Case Report

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Fatal Non O1/Non O139 *Vibrio cholerae* Bacteraemia without Gastrointestinal Manifestations: A Case Report

Microbiology Section

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ABSTRACT

Non-O1/non-O139 *Vibrio cholerae* (NOVC) are increasingly recognised as a cause of severe invasive infection including bacteraemia especially in people who are elderly or have co-morbidities. NOVC infections often lack gastrointestinal manifestations, and the frequent absence of exposure to well-recognised sources such as seafood and or marine/estuarine water lowers the index of suspicion for *Vibrio cholerae* and delays targeted diagnostic testing. Authors present here a case of NOVC bacteraemia in an 80-year-old female from West Bengal, India. She was brought in with acute respiratory distress and altered sensorium, and was found to be in septic shock at the time of admission. There was no history of diarrhoea, seafood consumption, or recent travel to the sea. Despite prompt initiation of intensive care and empirical broad-spectrum antimicrobial therapy, she rapidly deteriorated and died within 48 hours of admission. Blood cultures grew *Vibrio cholerae* which was confirmed as NOVC serotype O17 by a national reference laboratory. Antimicrobial Susceptibility Testing (AST) demonstrated susceptibility to all relevant antimicrobial agents. This case highlights the potential of NOVC to cause bacteraemia with an aggressive clinical course and lethal potential.

Keywords: Bacteraemia, Immunocompromised, Septic shock

CASE REPORT

An 80-year-old female, resident of West Bengal, India, presented to the emergency department at a tertiary-care hospital in Eastern India with acute-onset shortness of breath and altered sensorium since the morning of the day of admission. Her past medical history was significant for Chronic Obstructive Pulmonary Disease (COPD) and recurrent urinary tract infections. She had previous admissions for lower respiratory tract infections, electrolyte imbalance, and bilateral swelling of the legs two months back. She didn't report any diarrhoea, and there was no history of recent seafood consumption, exposure to marine/estuarine water, or travel to a coastal area.

Upon initial assessment, she exhibited signs of severe respiratory distress and circulatory compromise. The detailed patient profile has been compiled [Table/Fig-1].

Age/Gender	80 years/Female
Resident of	West Bengal, India
Known co-morbidities	Chronic Obstructive Pulmonary Disease (COPD)
Past medical history	Recurrent urinary tract infections with previous admissions for lower respiratory tract infections, electrolyte imbalance, and bilateral swelling of the legs two months back
Chief complaint	Acute-onset shortness of breath and altered sensorium since the morning of the day of admission
Vitals	Temperature: 36.4°C; Blood pressure: 80/60 mmHg; Pulse rate: 116 beats/minute; Respiratory rate: 38 breaths/minute; SpO_2: 73% in room air, Random blood glucose:74 mg/dL
Arterial Blood Gas analysis	pH: 7.463, pO2: 48.7 mmHg, pCO_2: 22.7 mmHg, hypokalaemia (2.0 mmol/L), and elevated lactate (8.5 mmol/L).
General examination	Pallor and bilateral pedal oedema
Respiratory system	Decreased bilateral vesicular breath sounds with crepitations
Cardiovascular examination	S1, S2 heard with no murmur
Neurological examination	GCS score of 8/15 (E2V2M4)
Abdominal examination	Non tender, distended abdomen
Chest radiography	Haziness in the right middle and lower zones
Echocardiography	Atrial fibrillation with rapid ventricular response; Ejection fraction: 60%
Abdominal ultrasonography	Moderate amount of free fluid was noted in the abdominal cavity → Ascites
Outcome	Death after 48 hours of hospitalisation

[Table/Fig-1]: Clinical profile and baseline characteristics of the patient at presentation.
*GCS: Glasgow coma scale

Differential diagnoses considered included severe community-acquired pneumonia, acute exacerbation of COPD with sepsis, cardiogenic shock, and septicaemia. Emergency endotracheal intubation was performed, and the patient was placed on ventilator support. Empirical antimicrobial therapy was initiated with piperacillin/tazobactam (4.5 g, intravenous, every 8 hours), doxycycline (100 mg, via Ryles tube, every 12 hours) and clindamycin (600 mg, intravenous, every 8 hours). To manage refractory hypotension (BP 80/60 mm Hg), vasopressor support was initiated along with intravenous fluid resuscitation. Other supportive measures included nebulisation with ipratropium plus levosalbutamol (0.5/1.25 mg, TDS), and formoterol plus budesonide (20 µg/0.5 mg, BD). Blood samples for complete blood count, serum biochemistry, and two sets of blood cultures were collected and sent to the laboratory immediately for microbiological testing.

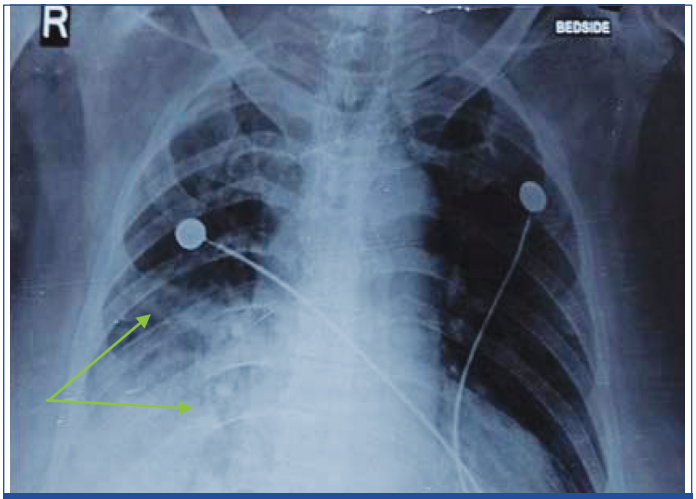
Laboratory investigations were remarkable for leucocytosis with neutrophilic predominance; elevated CRP and procalcitonin, as well as elevated markers of disseminated intravascular coagulation such as D-dimer and Prothrombin Time. Abdominal ultrasonography performed at an outside hospital prior to referral revealed the presence of ascites [Table/Fig-2].

Parameters	Values at admission	Reference range
Haemoglobin (g/dL)	11.0	12.0-15.0
TLC (10^3/µL)	19.85	4.0-10.0
DC-Neutrophils (%)	91	40-80
DC-Lymphocytes (%)	07	25-40
Platelet (10^3/µL)	240	150-450

CRP (mg/dL)	3.65	<0.8
NT-pro BNP (pg/mL)	3777	0.0-450.0
D-dimer (ng/ml)	9770	0.0-500.0
Procalcitonin (ng/mL)	12.7	0.01-0.1
Urea (mg/dL)	21	15.0-36.0
Creatinine (mg/dL)	1.4	0.66-1.25
Serum Potassium (mmol/L)	1.7	3.5-5.1
AST (U/L)	52	15.0-46.0
ALT (U/L)	15	0-35.0
Total Protein (g/dL)	6.5	6.3-8.2
Albumin (g/dL)	1.7	3.4-5.0
PT Test (sec)	28.2	10.7-14.1
PT INR	2.8	0.98-1.2
Glycosylated Hb (%)	4.5	4.0-5.6

[Table/Fig-2]: Significant laboratory findings at admission and on repeat testing. *TLC: Total leucocyte count; DC: Differential count; CRP: C-reactive protein; NT-proBNP: N-terminal pro-B-type natriuretic peptide; AST: Aspartate transaminase; ALT: Alanine transaminase; PT: Prothrombin time; INR: International normalised ratio

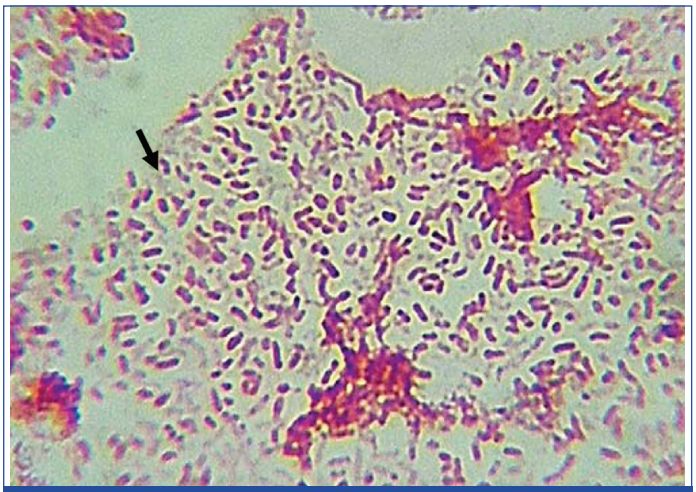
Chest radiography revealed haziness in the right middle and lower lung zones [Table/Fig-3].



[Table/Fig-3]: Chest radiograph demonstrating areas of haziness involving the right middle and lower lung zones, with the abnormal opacities indicated by a green arrow.

Urine and Bronchoalveolar Lavage (BAL) fluid, collected for microbiological analysis on the following day, subsequently isolated *Klebsiella pneumoniae.*

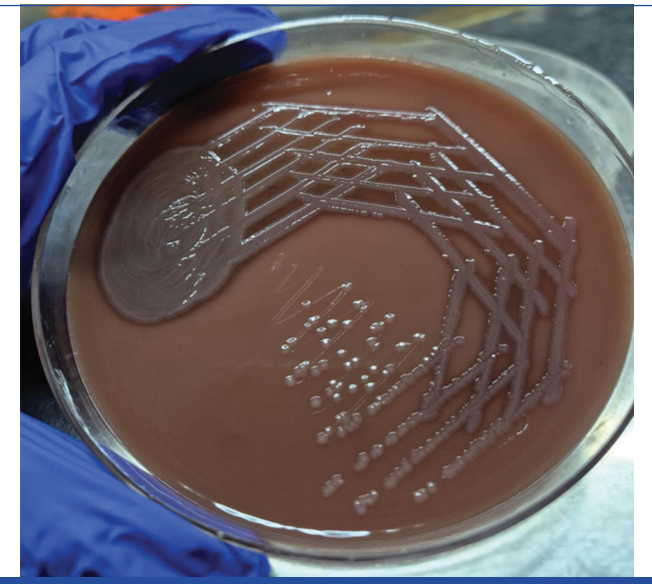
Blood culture bottles flagged positive for microbial growth after 24 hours of incubation in automated blood culture system (BD BACTEC™). Gram staining revealed Gram-negative bacilli, some of which were curved [Table/Fig-4].



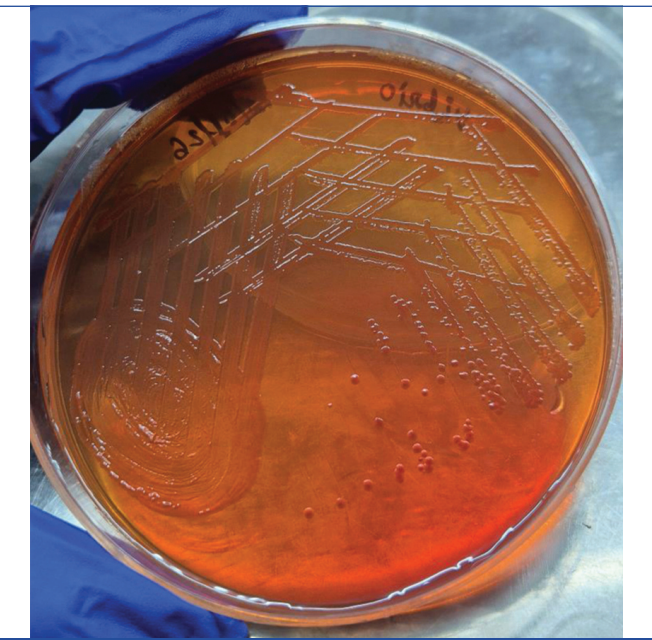
[Table/Fig-4]: Gram stain showing curved Gram-negative bacilli (1000×).

Subculture was done from the blood culture bottles and incubated overnight at 37°C. Haemolytic, oxidase-positive colonies were noted on the blood agar [Table/Fig-5]; smooth, round, moist, greyish colonies were observed on chocolate agar [Table/Fig-6] and non-lactose-fermenter colonies on MacConkey agar [Table/Fig-7].

[Table/Fig-5]: *Vibrio cholerae* isolate showing beta-haemolytic colonies on blood agar.

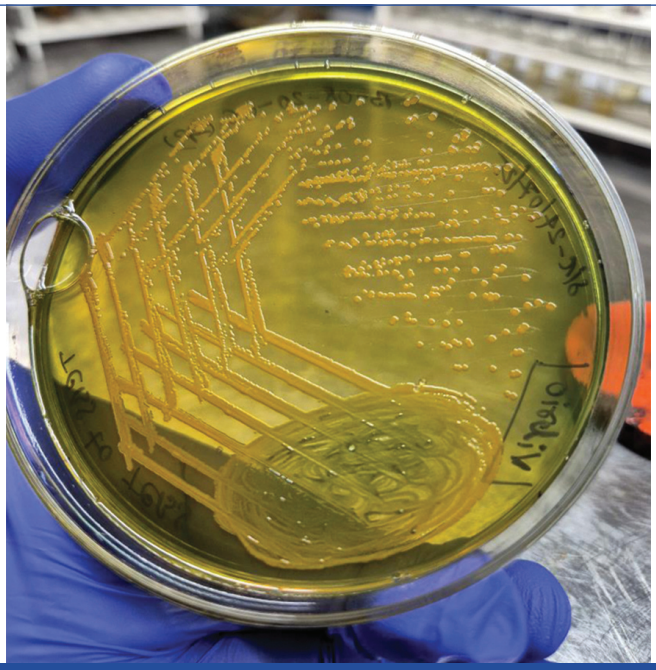


[Table/Fig-6]: *Vibrio cholerae* isolate showing smooth, round, moist, greyish colonies on chocolate agar.



[Table/Fig-7]: *Vibrio cholerae* isolate showing non lactose-fermenter colonies on MacConkey agar.

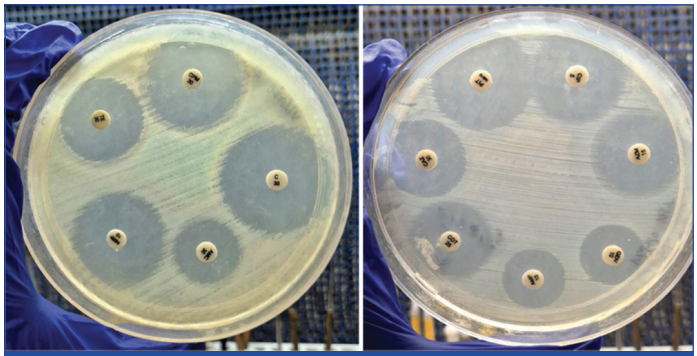
The isolate demonstrated characteristic darting motility on hanging-drop preparation. The isolate was identified as *Vibrio cholerae* with 99% probability by the Microscan WalkAway® plus system (Beckman Coulter, USA). Subculture on Thiosulfate-Citrate-Bile Salt-Sucrose (TCBS) agar showed characteristic yellow colonies [Table/Fig-8].



[Table/Fig-8]: *Vibrio cholerae* isolate showing yellow colonies on Thiosulfate-Citrate-Bile Salt-Sucrose (TCBS) agar.

Species confirmation was done by the ICMR–National Institute for Research in Bacterial Infections (NIRBI) Kolkata using standard biochemical methods. Slide agglutination with specific antisera identified the isolate Serotype O17.

AST of the isolated strain was performed by the Kirby-Bauer method on Mueller-Hinton Agar (HiMedia®) [Table/Fig-9], and results were interpreted according to Clinical and Laboratory Standards Institute (CLSI), M45 guidelines [1].



[Table/Fig-9]: Antimicrobial susceptibility profile of NOVC isolate.
*AMP: Ampicillin; *CAZ: Ceftazidime;*CPM: Cefepime; *AMC: Amoxicillin/clavulanate; *PIT: Piperacillin/Tazobactam; *AZM: Aztreonam; *CIP: Ciprofloxacin; *GEN: Gentamicin; *COT: Co-trimoxazole; *MRP: Meropenem;*TE: Tetracycline; *C: Chloramphenicol

The isolate was susceptible to the tested β-lactams, β-lactam/β-lactamase inhibitor combinations, carbapenems, aminoglycosides, fluoroquinolones, and trimethoprim-sulfamethoxazole.

The patient presented with sepsis, exhibiting a Sequential Organ Failure Assessment (SOFA) score of 9, indicating severe organ dysfunction and a high-risk of mortality [2]. Despite aggressive medical management, her clinical condition rapidly deteriorated to septic shock. Although AST later demonstrated in-vitro susceptibility to multiple antimicrobial classes, no modification of antimicrobial therapy could be undertaken as the patient succumbed within 48 hours of admission.

DISCUSSION

NOVC have increasingly been recognised as a cause of invasive infections, particularly bacteraemia, in elderly individuals and those with co-morbidities [1,3]. In contrast to *V. cholerae* O1 and O139, invasive NOVC infection often lacks diarrhoea and may instead present with non specific systemic features such as sepsis or respiratory distress, as observed in the present case. This atypical presentation poses a diagnostic challenge and may delay consideration of *Vibrio* species in the differential diagnosis [4].

The source of infection in this patient could not be definitively identified. There was no history of seafood consumption, travel, exposure to coastal or brackish water, or contact with open wounds. Importantly, no other cases of febrile illness or diarrhoeal disease were reported from the patient's household or neighbourhood, making a point-source outbreak unlikely. The absence of a history of seafood consumption or recent travel to coastal areas delayed any suspicion of *Vibrio cholerae* infection [5].

Exposure through municipal water supply remains a plausible route, as *Vibrio cholerae* is known to persist in freshwater environments and biofilms within water distribution systems [6-8]. However, the absence of similar illness among co-residents and neighbours suggests that, if waterborne exposure occurred, it was likely sporadic rather than due to widespread contamination.

The patient's advanced age, COPD, and the presence of ascites-suggestive of possible underlying hepatic dysfunction- likely contributed to impaired host defences, facilitating bloodstream invasion [7]. Several case reports describe similar presentations where no clear environmental source could be established, reinforcing the hypothesis that host-related factors play a dominant role in the pathogenesis of NOVC bacteraemia [8-15]. These previously reported cases are summarised in [Table/Fig-10].

Despite early empirical administration of broad-spectrum antimicrobial therapy and subsequent confirmation of antimicrobial susceptibility, the patient experienced rapid clinical deterioration culminating in refractory septic shock. This unfavourable outcome underscores that mortality in NOVC bacteraemia is often determined by rapid disease progression and host vulnerability rather than delayed or inappropriate antimicrobial therapy. Considering the increasing global incidence of *Vibrio* species linked to environmental factors

Publication by	Country, year	Age (years)/ gender	Risk factors	Mode of transmission	Clinical presentation	Treatment	Outcome
Chowdhury G et al., [8]	India, 2016	6.5/female	Burkitt's lymphoma	Could not be traced	Abdominal pain, vomiting	Cefepime/ Tazobactam, Teicoplanin, Ciprofloxacin	Death
		56/female	Diabetes mellitus	Could not be traced	Chills, swelling of the right lower leg	Piperacillin/ Tazobactam, Imipenem	Death
		72/male	None	Could not be traced	Fever, chills, nausea, dizziness	Not mentioned	Recovered
Kaki R et al., [9]	Saudi Arabia, 2017	62/male	Diabetes mellitus	Could not be traced	Fever, epigastric pain, and vomiting	Piperacillin-Tazobactam, Ciprofloxacin	Recovered

Shanley J et al., [10]	USA, 2019	62/male	Chronic hepatitis C with liver cirrhosis	Could not be traced	Right leg swelling, fatigue and chills	Ceftriaxone, Doxycycline	Recovered
Rodríguez JY et al., [11]	Colombia, 2023	79/female	Hypertension	Could not be traced	Abdominal pain, associated with constipation	Piperacillin-Tazobactam, Metronidazole	Death
Islam MT et al., [12]	Bangladesh, 2024	30/male	β-thalassemia	Could not be traced	Acute diarrhoea, abdominal pain, nausea, and fever	Meropenem, Metronidazole	Death
Marino A et al., [13]	Italy, 2024	71/male	Lung cancer with bone and liver metastases, hypertension, benign prostatic hypertrophy	Could not be traced	Fever, asthenia, confusion; a self-limiting episode of diarrhoea two months earlier	Ceftriaxone, Ciprofloxacin	Recovered
Zmeter C et al. [14]	Lebanon, 2018	74/female	Pancreatic adenocarcinoma with liver metastasis, diabetes mellitus	Could not be traced	Fever, nausea, vomiting, and abdominal pain	Ciprofloxacin	Recovered
Alex V and Moodley M [15]	South Africa, 2025	39/male	None	Could not be traced	Dyspnoea, chest pain, vomiting, diarrhoea and chronic bipedal oedema	Amoxicillin/ clavulanate	Death
Kumar A et al.,	India, 2026	80/female	COPD, Ascites	Could not be traced	Acute onset shortness of breath, altered sensorium, bipedal oedema→ sepsis with septic shock	Piperacillin/ Tazobactam Doxycycline, Clindamycin	Death

[Table/Fig-10]: Comparative analysis of previously reported cases of NOVC bacteraemia and the present case [8-15].

such as climate change and global warming, enhanced awareness regarding invasive NOVC infections is crucial [15].

CONCLUSION(S)

This case underscores that NOVC bacteraemia is a rare but potentially fatal condition, particularly in elderly patients with co-morbidities. Rapid progression to septic shock may occur despite early empirical therapy. Further research is warranted to ascertain host risk factors and pathogen virulence factors, and also to establish optimal treatment regimens.

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