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Bacteraemia due to non-toxigenic *Vibrio cholerae*: The risks of eating seafood in a cirrhotic patient☆

Bacteriemia por *Vibrio cholerae* no toxigénico: los riesgos del consumo de marisco en un paciente cirrótico

Vibrio cholerae (*V. cholerae*) is a Gram-negative, curved, mobile, oxidase- and catalase-positive, facultative anaerobe bacillus commonly found in marine environments.[1] More than 200 different serogroups have been identified based on the nature of the lipopolysaccharide O-antigen of its wall, and epidemic cholera is caused by the O1 and O139 serogroups.[1] The non-toxigenic serogroups (non-O1 and non-O139) tend to be linked to the onset of self-limiting gastroenteritis after seafood consumption, particularly bivalve filter-feeding molluscs, with symptoms potentially as severe as cholera.[2,3] Notwithstanding its potential involvement in otitis externa, cellulitis and wound infection, the enteroinvasive potential of the non-toxigenic serogroups is limited and only affects hosts with particular comorbidities.[4]

We present the case of a 56-year-old male patient with type 2 diabetes mellitus and alcoholic liver cirrhosis with a Child-Turcotte-Pugh class of A6 (9 points on the MELD score), who had developed ascitic and oedematous compensation, oesophageal and gastric varices and thrombosis of the splenoportal venous axis as prior complications. The patient was taking spironolactone, furosemide, propranolol, tinzaparin and insulin glargine. He attended A&E having experienced the onset, 24 h previously, of dysthermia with a temperature measured at home of 38.5 °C, drowsiness, odynophagia and cough without expectoration, as well as loose bowel movements. The physical examination revealed an axillary temperature of 36.8 °C (after taking an antipyretic), blood pressure of 113/59 mmHg, a heart rate of 87 bpm and clinical signs of chronic liver disease. The lab tests revealed thrombocytopaenia (26.0×10^9 platelets/l), increased acute phase reactants (C-reactive protein 14.9 mg/dl; normal range: 0.1–0.5), hypertransaminasaemia (gamma-glutamyl transpeptidase [GGT] 114 IU/l, alkaline phosphatase 134 IU/l) and mildly impaired liver function tests (total bilirubin 2.7 mg/dl, INR 1.29). The white blood cell count was normal (4.7×10^9 leukocytes/l). An abdominal ultrasound only found a minimal number of ascites barely consistent with underlying chronic liver disease, while the chest X-ray revealed no consolidations. Assuming a possible diagnosis of respiratory tract infection, empirical treatment was started with levofloxacin (500 mg every 24 h) and the patient was discharged after obtaining two sets of blood cultures (BacT/ALERT® 3D system, bioMérieux, Marcy l'Etoile, France). After 24 h, a curved, lactose-and oxidase-positive Gram-negative bacillus was isolated in the MacConkey agar plate of one of the sets, which formed greenish colonies in the blood agar plate (Fig. 1). Using biochemical testing (MicroScan WalkAway® system, Siemens, California, USA), mass spectrometry (Bruker Daltonics, Bremen, Germany) and direct agglutination (BD Difco *Vibrio cholerae* Antisera, Becton Dickinson, Sparks, MD, USA), non-toxigenic *V. cholerae* was identified. The antibiogram confirmed susceptibility to aminopenicillins and trimethoprim/sulfamethoxazole. At that time the patient was asymptomatic and afebrile, so it was decided to maintain treatment with levofloxacin for 10 days. Upon questioning, the patient mentioned having eaten a large amount of shellfish a few days prior to symptom onset, including razor clams (*Ensis* spp.), steamed cockles (*Cerastoderma edule*) and mussels (*Mytilus galloprovincialis*), as well as grilled shrimp (*Aristaeopsis edwardsiana*). His partner, who had also eaten this seafood, coincidentally experienced mild gastroenteritis.

Although the clinical significance and public health impact of the non-toxigenic serogroups of *V. cholerae* (also known as non-agglutinating or non-choleric) was questioned for a long time, its involvement in outbreaks of diarrhoea in immunocompetent subjects after the consumption of contaminated seafood has been documented in several European countries.[2] A recent study demonstrated an elevated prevalence of non-toxigenic *V. cholerae* in prawns (17%) and mussels (9%) harvested off the Italian coast.[5] Given that it requires a temperate saline aquatic environment for optimal growth (>15 °C), it has been suggested that rising average sea temperatures as a result of global warming could explain the increased incidence of *V. cholerae* infection, including at more northern latitudes.[2,5]

The pathogenicity of non-toxigenic *V. cholerae* may be increasing due to the presence of a wide spectrum of virulence factors, including extracellular enzymes, enterotoxins and haemolysins.[5,6] As a result of iron overload and inhibited opsonophagocytosis and reticuloendothelial clearance (which favour bacterial translocation from the lumen of the gastrointestinal tract), liver cirrhosis is one of the most common comorbidities associated with the onset of enteroinvasive *V. cholerae* infections.[4,7] Examples of bacteraemia, spontaneous bacterial peritonitis and

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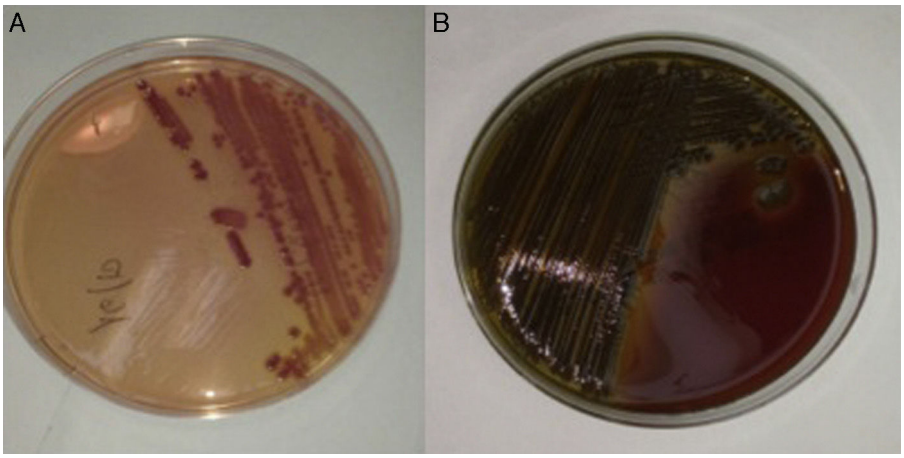

Figure 1 Lactose-positive colonies in the MacConkey agar plate (A) and dark grey, oxidase positive in the blood agar plate (B), later identified as non-toxigenic *Vibrio cholerae*.

endophthalmitis can be found in the literature.[4,6–9] Many of these cases arise in South-east Asia[7–9] and have only been reported anecdotally in Spain.[10] The mortality rate of bacteraemia episodes is believed to be around 47%.[4] Most non-toxigenic *V. cholerae* isolates are susceptible to third-generation cephalosporins, tetracyclines, trimethoprim/sulfamethoxazole and fluoroquinolones.[2,7,8] Thanks to their *in vitro* bactericidal action and good oral bioavailability, fluoroquinolones represent an excellent therapeutic option, as our case study shows.

In conclusion, it is hoped that the non-toxigenic *V. cholerae* serogroups will be considered in the future as an emerging cause of gastrointestinal infection in our setting. Cirrhotic patients are particularly susceptible to bacteraemia and other forms of enteroinvasive infection, which may be associated with significant mortality. This should be taken into account in order to effectively question the patient about their recent eating habits and to potentially administer empirical antibiotic therapy should suggestive symptoms present. Finally, patients with cirrhosis should be warned of the risks that could arise from consuming raw or semi-raw seafood.

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Conflicts of interest

The authors declare that there is no conflict of interest with regards to this article.

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Endoscopic alternative to buried bumper syndrome secondary to Duodopa® pump treatment☆

Alternativa endoscópica al síndrome de buried bumper secundario a bomba de Duodopa®

Continuous intraduodenal levodopa/carbidopa infusion (Duodopa®) is indicated in patients with advanced Parkinson's disease refractory to conventional treatment. This type of novel pump may give rise to adverse effects that tend to be similar in nature to a gastrostomy tube used for other indications such as infection, granuloma, haemorrhage, pneumoperitoneum or buried bumper syndrome.[1,2] We present the case of a 74-year-old male patient with advanced Parkinson's disease who has been undergoing Duodopa® pump treatment for the last two years. He attended A&E owing to a lodged tube and abdominal pain.

An abdominal CT scan was performed which revealed a buried bumper (Fig. 1, arrow). An oral panendoscopy under propofol anaesthesia was then performed using the Olympus EVIS EXERA II® GIF-H180 conventional gastroscope (2.8 mm lumen). A mamelon in the antrum was found with the internal bumper completely buried and the tube protruding through it. The internal tubing was removed through the gastrostomy tube. A 0.0035 guidewire was introduced through the external pump to check its access to the gastric cavity and a conventional sphincterotome was placed following the guidewire (Fig. 1).

First, a number of tangential incisions were made to the exit port with a conventional sphincterotome, and then with a precut needle knife or MicroKnife to expose the bumper and dislodge it. Using the push-pull T technique, the tube was cut about 3 cm from the abdominal wall and then a conventional polypectomy snare was fed through the gastroscope channel before passing through the gastrostomy tube (following the guidewire) and ultimately exiting the abdominal wall. The snare was then tied to the cut end of the tube and perpendicular to the tube to form a ''T'', before being pulled back into the gastric cavity, where it dragged the bumper with it to be removed through the mouth. Finally, a new Freka® PEG Gastric FR 15 tube was inserted (Fig. 2). A Freka® FR 9 enteral tube was then inserted. The distal end of the tube was fed to the duodenum and its correct operation was confirmed. Currently, six months later, the patient is completely asymptomatic.

Whilst the combination of techniques conducted (Needle Knife technique and push-pull T technique) have been previously reported in conventional gastrostomy feeding tubes, this case is of particular interest because they have never been reported in Duodopa® pumps. In this case, an Olympus 120 W Needle Knife at a diathermy power of 80 W was used. The Needle Knife facilitates exposure of the internal bumper, reducing the resistance exerted by the gastric wall on the tube thereby making it easier to dislodge.[3,4]

Although still new, use of Duodopa® pumps in patients with advanced Parkinson's is steadily increasing thanks to its excellent results.[1,2] This means that gastroenterologists will come across this type of tubing and its adverse effects, including perforation and haemorrhage, more and

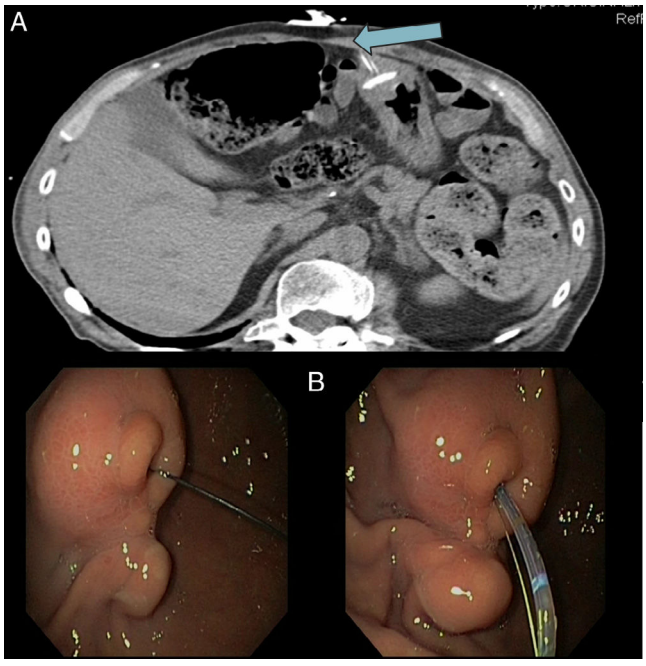

Figure 1 (A) The upper left image of the abdominal CT scan shows the buried bumper. (B) The endoscope image shows the guidewire from the outside through the external tube and the sphincterotome following the guidewire.

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