and pain score - 2/10. Systemic cardiovascular examination revealed sinus tachycardia with normal heart sounds (S1S2+) and absent murmurs. The JVP (jugular venous pressure) was not elevated. Lungs were clear, had equal bilateral air entry, and had no added sounds, with SpO₂ improving to 99% on 2L O₂. Abdominal examination revealed abdominal distension with tenderness over the right lumbar, right hypochondriac, left hypochondriac, and epigastric regions with bowel sounds present. Neurological assessment showed no focal deficits, with a Glasgow Coma Scale score of 15/15, E4V5M6.

Upon arrival at the emergency room, the patient was initially managed with IVF-NS 500 ml, IV dextrose 25% 50 ml, Inj. Pantoprazole 40 mg IV, and Inj. Emeset 4 mg IV. A Ryle's tube was inserted for gastric decompression, and a Foley's catheter was placed for urine output monitoring. The patient was kept NPO with Ryle's tube in situ.

Routine investigations were done as per protocol. A complete blood count showed anemia with an increased percentage of neutrophils and thrombocytopenia [TLC - 8400 cells/mm³, RBC - $3.22 \times 10^{12}/L$, Hb - 10.8 g/dL, MCV - 101.5 fL, MCH - 33.5 pg, MCHC - 33.1 g/dL, RDW - 16.6%, platelet count - 45,000 cells/mm³, Neutrophils (N) - 86.9%, Eosinophils (E) - 0.6%, Basophils (B) - 0.1%, Lymphocytes (L) - 4.7%, Monocytes (M) - 7.7%, serum electrolytes showed reduced bicarbonate indicating metabolic acidosis [Na⁺ - 134 mmol/L, K⁺ - 4.4 mmol/L, Cl⁻ - 101 mmol/L, HCO₃⁻ - 17.1 mmol/L], LFT was abnormal with reduced albumin and increased bilirubin [Total protein - 6.1g/dl, albumin - 2.6g/dl, globulin - 4.1g/dl, A/G ratio - 0.5, T. Bilirubin - 9.6 mg/dl, D. Bilirubin - 6.5 mg/dl, AST - 171 U/L, ALT - 60 U/L, ALP - 49 U/L, GGT - 40U/L, S. amylase - 56U/L, S. lipase - 54U/L], RFT showed an elevated serum creatinine (S. Cr - 1.91mg/dL, BUN - 14mg/dL), coagulation profile [PT - 51.1 sec, APTT - 11.7 sec, INR - 4.97], Sepsis markers [CRP - 75.75mg/L (reactive), Procalcitonin - >50 ng/mL (positive), LDH - 227 U/L, ESR - 11 mm/hr], ABG [PH - 7.21, HCO₃ - 17.1 mmol/L, Lactate - 8.7 moml/L, pO₂ - 46.8 mmHg, pCO₂ - 43.9 mm Hg] were performed. USG of the whole abdomen showed grade I-II fatty liver with ascites and B/L pleural effusion, R>L. ECG showed normal sinus rhythm with sinus tachycardia, and echo revealed normal

LV function with EF 58% and trivial TR. HIV, HCV, and HBsAg were non-reactive. Urine culture and ascitic fluid culture showed no growth.

Elevated procalcitonin level was indicative of septic shock. The patient deteriorated rapidly, requiring ICU transfer and intensive interventions, including IV crystalloids, IV sodium bicarbonate to correct his severe metabolic acidosis and broad-spectrum IV antibiotics (Inj. Piperacillin-Tazobactam 4.5g IV, Inj. Vancomycin 1g IV) in view of elevated sepsis markers. He was initially provided oxygen via a Non-Rebreather Mask (NRBM), but due to worsening respiratory distress, he was escalated to Non-Invasive Ventilation (NIV). As respiratory failure progressed and lactic acidosis persisted, he was intubated under ionotropic support (noradrenaline + vasopressin) and placed on mechanical ventilation [pressure support mode, FiO₂ - 60%, PEEP - 5 cm H₂O, RR - 25 breaths/minute, PC above PEEP 20 cm H₂O]. Post intubation vitals were BP - 110/70 mm Hg, PR - 113 bpm, SPO₂ - 99% under MV, and RR - 25 breaths/minute. With worsening renal function (I/O - 2400 mL/380 mL) and refractory, severe metabolic acidosis, the patient underwent Sustained Low-Efficiency Dialysis (SLED) for metabolic stabilization. Despite intensive treatment, including fluid resuscitation, vasopressors, ventilator support, and SLED, the patient progressed to refractory shock and multiorgan failure leading to cardiac arrest. Advanced cardiac life support (ACLS) was initiated; however, spontaneous circulation (ROSC) was not achieved. Given the lack of response, the patient was declared deceased.

Blood cultures sent to the Microbiology Lab and processed in the Bactec FX 40 machine grew motile, curved, Gram-negative bacilli after 20 hours of admission. The organism was identified as *Vibrio cholerae* using the routine biochemical investigations and VITEK 2 compact automated system, based on its characteristic biochemical and morphological features. Gram staining of the blood culture broth revealed curved Gram-negative bacilli, while hanging drop motility demonstrated darting motility, consistent with *Vibrio* species. On blood agar, the organism displayed transparent colonies with hemodigestion and a greenish tint. Thiosulfate-Citrate-Bile-Sucrose (TCBS) agar was inoculated for confirmation, and the isolate produced characteristic yellow colonies, while MacConkey agar revealed non-lactose-fermenting colonies. Biochemical testing showed the organism was oxidase-positive, catalase-positive, indole-positive, citrate-negative, TSI - A/A, and urease-negative. The strain failed to agglutinate with O1 (Ogawa and Inaba) antisera, suggesting a non-O1 *Vibrio cholerae*. Further identification was confirmed through MALDI-TOF mass spectrometry at Rela Hospital, Chennai, and serotyping at ICMR-NICED, Kolkata, the National Referral Centre for *Vibrio* spp., which confirmed the strain as non-O1/non-O139 *Vibrio cholerae* (Serotype O27). Genetic screening has been

performed by PCR assays of virulence-associated genes like *ctxA* (cholera toxin A subunit), *ctxB* (cholera toxin B subunit), *tcpA* (toxin co-regulated pilus), *rtxA* (repeats in toxin), *hlyA* (hemolysin A), & *TTSS* (type III secretion system), and it was found to be positive for *rtxA* (El Tor type) and *hlyA* and negative for the *ctxA*, *ctxB*, *tcpA*, and *TTSS*. The isolate was checked for antibiotic sensitivity profile against ampicillin (10 µg), tetracycline (30 µg), chloramphenicol (30 µg), sulfamethoxazole-trimethoprim (23.75/1.25 µg), ciprofloxacin (5 µg), nalidixic acid (30 µg), norfloxacin (10 µg), ofloxacin (5 µg), gentamycin (10 µg), streptomycin (10 µg), ceftazidime (30 µg), ceftriaxone (30 µg), cefotaxime (30 µg), azithromycin (10 µg), and doxycycline (30 µg) by the Kirby-Bauer disk diffusion test using commercial disks (Difco, B.D., USA) in accordance with the protocol and breakpoints suggested by the European Committee on Antimicrobial Susceptibility Testing (EUCAST, 2021). All the strains were found to be sensitive towards all the antibiotics, such as ampicillin, chloramphenicol, tetracycline, sulfamethoxazole-trimethoprim, streptomycin, nalidixic acid, ciprofloxacin, norfloxacin, ofloxacin, gentamycin, ceftriaxone, ceftazidime, cefotaxime, azithromycin, and doxycycline.

Discussion

Invasive bloodstream infection caused by non-O1/non-O139 *Vibrio cholerae*, including serotype O27, is uncommon and most often documented in patients with underlying chronic liver disease or other forms of immune compromise. The present case adds to this small body of evidence: a 39-year-old man with decompensated chronic liver disease developed *V. cholerae* O27 bacteremia confirmed by blood culture, MALDI-TOF identification, and reference-laboratory serotyping at ICMR-NICED, Kolkata. Urine and ascitic fluid cultures were sterile, with no alternative urinary or peritoneal source identified. In hospitalized patients with bloodstream infection, catheter-related infection is an important alternative source that should also be considered when intravascular devices are present.⁹

The molecular profile of this isolate is informative. *ctxA* and *ctxB* were absent, indicating that the strain lacked the canonical cholera toxin genes carried by epidemic O1 and O139 strains.^{1,6,7} *tcpA* was likewise absent, suggesting that the isolate did not harbor this major colonization pilus.⁷

In contrast, *rtxA* and *hlyA* were detected by PCR. The *rtx* toxins have been associated with host cytoskeletal disruption and immune modulation, while *hlyA* hemolysin contributes to host cell injury and inflammation.^{10,11, 12} Although expression and functional activity were not formally assessed, the presence of these genes in an isolate that produced fulminant bacteremia suggests they may have contributed to cytotoxic injury, tissue damage, and invasive potential in this patient. The absence of the type III secretion system and the classical toxin and colonization

determinants indicates that other virulence-associated factors, including *rtxA* and *hlyA*, may have contributed to the invasive phenotype; however, their expression and functional contribution were not assessed.

Other virulence-associated characteristics described among non-O1/non-O139 *V. cholerae* strains were not evaluated in the present isolate. Published observations in other O27 strains may provide useful context; however, these characteristics cannot be attributed to the present isolate without specific testing.^{2,12,13}

The patient's underlying decompensated chronic liver disease is a key factor in this case. Cirrhosis-associated immune dysfunction impairs neutrophil and complement activity and dampens innate responses, while portal hypertension, altered gut barrier function, and impaired mucosal immunity further increase vulnerability to invasive gram-negative infection.^{14,15} These mechanisms offer a plausible explanation for how an organism lacking classical cholera virulence genes nevertheless established sustained bloodstream infection in this host. The sterile urine and ascitic fluid cultures support a possible intestinal translocation route of entry, although translocation was not definitively demonstrated.¹⁶

Finally, the antimicrobial susceptibility pattern is clinically instructive. The isolate was susceptible in vitro to all 15 agents tested, yet the patient progressed within hours to refractory septic shock and multi-organ failure despite appropriate empirical therapy. This discrepancy underscores the fact that in vitro susceptibility does not, on its own, predict outcome in fulminant *V. cholerae* bacteremia when host defenses are profoundly impaired. Early clinical suspicion, prompt blood-culture sampling, and timely escalation of supportive care therefore remain as important as targeted antimicrobial selection in this setting.

Conclusion

This case demonstrates that non-O1/non-O139 *Vibrio cholerae* O27 can cause fulminant bacteremia in patients with decompensated chronic liver disease despite the absence of classical cholera virulence determinants. Detection of *rtxA* and *hlyA* may contribute to its invasive phenotype, although functional activity was not assessed. Rapid progression to septic shock despite susceptibility to all tested antimicrobials highlights the importance of early recognition, prompt diagnosis, and timely appropriate therapy in high-risk patients.

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