CASE REPORT

_Campylobacter jejuni_ in the stomach

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**Summary.** _Campylobacter jejuni_ is the commonest cause of acute bacterial enteritis in the UK. However, in this case a 74-year-old lady underwent gastroscopy for an upper gastrointestinal haemorrhage and was noted to have a gastric ulcer. Gastric biopsy revealed spiral gram-negative bacteria and culture yielded a moderate growth of _C. jejuni_. Identification was confirmed by growth characteristics, biochemical tests and PCR amplification of the species-specific flagellin gene—_fla A_. To prevent misidentification, it is important that laboratories routinely culturing gastric biopsies for _Helicobacter pylori_ should perform a rapid urease test and not rely solely on microscopic morphology.

**Introduction**

_Campylobacter jejuni_, a species of curved, motile, gram-negative bacilli, is the commonest cause of infective diarrhoea in the UK. Infections have a seasonal variation peaking at the end of May. Known sources of infection are poultry, milk, water and pets; 10% of infections reported in UK are acquired abroad. Symptoms include profuse watery diarrhoea with fresh blood, nausea, abdominal cramps and fever. The profuse diarrhoea suggests that _C. jejuni_ infection involves the small intestine and evidence of ileitis has been found in some patients. However, the fresh blood, pus and mucus in stools also suggest that the colorectal region is affected. Symptoms normally last 2–7 days but may be prolonged (> 4 weeks). The abdominal pain of campylobacter infection can be very severe and patients with toxic megacolon have been described. Other complications include hepatitis, cholecystitis, cystitis, meningitis, endocarditis, bacteraemia, pancreatitis, reactive arthritis and infective arthritis. An association between campylobacter infection and Guillain-Barré syndrome has been postulated. Infection may also result in neonatal sepsis. A morphologically similar organism—_Helicobacter pylori_—is associated with the pathogenesis of gastritis and peptic ulcer disease. However, association of _C. jejuni_ with gastric ulcer has never been reported. In this report, an unusual case of peptic ulcer syndrome is described in which _C. jejuni_ was isolated from the stomach.

**Case report**

A 74-year-old woman underwent gastroscopy to investigate upper gastrointestinal haemorrhage associated with indomethacin treatment for osteoarthritis. Direct observation showed mild oesophagitis and a superficial ulcer over the lesser curve of the stomach but a normal duodenum. The patient had no symptoms attributable to her large bowel. Histological examination of biopsy samples from the gastric antrum revealed a mild active chronic gastritis with focal areas of intestinal metaplasia and the presence of few spiral gram-negative bacteria consistent in appearance with _H. pylori_ infection. The patient was treated with omeprazole 20 mg daily for her gastric ulcer. Endoscopy after 6 weeks showed that the gastric ulcer had healed and biopsy revealed no organisms present. However _C. jejuni_ was isolated from a gastric biopsy taken on the first occasion. A sample of faeces subsequently collected from the patient did not contain _C. jejuni_.

**Identification of isolate**

The _C. jejuni_ isolate, designated DML 193, was preserved at −70°C in Brain-Heart Infusion Broth (Oxoid) 37 g/L, yeast extract (Oxoid) 4 g/L, horse serum 10% v/v and glycerol (BDH chemicals) 10% v/v. After subculture on to selective campylo-
Table. Properties of various human spiral bacteria and isolate DML 193

<table>
<thead>
<tr>
<th>Organism</th>
<th>Catalase</th>
<th>Oxidase</th>
<th>Rapid urease</th>
<th>Growth at 27°C</th>
<th>Growth at 37°C</th>
<th>Growth at 43°C</th>
<th>H₂S production</th>
<th>NO₃ reduction</th>
<th>Hippurate hydrolysis</th>
<th>Susceptibility to nalidixic acid (30 μg)</th>
<th>Susceptibility to cephalexin (30 μg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>DML193</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>S</td>
<td>R</td>
</tr>
<tr>
<td>C. jejuni subsp. jejuni</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>S or (+)</td>
<td>S</td>
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<tr>
<td>GCLO-211</td>
<td>(+)</td>
<td>+</td>
<td>-</td>
<td>+</td>
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<td>-</td>
<td>-</td>
<td>or (+)</td>
<td>+</td>
<td>S</td>
<td>S</td>
</tr>
<tr>
<td>UA 768</td>
<td>-</td>
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<td>-</td>
<td>+</td>
<td>+</td>
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<td>-</td>
<td>R</td>
<td>S</td>
</tr>
<tr>
<td>H. pylori</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>R</td>
<td>S</td>
</tr>
</tbody>
</table>

(+): Weak reaction.

Figure. Gel obtained after electrophoresis of PCR-amplified DNA with campylobacter specific primers. Track 1, 100 bp ladder size standard; 2, C. jejuni Penner serotype O:44; 3, test strain; 4, 100 bp ladder size standard. Other tracks contain samples not included in this study.

bacter medium G.C. agar base (Oxoid) with defibrinated, lysed horse blood 10% v/v, G.C. supplement 1% v/v and VCAT antibiotic supplements (Oxoid),12 standard laboratory tests for identification of human spiral bacteria were performed (table).

Identification by PCR

The presence of the C. jejuni specific flagellin gene was determined by PCR.13 The fla A gene of C. jejuni comprises a variable region (V1), thought to encode the part of the flagellin protein responsible for antigenic variation, flanked by two constant regions (C1 and C2). The forward primer was located in region C1, and the reverse primer in region C2; these primers yielded a 1.3-kb amplicon.

Isolate DML 193 was identified as C. jejuni subsp. jejuni and could be differentiated clearly from the other spiral bacteria which have been isolated from the human stomach. PCR amplification of the flagellin gene with primers specific for C. jejuni subsp. jejuni confirmed the identity of the isolate DML 193 (figure).

Discussion

Spiral bacteria have been observed in the stomach of human patients since Bottchen’s original report in 1874.14 Warren and Marshall15 characterised a curved micro-aerophilic organism from the stomachs of patients with active gastritis in 1983; this organism is now classified as H. pylori. Other helicobacters known to inhabit the human stomach are H. felis and Gastrospirillum hominis which has recently been moved to the genus Helicobacter as H. heilmannii. The latter is a tightly coiled spiral bacterium, found in c. 0.3% of children and in adults;16 it is urease positive, is associated with active chronic gastritis but has not been cultured in vitro.17 H. felis is found predominantly in cats and dogs but has been known to infect man.

Another spiral organism isolated from the human stomach is referred to as gastric campylobacter-like organisms type 2 (GCLO type 2).18 These bacteria are distinct from other spiral bacteria found in the stomach; they have a predominantly monotrichate flagellar arrangement, a DNA guanine + cytosine base composition of 29 mol %, are incapable of growth at
43°C and are susceptible to cephalothin. Another novel bacterium, UA 768, was isolated from the gastric biopsy of a patient with peptic ulcer syndrome. This organism is unrelated to the genera Campylobacter and Helicobacter, produces H₂S, and is oxidase and catalase negative. Its DNA base composition does not resemble that of any known bacterial species.¹⁹

We have not found any report of the isolation of C. jejuni from the stomach, although it is a well described cause of diarrhoea and is frequently cultured from stool samples. The isolate described here, which was cultured in significant numbers from the gastric biopsy specimen, had growth and biochemical characteristics typical of C. jejuni subsp. jejuni and was also identified by PCR amplification of the specific fla A gene. The relationship of C. jejuni DML 193 with the features of peptic ulcer syndrome in the patient is unclear. It is possible that transient colonisation of the stomach by C. jejuni may occur early in the pathogenesis of campylobacter enteritis. However, as many laboratories do not identify bacteria grown from gastric biopsies with tests that can distinguish between H. pylori and C. jejuni, there are no data to confirm this. Laboratories routinely culturing gastric biopsy samples for H. pylori, a procedure which may be performed more commonly to detect antibiotic resistance, should always check suspected isolates of H. pylori with a rapid urease test to exclude the misidentification of rare isolates of C. jejuni. This report underlines the importance of accurate identification of bacteria cultured from antral biopsy samples and not relying solely on the histopathological appearance.

References