A fatal non-O1 *Vibrio cholerae* septicemia in a patient with liver cirrhosis

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*Vibrio Cholerae* (*V. cholerae*) strains are usually divided into O1 and non-O1 serogroups according to the different antigens that they synthesize.1 *Vibrio cholerae* species cause gastrointestinal infections (especially O1), or extra intestinal infection (particularly non-O1). There has been several reports of bacteremia and other septic conditions associated with non-O1 *V. cholerae*, many of these infections have followed a fatal course, presenting as a fulminant septicemia in patients with liver diseases, who had ingested raw or undercooked seafood.2 In this report, we present the case of a Saudi male with Lawrence Moon Biedl syndrome, cirrhosis and diabetes mellitus (DM), who developed fatal septicemia caused by non-O1 *V. cholerae*.

A 34-year-old Saudi male, known patient of Lawrence Moon Biedl syndrome, DM, liver cirrhosis and positive hepatitis B virus, was admitted through the Emergency Room to the medical unit of King Abdul-Aziz University Hospital with diarrhea, abdominal pain and distention of 10 days duration. On physical examination, he was alert, icteric, with a temperature of 36.3°C, heart rate of 84 beats/minute, and blood pressure of 129/71mm Hg. Ascites was present with abdominal tenderness. The patient was treated with osteo-rectal on tablet po od, essential one tab po od, legolon 70mg tid, motilium 10mg tab po tid, pentazolone 20mg one tablet bid and ceftriaxone one gram intravenously (IV) bid. A specimen of fluid, the leukocyte count was 2400/mm3 with 80% polymorphonuclear cells. The laboratory investigations showed a high blood leucocyte count of 15.6/ul (normal 3-11000/ul) with 85% neutrophils, lymphocytes 3.4%, monocytes 9.7% and eosinophils 2%. Hemoglobin level was 11.5g/dl (normal 13-17g/dl). His sodium serum level was 129mmol/L (normal 135-148mmol/L), potassium 5.4mmol/L (normal 3.5-5.5mmol/L), serum creatinine was 281µmol/L (normal 40-120µmol/L), total protein 49g/L (normal 60-87g/L), albumin 12g/L (normal 34-50g/L), aspartate aminotransferase 732 iu/L (normal 5-50 iu/L), alanine aminotransferase 249 iu/L (normal 5-65 iu/L), total bilirubin 593 µmol/L (normal 0-17 µmol/L) and direct bilirubin 457 µmol/L (normal 0-7 µmol/L). Despite treatment, the patient’s condition quickly worsened, a septic shock was developed, blood pressure dropped to 88/53mm Hg, the patient was moved to the intensive care unit and was given intravenous life support therapies and piperacillin/tazobactam 4.5g IV every 6 hours, but 24 hours later he died.

Blood culture was performed using the automated blood culture system BacT-Alert [Organon Teknika, United States of America (USA)]. Ten milliliter of the patient’s blood was inoculated into each bottle of blood culture, one for aerobic and the other for anaerobic growth. Both bottles were positive after 10 hours incubation, the gram-stained smears of the bottles showed gram-negative curved rods. Subculture of the positive blood culture was carried out onto sheep blood agar and chocolate agar that were incubated in an atmosphere of 5% CO2 at 37°C for 24 hours. The isolated colonies were beta-hemolytic on blood agar, oxidase positive were identified as *V. cholerae* with the use of API 20E (Analytab, Inc.) and the growth on thiosulfate-citrate-bile-salt-sucrose agar (TCBS), as large raised shiny yellow colonies (Figure 1). Further identification as *V. cholerae* was carried out by using the Vitrek-2 System (bioMerierieux Inc., Hazelwood, MO, USA). The susceptibility testing of the isolate was performed by both the Vitrek-2 System, and by Kirby-Bauer disk diffusion method, according to the National Committee for Clinical Laboratory Standards guidelines,3 and showed it to be sensitive to all the antibiotic tested including ampicillin, amoxyccillin/clavulanic acid, gentamicin, amikacin, cefoxime, piperacillin, piperacillin/tazobactam, ciprofloxacin, meropenem and ceftriaxone. The organism was identified as non-O1 *V. cholerae* as it failed to agglutinate with the *V. cholerae* O1 antiserum (Difco Laboratories, Hazelwood, MO, USA). The ascitic fluid was processed in blood culture bottles, which were directly inoculated with 10ml of ascitic fluid at the bedside at the time of paracentesis. The ascitic fluid culture was negative for any organisms after 7 days of incubation in the BacT-Alert.
The stool specimen received from the patient was processed for common enteric pathogens including Salmonella, Shigella and Campylobacter species, by inoculation into MacConkey agar, desoxycholate agar, and xylose-lysine-desoxycholate agar (Saudi Prepared Media Company, Saudi Arabia) as well as TCBS agar for V. Cholerae. No pathogens were isolated from the stool specimen. To assess the clinical features and susceptibility of cirrhotic patients to non-O1 V. cholerae bacteremia, 21 patients with underlying cirrhosis and the aforementioned bacteremia were retrospectively reviewed by Lin et al.; seafood ingestion (7 cases), seawater exposure (2 cases), were risk factors, but nosocomial infections were also noted in 6 cases. Presenting symptoms and signs included ascites (95.2%), fever (81%), abdominal pain (52%), diarrhea (33%) and cellulitis and bullae formation (19%). The over-all case-fatality rate was 23.8%, 75% of the deaths were observed in patients with skin manifestations. Our study is consistent with these findings, where our patient had a history of seafood ingestion 2 days before his illness, and the main presenting symptoms and signs were ascites, fever, abdominal pain and diarrhea. Another study in septicemia with non-O1 V. cholerae bacteremia in patients with liver cirrhosis; 5-Year experience from a single medical center. Am J Gastroenterol 1996; 91: 336-340.

References