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CASE REPORT

Non-O1 non-O139 *Vibrio cholerae* Bacteraemia and Peritonitis Associated with Chronic Liver Disease

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ABSTRACT

A 53-year-old Bangladeshi male was admitted to the hospital with a 2-day history of watery diarrhoea associated with fever, vomiting and abdomen pain. The patient admitted that his symptoms started after he had eaten fish at a local restaurant. Past medical history was remarkable for liver cirrhosis due to hepatitis B virus. Clinical examination showed positive shifting dullness. Both samples of blood and ascitic fluid grew *Vibrio cholerae* non-O1 non-O139, which was sensitive to ampicillin, amoxicillin-clavulanic acid, ceftriaxone and cotrimoxazol. The results were confirmed by the reference laboratory (Bioscience - Germany). Tazocin (piperacilline-tazobactam) was given for 10 days. On the following days, the patient recovered; diarrhoea and fever were subsided and consequently the patient was discharged.

Key words: Non-O1 *Vibrio cholerae*, peritonitis, hepatitis B virus

Introduction

Vibrio cholerae is divided into three main subtypes: O1 toxigenic, O1 non-toxigenic and non-O1 strains [1]. *V. cholerae* species cause gastrointestinal infections (especially O1) and extraintestinal infection (particularly non-O1). There have been several reports of bacteraemia and other septic conditions associated with non-O1 *V. cholerae*, many of these conditions have followed a fatal course, presenting a fulminant septicaemia in patients with liver disease, who had ingested raw or under-cooked seafood [2].

Septicaemia due to non-O1, non-O139 *V. cholerae* is very rare, usually occurs in patients with liver cirrhosis. We report a 53-year-old male with a history of liver cirrhosis due to hepatitis B virus, who developed septicaemia and bacterial peritonitis due to *V. cholerae* non-O1, non-O139.

Case Report

A 53-year-old Bangladeshi male was admitted to the hospital with a 2-day history of watery diarrhoea associated with fever, vomiting and abdomen pain. The patient admitted that his symptoms started after he had eaten fish at a local restaurant. Past medical history was remarkable for liver cirrhosis due to hepatitis B virus. Other medical history was unremarkable. On initial examination, the patient was conscious and oriented and looked ill and dehydrated, with a temperature of 39.5°C, pulse 108/min and blood pressure 115/65 mmHg. Abdominal examination revealed

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positive shifting dullness but no organomegaly, and there were no stigmata of chronic liver disease. Rectal examination was normal and examinations of other systems were unremarkable.

Initial investigations are shown in [Table/Fig 1]. Blood specimens were submitted for culture prior to his treatment with intravenous hydration and broad-spectrum antibiotic Tazocine (piperacilline-tazobactam). Abdomen paracentesis was done and samples were sent for cell count, gram stain, culture and AFB. See [Table/Fig 2].

Table/Fig 1

Total WBC count	7800/ μ l
HB%	11.1 g/dL
Platelets	78000/ μ l
ESR	29 mm/hr
Bicarbonate	18 mEq/l
BUN	3.6 mmol/l
Creatinine	89 μ mol/l
Sodium	133 mEq/l
Potassium	3.4 mEq/l
Total protein	6.4 g/dl
Albumin	2.0 g/dl
AST	106 U/l
ALT	78 U/l
ALP	148 U/l
Total bilirubin	30 μ mol/l
Alpha fetoprotein	29 U/l
PT	23.8 s
PTT	66.8 s
INR	2.7

Laboratory findings

Table/Fig 2

Total protein	1.8 g/dl
Albumen	0.9 g/dl
LDH	575 U/l
WBC	612/ μ l
Neutrophils	65%
Lymphocytes	35%
Gram stain and culture	<i>V. cholerae</i> non-O1 non-O139
Acid fast bacilli	Negative
Tuberculosis culture	Negative

Ascitic fluid findings

However, both samples of blood and ascitic fluid grew *V. cholerae* non-O1 non-O139, which was sensitive to ampicilline, amoxil-clavulanic acid, ceftriaxone and septrin. The results were confirmed by the reference laboratory (Bioscience –

Germany). Stool was sent for culture to identify *V. cholerae* non-O1, but it was not detected. Tazocine was continued for 10 days; during this period the diarrhoea and fever subsided and the patient was discharged.

Discussion

V. cholerae serogroup O1 is generally regarded as a noninvasive enterotoxigenic organism causing gastroenteritis of various severities [3]. In contrast, *V. cholerae* non-O1, although biochemically indistinguishable from *V. cholerae* O1, has been often associated with extraintestinal infection [4]. Non-O1 *V. cholerae* can cause small outbreaks of diarrhoeal illness related to contaminated seafood. There are, however, numerous case reports of bacteraemia caused by non-O1 *V. cholerae* in persons with predisposing conditions, most commonly cirrhosis [5], but also nephrotic syndrome, diabetes, haematologic malignancies, gastrectomy and AIDS/lymphoma [6],[7]. Our patient had liver cirrhotic due to hepatitis B virus. Although non-O1 non-O139 *V. cholerae* was reported once before in Qatar [8], this reported case is the first with identified cause and good outcome.

In one study of 30 cases, three major clinical presentations were found: bacteraemia 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 necrotising fasciitis or cellulitis that often resulted from a wound on extremities. Other manifestations included fatal pneumonitis in a drowned man and acute pyosalpinx [5].

The relevant explanations for the mechanism of invasive vibrio infections frequently occurring in patients with cirrhosis remain obscure; there are many hypotheses, such as decreased serum bactericidal activity, impaired filtration function in the cirrhotic liver or increased serum iron levels [6].

Third-generation cephalosporin and tetracycline analogue or a flouroquinolone alone is recommended for treatment of severe *Vibrio* infection [5].

Nonetheless, this isolate was sensitive to ampicilline, amoxil-clavulanic acid, ceftriaxone and co-trimoxazol in vitro.

The crude case fatality rates among cirrhotic patients with non-O1 *V. cholerae* bacteraemia have been high, ranging from 23.8% [2] to 61.5% [9]. The poor outcome appears in part to be related to pre-existing cirrhosis [5]. Our patient survived despite being cirrhotic. Prevention is the best treatment. Therefore, particularly for cirrhotic patients, it is wise to avoid any contact with contaminated seawater or fresh water or consumption of raw or under-cooked sea food [5].

In conclusion, non-O1 *V. cholerae* septicaemia should be considered in the differential diagnosis of fever, abdominal pain and diarrhoea in patients with underlying chronic liver disease.

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