SEPTICEMIA CAUSED BY A NON-01 *VIBRIO CHOLERAE*

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Strains of non-01 *Vibrio cholerae* are morphologically and biochemically similar to 01 *Vibrio cholerae*, but they do not agglutinate with *Vibrio cholerae* group 01 antiserum.[1] These strains, previously known as non-agglutinating vibrios (NAG) or non-cholera vibrios (NCV) have been associated with gastroenteritis and sometimes cholera-like disease.[2] However, unlike 01 *V. cholerae*, which rarely causes extraintestinal infections, non-01 *V. cholerae* strains were reported to cause a variety of extraintestinal clinical syndromes, including septicemia.[1,3-5] Septicemia due to non-01 *V. cholerae* is usually associated with liver diseases, malignancy or immunosuppression.[6] The organism has also been incriminated as a cause of septicemia following the consumption of seafoods.[1,6]

We report a case of septicemia due to non-01 *V. cholerae* in a patient suffering from a malignant non-Hodgkin's lymphoma. To the best of our knowledge, this is the first reported case from the Arabian peninsula. A brief review of the subject is also given.

Case Report

A 60-year-old Saudi male was referred to King Khalid University Hospital complaining of gradual abdominal swelling of one month's duration. He also had had on-and-off attacks of moderate grade fever, anorexia and weight loss over the last five months. Four weeks prior to his admission, he noticed small swellings in the cervical and axillary regions. He was diabetic and had been on hypoglycemic drugs for the last two years. He had not been to the seaside during the previous three months. It was not clear whether seafood had been consumed recently. He had not had diarrhea during the previous two weeks.

Examination showed an ill-appearing patient with normal vital signs. Cardiovascular system, chest and central nervous system were all normal. Abdominal examination revealed distended abdomen with tense ascites. The lower limbs showed bilateral pitting edema.

On admission, laboratory investigations showed the following findings: white cell count (WBC) 10.4×10^9/L, 60% segmented neutrophils, 30% lymphocytes, 9% monocytes, 1% eosinophils. Erythrocyte sedimentation rate (ESR) 3 mm/h, red blood cell count was 4.7×10^{12}/L, Hgb 14.1g/dL. Sodium (Na) was 140 mmol/L, potassium (K) 4.1 mmol/L, chloride (Cl) 106 mmol/L, CO_2 24 mmol/L, urea 8.5 mmol/L, creatinine 70 µmol/L.

Liver function tests (LFTs) showed total bilirubin of 10 µm/L, total protein 60 g/L, albumin 35 g/L, random blood sugar 14.7 mmol/L. All other biochemical investigations were normal. A thick chylous ascitic fluid, collected only after several failed attempts, had a pH of 7.5 and a protein content of 34 g/L. Chest x-ray showed hilar lymphadenopathy and elevation of the right diaphragm with mild atelectasis at the base of the right lung. Computerized tomograph (CT) scan of the abdomen revealed extensive retroperitoneal and mesenteric lymphadenopathy encasing the aorta and mesenteric roots, hepatosplenomegaly and massive ascites. Cytology of the chylous ascitic fluid showed mature lymphocytes; no malignant cells were seen. The histological diagnosis of the cervical lymph node biopsy was malignant non-Hodgkin's lymphoma of the follicular type. The following chemotherapy regimen was given intravenously on day 1: cyclophosphamide 750 mg/m^2, vincristine 1.4 mg/m^2, mitoxantrone 10 mg/m^2. In addition, prednisolone 100 mg by the oral route was also given once daily for five days. On the 7th day after the start of chemotherapy, the patient started to have low-grade fever (37.9°C), and hypotension (90/50). He was put on amikacin and ceftazidime.

The patient failed to respond to this therapy, developed septicemic shock and died on the 8th day after the start of chemotherapy. Two previously collected sets of blood culture were negative, but a blood sample collected while the patient was febrile (day 7) grew an oxidase-positive, curved-gram-negative bacillus from both aerobic and anaerobic bottles. It showed β hemolysis on blood agar plate and yellow colonies on thiosulphate citrate sucrose bile salt agar (TCBS).

The organism was later biochemically identified by API 20E as *V. cholerae*. However, it failed to agglutinate with

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