**Campylobacter jejuni** from perforated gastric ulcer: A rare case

**Abstract**

*Campylobacter* species induced enteritis is among the most common in the world. However, its association with gastric perforation is not well documented. We report here a case of a 27-year-old male who presented with a gastric perforation. Gastric biopsy sample revealed Gram-negative spiral bacilli and culture on modified Thayer-Martin agar yielded *Campylobacter jejuni*. The identification was confirmed by standard biochemical tests. This is probably the first documented case report from India of *C. jejuni* from the gastric biopsy.

**Key words:** *Campylobacter jejuni*, gastric biopsy, *H. pylori*

**INTRODUCTION**

Spiral bacteria have been observed in the stomach of human patients since 1874. Warren and Marshall first identified *Helicobacter pylori* in patients of active gastritis in 1983.[1] Other *Helicobacters* known to inhabit the human stomach are *H. felis* and *H. belmanii*.[2]

*Campylobacter jejuni* associated with gastric ulcers and perforations is a rare entity. After a thorough internet search, documented evidence of only one case of *C. jejuni* isolated from a gastric ulcer was found.[2]

We report here a case of *C. jejuni* from gastric biopsy following gastric ulcer perforation. This is probably the first documented case report from India.

**CASE REPORT**

A 27-year-old male patient, resident of Dharavi, Mumbai came to the Surgery Emergency Department on December 2, 2012 with complaints of severe pain in the abdomen of 1-day duration. The pain was generalized, not associated with consumption of food and nonradiating, which increased on movement and decreased on lying still. Patient also gave a history of moderate grade, intermittent fever of the same duration. Patient had no history of vomiting, jaundice, constipation or burning micturition. There was neither history of intake of any medication for prolonged periods of time nor any history of trauma, tuberculosis, hypertension or any other debilitating illnesses in the past.

On examination, his general condition was fair; he was febrile (100.4°F), pulse 90/min, and blood pressure 110/70 mmHg. Per abdomen examination revealed a tender abdomen with generalized pain on palpation. His respiratory system, cardiovascular system, and the central nervous system showed no abnormalities.

Laboratory investigations showed his hemoglobin to be 16.3 g/dL, total leukocyte count 7000/mm³, platelet count 1,60,000/mm³, BUN 12 mg/dL, creatinine 1.0 mg/dL, random blood sugar 193 mg/dL, serum sodium 138 mEq/L, and serum potassium 5.4 mEq/L. His liver function tests were normal. On erect X-ray abdomen, gas under diaphragm was detected, suggestive of perforation.[Figure 1a].

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Patient was taken up for an exploratory laparotomy on the same day. A 2 cm × 2 cm perforated ulcer was seen on the lesser curvature of the stomach in lower one-third. No bleeding and no contamination from gastrointestinal contents were evident; no major blood vessels were involved. The gastric perforation was subsequently repaired. Postoperatively, patient was started on injection amikacin
750 mg once daily, injection metronidazole 100 cc 3 times a day, and injection piperacillin-tazobactam 4 g once daily. Patient’s condition improved and he was discharged without any complications on December 12, 2012.

Edge biopsy of the ulcerated area revealed mucosal ulceration. Lamina propria was ulcerated showing inflammation and infiltration with increased lymphocytes and few polymorphonuclear leucocytes. Blood vessels were dilated and all serosa were consistent with exudate and increased polymorphs. On biopsy, diagnosis was perforative peritonitis with no evidence of granuloma/malignancy.

A 0.2 cm × 0.2 cm edge biopsy from the gastric perforation was sent to the Microbiology Laboratory in Stuart’s Transport medium on December 2, 2012. The sample was divided into three pieces. The pieces were inoculated on blood agar, chocolate agar, and modified Thayer-Martin agar plates respectively and put in a McIntosh Fildes’ jar. A microaerophilic environment was created by vacuum evacuation and the jar was incubated at 37°C. The plates were examined on days 3, 5, and 7. On day 7, 2–3 mm grey-white, moist, glistening, opaque, low convex colonies with entire edges were seen on chocolate agar, and modified Thayer-Martin agar plates. The colonies showed confluent growth along the streak lines on the agar surface. On Gram staining, Gram-negative, spiral bacilli and some coccoid forms were seen [Figure 1b and c]. The growth was catalase and oxidase positive. The organism showed growth at both 37°C and 43°C, reduced nitrates to nitrites, hydrolyzed hippurate, and showed susceptibility to Nalidixic acid and resistance to cephalothin. The organism did not hydrolyze urea and did not show hydrogen sulfide production on triple sugar iron slant [Figures 2 and 3].

The bacterium was hence identified as C. jejuni spp. jejuni according to the biochemical reactions.

A stool sample was subsequently taken from the patient and cultured on Campylobacter charcoal differential agar under microaerophilic conditions. Culture was negative for C. jejuni.

**DISCUSSION**

*Campylobacter jejuni* is a Gram-negative, curved, nonsporing, noncapsulated, motile bacillus. It is usually the most common cause of community acquired inflammatory enteritis in the world. Ingestion of raw milk, partially cooked meat or contaminated water are the most common sources of infection. Enteritis with *C. jejuni* is characterized by abdominal pain, bloody diarrhea, chills and fever. The symptoms are generally self-limited and normally last 2–7 days. Complications such as septic arthritis, meningitis, proctocolitis, and Guillain Barre’s syndrome have been documented.

The most common microaerophilic, curved bacillus isolated from the human stomach is *H. pylori*. However it is rapid urease positive, doesn’t grow at 43°C, doesn’t reduce nitrates to nitrites and is resistant to Nalidixic acid. Another spiral organism infrequently isolated from the human stomach is referred to as gastric...
Campylobacter-like organism type 2. These bacteria don’t grow at 43°C, show variable nitrate reduction and show susceptibility to both nalidixic acid and cephalothin. Another novel bacterium, UA 768, isolated from the gastric biopsy of a patient with peptic ulcer disease was reported by Taylor et al. in 1991. This bacterium is unrelated to the genera Campylobacter and Helicobacter, produces H₂S, is oxidase and catalase negative.

Until now, only one case of C. jejuni isolated from the gastric ulcer is documented. The case was that of a 74-year-old lady who underwent gastroscopy for upper gastrointestinal hemorrhage and was found to have a gastric ulcer. Biopsy of the ulcer revealed spiral Gram-negative bacilli and culture showed a growth of C. jejuni.

It is possible that transient colonization of the stomach by C. jejuni may occur early in the pathogenesis of C. jejuni enteritis. However, a sample of feces collected subsequently from the patient did not reveal C. jejuni.

Though the gastric biopsy sample was processed for H. pylori, Campylobacter species also show moderate growth on chocolate agar. Moreover, modified Thayer-Martin agar may be also tried for isolation of C. jejuni.

Although the primary pathogen implicated in the causation of gastric ulcers is H. pylori, isolation of C. jejuni in this patient indicates the need for a high degree of suspicion and a thorough microbiological investigation of the sample for the presence of organisms other than H. pylori.

REFERENCES

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