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Case Report

Vibrio cholerae O1 (serovar inaba) as a cause of fatal bacteremia in a patient with relapsed acute lymphoblastic leukemia

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ABSTRACT

Vibrio cholerae O1 is an uncommon cause of bacteremia with only a few cases reported in published literature. We report a rare and fatal case *of dual E. coli* and *V. cholerae* O1 serovar inaba bacteremia in a 29-year-old male patient on chemotherapy for relapsed and refractory B-cell acute lymphoblastic leukemia. He had no associated intestinal symptoms.

1. Introduction

Vibrio cholerae are gram-negative bacteria that are differentiated into O1/O139 and non-O1/non-O139 serogroups depending on their ability to agglutinate with specific antiserum. *V. cholerae* O1 and O139, the causative agents of cholera, are morphologically and biochemically identical to the other non-O1 *V. cholerae*, but are antigenically, epidemiologically and clinically distinct. Non-O1 *V. cholerae* can cause small outbreaks of diarrheal illness related to contaminated seafood.[1] While there are, numerous case reports of bacteremia caused by non-O1 *V. cholerae* in persons with predisposing conditions, most commonly cirrhosis but also nephrotic syndrome, diabetes, hematologic malignancy, gastrectomy, and AIDS/lymphoma there are only a handful of cases of bacteremia caused by *V. cholerae* O1.[2,3]

We report a rare case of *V. cholerae* O1 serovar inaba bacteremia from India in a patient with severe sepsis but no preceding or concomitant intestinal symptoms.

2. Case History

A 29-year-old male was admitted for chemotherapy for relapsed refractory B-cell Acute Lymphoblastic Leukemia (B-ALL) in August 2020. The primary diagnosis of B-ALL was established in January 2014 following which he relapsed in 2015 and since then was on multiple salvage chemotherapy regimens. The patient had history of recent admission to our hospital, for one month duration in April 2020, for intensive chemotherapy.

During the current admission, chemotherapy with bortezumib, rituximab, doxorubicin and dexamethasone was initiated after inserting a PICC (peripherally inserted central catheter) line. He had no urinary catheter. The patient was on a neutropenic diet, no uncooked fruits and vegetables were served to him and only bottled mineral water was used. The patient developed fever on day five of admission with no associated symptoms of diarrhea, dysuria or flank pain. Routine investigations were repeated along with paired

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Table 1: Reported cases of invasive disease caused by V. cholerae O1 [4–10]

No.	Age, sex	Known predisposing factor/s	Clinical presentation	Outcome	Year of publication
1	6 years, Female	Autoimmune disease, achlorhydria	Diarrhea, severe sepsis syndrome	Survived	1977
2	6 days, Male	Neonate	Diarrhea, afebrile, neutrophilia, uremia	Died	1983
3	8 months, Female	None	Diarrhea, febrile, neutrophilia	Survived	1992
4	6 years, Female	Chemotherapy	Meningitis, blood culture negative	Died	1996
5	79 years, Male	Obese, alcoholic, portal hypertension without cirrhosis	No diarrhea, h/o travel Canary Islands, fever	Survived	2000
6	2 days, Male	Neonate	No diarrhea	Died	
7	45 years, Female	None	Diarrhea transiently bloody, afebrile	Died	2001
8	65 years, Female	None	Diarrhea, neutrophilia, renal failure secondary to dehydration	Died of renal failure after 2-3 weeks	
9	70 years, Male	None (past h/o myocardial infarct 40 days back)	Fever, diarrhea	Died	2007
10	47 years, Male	Primary peritonitis with liver cirrhosis	No diarrhea, hematemesis, dizziness, hypotension & tachycardia	Survived	2009
11	1 day, Female	Prematurity (27 weeks), mother had premature rupture of membranes for over 36 h, no vomiting or diarrhea in mother	Severe respiratory distress and septic shock	Unknown, was moved to another hospital and lost to follow up	2010 Analysis of 8 cases (1 reported previously Ref Sr No.3 - 1992)
12	11 days, Male	None	Watery diarrhea, dehydration, septic shock, respiratory distress, leukocytosis, deranged coagulation	Died	
13	3 months, female	Concurrent Campylobacter spp. enteritis	Diarrhea, vomiting, leukocytosis	Survived	
14	7 months, Female	Concurrent Staphylococcus aureus, supraclavicular abscess	Vomiting, diarrhea, fever, leukocytosis	Survived	
15	4 years, Female	None	Vomiting, diarrhea, 4/HPF pus cells in stool	Survived	
16	39 years, Female	Hepatitis B virus-associated end-stage liver disease	Fever, ascites	Survived	
17	75 years, Female	Malignancy	Diarrhea, vomiting, leukocytosis, deranged coagulation, elevated hepatic enzymes, 4/HPF pus cells in stool	Died	
18	29 years, Male	Chemotherapy	No diarrhea	Died	This report

blood cultures and urine cultures. Automated blood culture system was used (BACTEC, BD-USA). The investigations revealed bicytopenia, with a white blood cells of 350/cmm and platelets of 13,000/cmm. He had elevated serum C Reactive Protein (47 mg/dl) and serum procalcitonin (98 ng/ml) levels. In view of febrile neutropenia, treatment with intravenous cefoperazone sulbactam, amikacin and caspofungin was initiated as per institutional protocol. The condition of the patient further deteriorated on the same day with hypotension (blood pressure 86/50 mm Hg), tachycardia (170 beats per minute) and hypoxia. He was shifted to the intensive care unit and supportive treatment initiated. The antimicrobials were escalated to meropenem, teicoplanin & polymyxin B. On day two of fever owing to further deterioration, relatives were explained the need for invasive ventilation, and hemodialysis but they were not willing for the same. Patient had asystole, apnoea and succumbed on the same day.

The blood culture (flagged after patient succumbed) and urine culture reports were available after the patient's death. Patient's paired blood culture sent on day one of fever (Left Brachial & Right PICC), grew *E. coli* & *V. cholerae* with a differential time to positivity (DTP) of one hour. Identification was done by MALDI-TOF (Vitek MS, Biomerieux- France) and susceptibility testing by automated system (VITEK 2). Identification of *V. cholerae* was confirmed by serotyping – *V. cholerae* serovar Inaba at a reference lab (Anti Sera Kit Denka Seiken – Central Research Institute Kasauli). The *V. cholerae* isolate was sensitive to ampicillin, cephalosporins, ciprofloxacin and trimethoprim/sulfamethoxazole. The urine culture grew *E. coli* (10^5 cfu/ml). Antimicrobial susceptibility pattern of *E. coli* isolated from blood and urine was identical. The isolate was sensitive only to amikacin, and carbapenems, and resistant to beta lactam- beta lactamase inhibitor (BL-BLI) combinations, ciprofloxacin and third generation cephalosporins.

3. Discussion

The patient died with dual infection with *E. coli* and *V. cholerae* and the relative contribution of each of these cannot be delineated. The initial empiric therapy, partially covered *E. coli* (amikacin) but was appropriate for the *V. cholerae* isolate. The rapid deterioration despite escalation of antibiotics to meropenem (active against *E. coli*) was probably due to lack of host defenses, very low neutrophil count, damaged mucosal barriers and dual infection. While the *E. coli* sepsis possibly originated from the patient's own endogenous flora of the gut/urine, the source of *V. cholerae* bacteremia (community acquired or hospital acquired infection) cannot be ascertained. The differential time to positivity of one hour rules out contamination of the central line as a cause of the *V. cholerae* bacteremia. While we did not culture the stool of the patient as the patient did not have diarrhea, it is reasonable to assume that colonization of the gut from contaminated water/ food followed by translocation to the blood stream was the pathogenetic mechanism. We also initiated an investigation wherein food, water/environmental surfaces were cultured and practices in the kitchen audited. No source could be identified and there was no other case of *V. cholerae* infection in the hospital.

V. cholerae serogroup O1 is generally regarded as a non-invasive enterotoxigenic organism causing gastroenteritis of various severities. This contrasts with non-O1/non-O139 *V. cholerae*, which can invade the bloodstream causing bacteremia and septicemia. *V. cholerae* O1 rarely causes bacteremia or invasive extraintestinal disease. The limited reported cases of invasive disease caused by *V. cholerae* O1 are summarized in Table 1.[4–10]

Pathogenic mechanisms causing invasion in *V. cholerae* infections are not well recognized. Possible reasons could be pre-existing disruption of mucosal barrier, achlorhydria, simultaneous infection with an invasive pathogen, translocation of viable *V. cholerae* via M cells and hemolysin production. Immunocompromised status and prematurity have also been addressed as risk factors.[4–10] In our case, the patient had leukemia, chemotherapy, damaged mucosal barriers and associated infection with *E. coli* as predisposing factors. The patient did not have any history of travel or any episode of diarrhea prior to current admission.

4. Conclusion

Our case shows that serotype 01 can also cause bacteremia similar to V. cholera serotypes non-01/non-139. We recommend clinicians to remember this, as early diagnosis and treatment can be curative in certain patients.

5. Conflicts of Interest

The authors declare no conflicts of interest.

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