

# A Case of O1 Vibrio Cholera Bacteremia and Primary Peritonitis in a Patient With Liver Cirrhosis

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## Abstract

*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. In contrast to non-O1/non-O139 *Vibrio cholerae*, which are more prone to invade the bloodstream, *Vibrio cholerae* O1 is rarely the cause of bacteremia. We report a case of O1 *Vibrio cholera* bacteremia and primary peritonitis in a patient with liver cirrhosis. The literature on the subject is also reviewed.

**Keywords:** *Vibrio cholera*; Bacteremia; Peritonitis

## Introduction

*Vibrio cholera* is a facultative, gram negative anaerobic organism. It is classified on the basis of the somatic "O" surface antigen and based on this, they are divided into O1/O139 and non-O1/non-O139 serogroups [1]. *Vibrio cholerae* O1 is generally regarded as a non-invasive enterotoxigenic organism causing gastroenteritis of various severities [1]. This is in contrast to *Vibrio cholera* non-O1, although biochemically indistinguishable from *Vibrio cholera* O1, has been often associated with extra intestinal infection including septicemia, wound infection, peritonitis, skin infection, cellulitis, necrotizing fasciitis, endophthalmitis, ear infection, cholecystitis and meningitis [2-11]. This is usually seen in patients with diabetes mellitus, liver cirrhosis, and chronic renal failure on dialysis, hematological diseases, immunocompromised

patients and post splenectomy [2, 6]. These infections are often associated with ingestion of contaminated seafood and associated with a high mortality rate [2, 6, 7, 11]. We report one unusual and rare case of bacteremia and primary peritonitis caused by O1 *Vibrio cholera* serotype Ojawa in a patient with liver cirrhosis.

## Case Report

A 47-year-old male presented to our emergency room with severe hematemesis and melena of two days duration and dizziness on the day of presentation. He is a known diabetic and hypertensive for the last 10 years and receiving insulin, atenolol and aspirin. He gave history of taking non-steroidal anti-inflammatory drugs for one week prior to presentation following tooth extraction. There was no history of previous similar attacks, no history of blood transfusion, alcohol intake or drug abuse. There was no history of diarrhea or intake of raw fish. Clinically, he was sick looking, pale, hypotensive and tachycardic. His temperature was 37.5°C, pulse 120/minute and blood pressure was 90/70 mmHg with postural drop. His chest was clear and had normal heart sounds. Abdominal examination revealed distension with ascites, epigastric tenderness and splenomegaly. There was no lower limb edema. His initial investigations showed a hemoglobin of 7.1 g/dl, white cell count of  $19.29 \times 10^3/\text{mm}^3$  and platelets of  $158,000/\text{mm}^3$ . His total bilirubin was 29.9 mmol/L, direct bilirubin of 9.92 mmol/L, ALT (alanine transaminase) was 266 u/dl, and AST (aspartate transaminase) was 335 u/dl. His BUN, creatinine and electrolytes were normal. His PTT (Partial Thromboplastin Time) was normal while his PT (Prothrombin Time) was 17.52 seconds (normal: 10.4 - 14 seconds) and his INR (International Normalized Ratio) was 1.52 (normal: 0.8 - 1.2). He was admitted to the intensive care unit and started on intra venous octreotide, pantoprazol, intravenous fluids and blood transfusion. His blood pressure dropped further and he was electively intubated, ventilated and started on nor-epinephrine and intravenous antibiotics (Piperacillin and Tazobactam). Tapping of the ascetic fluid was done and this was diagnostic of spontaneous bacterial peritonitis (white blood cell count of  $2,982/\text{mm}^3$ , polymorph

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neutrophils were 92% and red blood cells of 618/mm<sup>3</sup>). He had upper gastrointestinal endoscopy which showed grade III esophageal varices with red color sign for which band ligation was performed. There were also fundal gastric varices, gastropathy and duodenopathy. His preliminary blood culture showed gram negative bacilli. He improved gradually, extubated and then shifted to the ward. He was investigated for chronic liver disease which showed negative hepatitis B and C. His abdominal ultrasound showed cirrhotic liver with signs of portal hypertension and splenomegaly but no focal liver lesion. Schistosomiasis screening was positive with a titer of 1:256, for which he was given praziquantel (40 mg/kg). The ascetic fluid culture showed no growth but the blood culture final result grew *Vibrio cholera* O1 serotype Ojawa sensitive to ampicillin, ciprofloxacin, bactrim, ceftazidime and tetracycline. He was started on levofloxacin in addition to Tazocin. His stool culture was negative. His repeat blood cultures came also to be negative. The initial blood culture was sent to another hospital for reconfirmation and the result came to be positive for *Vibrio cholera* O1 serotype Ojawa. The patient improved markedly and was discharged home in a good general condition to be followed-up for liver transplant workup.

## Discussion

*Vibrio cholera* is classified according to the somatic “O” surface antigen. Those that agglutinate with “O” antiserum are called O1 which are responsible for all epidemics of cholera. All other types that do not agglutinate with “O” antiserum are collectively called non-O1 *Vibrio cholera* [1]. The *Vibrio cholera* O1 group exists as two biotypes, classic and El Tor and both of them contain two major serotypes, the Ogawa and Inaba. An additional serotype, Hikojima was also described but this is rare. A new serotype was also discovered causing an epidemic in Bangladesh and India which was given the designation O139 Bengal after the area where it was first discovered. Since the discovery of this serotype, *Vibrio cholera* is classified into O1 and O139 and all other serogroups as non-O1/non-O139 [1].

Non-O1/non-O139 *Vibrio cholerae* are distributed worldwide. They are concentrated in fish and shellfish and contaminated undercooked seafood. Non O-1 *Vibrio cholera* infection is often associated with ingestion of contaminated seafood and its common presentation is sporadic gastroenteritis. They can however cause serious life threatening infections following the consumption of raw seafood or exposure of damaged skin to contaminated salt water. This is seen in patients with predisposing illnesses such as liver cirrhosis, chronic renal failure on dialysis, immunosuppressed patients, hematological diseases, diabetes mellitus and postsplenectomy [2, 6]. A mortality as high as 50% has been reported in affected patients [12]. A variety of infections caused by non-

O1/non-O139 serotypes in such patients have been reported. These include septicemia, bacteremia, peritonitis, cellulitis, necrotizing fasciitis, skin infection, endophthalmitis, cholecystitis, meningitis and ear infection [2-11]. Lin et al reported a large series of 21 patients with liver cirrhosis who had non-O1 *Vibrio cholera* bacteremia and presented with ascites (95.2%), fever (81%), abdominal pain (52.4%), diarrhea (33.3%), and cellulitis with bullae formation (19%). In 10 of them there was concurrent spontaneous bacterial peritonitis [2]. The overall case-fatality rate was 23.8%, but 75% of the deaths were observed in patients with skin manifestation. Thamlikitkul on the other hand reported a 50% mortality among 20 patients with *Vibrio cholera* bacteremia, the majority of them had liver cirrhosis [12]. In 23 previously reported cases, the fatality rate was found to be 61.5% [11]. Ko et al reported 30 episodes of non-O1, non-O139 *Vibrio cholerae* infections [6]. They found three major clinical presentations: bacteremia with concurrent spontaneous bacterial peritonitis or invasive soft-tissue infections that occurred solely in cirrhotic patients; self-limited acute febrile gastroenteritis that occurred in patients with no underlying medical disease; and necrotizing fasciitis or cellulitis that often resulted from a wound on extremities. Other manifestations in their series included fatal pneumonitis in a drowned man and acute pyosalpinx [6].

The majority of the reported cases of non-O1/non-O139 *Vibrio cholera* infections were in patients with liver cirrhosis and taking the high mortality in consideration, patients with liver disease should be warned about the potential dangers of consuming raw or undercooked seafood as well as avoiding exposure of wounds to seawater. It is also important for physicians to consider this diagnosis in patients who have underlying risk factors specially where there is history regarding recent seafood ingestion.

*Vibrio cholerae* serogroup O1 is generally regarded as a non-invasive enterotoxigenic organism causing gastroenteritis of various severities. In contrast to non-O1/non-O139 *Vibrio cholerae*, which can invading the bloodstream causing bacteremia and septicemia, *Vibrio cholerae* O1 rarely causes bacteremia or invasive extraintestinal disease. Restrepo et al reported two patients with hepatitis C liver cirrhosis who developed bacteremia due to non-O1 *Vibrio cholera* in one and O1 serotype in the other [13]. They postulate that the hemolytic properties of these organisms contributed to their virulence in immunocompromised hosts. Thamlikitkul in 1990 reported 26 isolates of *Vibrio cholera* from the blood of patients, mostly adults with liver cirrhosis [12]. Only 20 of them were available for clinical analysis. The isolates in these patients were classified as non-O1 in 13 patients, 3 were *Vibrio vulnificus* and 10 were other *Vibrio* species. There were no details regarding the underlying diseases in these 10 patients. To our knowledge, this case report represents the second documented case of *Vibrio cholera* type O1 to cause bacteremia with spontaneous primary bacte-

