Conferences and Reviews

Clinical Aspects of *Campylobacter jejuni* Infections in Adults

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***Campylobacter jejuni* is an almost ubiquitous, microaerophilic, gram-negative rod. Outbreaks have been associated with drinking raw milk or contaminated water and eating poultry. *Campylobacter jejuni* accounts for 3.2% to 6.1% of cases of diarrheal illness in the general population of the United States, and infected patients frequently present with abdominal pain and fever. Less frequently, *C jejuni* is responsible for bacteremia, septic arthritis, septic abortion, and other extraintestinal infections. Reactive arthritis, Reiter's syndrome, the Guillain-Barré syndrome, and pancreatitis may accompany or follow *C jejuni* enterocolitis. *Campylobacter jejuni* is an important cause of diarrheal illness and is a more commonly identified stool organism than *Salmonella* or *Shigella* species. Recurrent and chronic infection is generally reported in immunocompromised hosts.**

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Campylobacter jejuni is a common cause of enterocolitis. In this article I review the clinically relevant features of this frequent but often unrecognized cause of disease. In scope, this review is limited to *C jejuni* and *Campylobacter coli* infections in adults. I review the microbiology, epidemiology, manifestations, diagnosis, and treatment of the disease.*

Methods

Relevant articles were identified through a MEDLINE search from 1981 to early 1993 and from bibliographies of the selected articles. Articles selected for detailed review included original research articles, review articles, book chapters, and letters related to *C jejuni* and *C coli* infections in adults. A total of 312 articles were reviewed in detail. The reproducibility of methods for extracting data and selecting articles for this review was not assessed, but the results are similar to those of older published reviews.

Microbiology and Taxonomy

The word "campylobacter" is derived from the Greek *campylo* ("curved") and *bacter* ("rod"). *Campylobacter* species are slender, spirally curved, gram-negative rods. They have a characteristic corkscrew-like, darting motility by means of a single polar flagellum at one or both ends. *Campylobacter* species are, in general, microaerophilic and require oxygen concentrations of 3% to 15% and carbon dioxide concentrations of 3% to 5%. They produce catalase but do not ferment or oxidize carbohydrates.[1]

The campylobacters were originally classified with members of the genus *Vibrio* as *Vibrio fetus.* In humans, the current *Campylobacter fetus* is most commonly a bloodstream isolate associated with a febrile illness,[2] but it has been associated with such illnesses as thrombophlebitis, endocarditis, infected aortic aneurysm, septic arthritis, and osteomyelitis. In 1957 a subgroup of *Vibrio fetus* was described that was called the "related vibrios," which grew at 37°F and 42°F but not at 25°F, unlike *V fetus,* which grew at 25°F and 37°F but not 42°F.[3] In 1973 *V fetus* and the "related vibrios" were reclassified into the genus *Campylobacter* based on differences from the vibrios in the guanine and cytosine content and cell membranes.[4]

Of the thermotolerant campylobacters, *C jejuni* and *C coli* have been recognized as the most important causes of human disease. *Campylobacter jejuni* is a common cause of enteritis in humans; it almost always hydrolyzes hippurate in the laboratory. *Campylobacter coli* also causes human disease but is also frequently identified in swine and does not hydrolyze hippurate. *Campylobacter pylori* was recently reclassified into the genus *Helicobacter;* a number of other *Campylobacter* species have been described and are discussed elsewhere.[5]

For the purposes of this review, *C jejuni* and *C coli* will be referred to collectively as *C jejuni* unless otherwise specified because their clinical manifestations are similar and many earlier reports in the literature did not differentiate between the two species.

Epidemiology

Campylobacter jejuni is the cause of 3.2% to 6.1% of cases of diarrheal illness in the general population of the United States,[6-12] whereas species of *Salmonella* represent 2.1% to 3.6% and those of *Shigella* 0% to 2.9% of cases

*See also the editorial by B. Mishu, MD, and M. J. Blaser, MD, "Do You Have a *Campylobacter* in Your Future?" on pages 186-187 of this issue.

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ABBREVIATIONS USED IN TEXT

AIDS = acquired immunodeficiency syndrome
Ig = immunoglobulin

of similar illness.[8-12] Similar *C jejuni* isolation rates have generally been noted in Canada, Great Britain, Belgium, Australia, the Netherlands, and Sweden, although several series from developed countries have reported isolation rates of greater than 10% in patients with diarrheal illnesses.

Campylobacter jejuni is an important cause of travelers' diarrhea, accounting for 5.8% to 15.2% of cultured cases. It is infrequently identified in healthy controls in developed countries, but it was identified in stool specimens of 14.8% of healthy persons without diarrhea in southern India.[13]

A bimodal curve describes the age-specific isolation rate of *C jejuni,* with infants and young adults having the highest age-specific rates of isolation. In most series, rates of infection are highest in the summer months.[14,15] Most sporadic cases probably result from ingesting contaminated foods (such as poultry),[14-16] drinking raw milk,[9,16] drinking untreated water,[16,17] or contact with animal reservoirs.[15,16]

Campylobacter jejuni has been isolated from both fresh water and seawater, and several outbreaks of *C jejuni* enteritis have been associated with drinking contaminated water, including a large outbreak in Bennington, Vermont, in 1978 involving 2,000 to 3,000 people.[18]

Outbreaks have also been traced to unpasteurized[19,20] and improperly pasteurized milk. In some instances, *C jejuni* has been identified in rectal swabs of implicated dairy cattle[19,20] or from milk-processing equipment at an implicated dairy. Raw goat's milk has also been identified as the source of an outbreak. The consumption of cake (or cake icing), eggs, and beef has been associated with illness in a few outbreaks.

Outbreaks of *C jejuni* enteritis have been associated with the consumption of chicken and turkey. The handling of raw poultry has also been linked to human infection. Between 68% and 91% of live chickens and nearly 100% of commercial turkeys carry *C jejuni.* Cultures have been positive in 33% to 43% of poultry carcasses and 23.1% to 36% of retail chicken specimens. In contrast, *C jejuni* was cultured from only 1.6% of a large sampling of pork, beef, and lamb specimens at abattoirs and retail outlets.[21]

Less frequently, contact with dogs and cats has been reported as a source of infection in humans. In one study, 3,068 fecal, caecal, intestinal, and rectal specimens from animals were tested, and *C jejuni* was found in 66% of hogs, 43% of cattle, 23% of sheep, 35% of ducks, 17% of pigeons, 10% of other birds, 13% of dogs, 6% of hoofed zoo animals, and only 4% of reptiles.[22]

Person-to-person transmission is reported but appears to be infrequent.[14,18] In one case, laboratory-acquired *C jejuni* enteritis developed after a broth culture was accidentally spilled.[23] A case of "post-transfusion septicemia" developed in a patient with Waldenström's macroglobulinemia after he received blood from a person with serologic evidence of recent *C jejuni* infection.[24]

Clinical Manifestations

Enterocolitis

The common manifestations of *C jejuni* enterocolitis are presented in Table 1. Most frequently there is a self-limited diarrheal illness with associated abdominal pain that may be severe. Malaise, fever, anorexia, headache, nausea, vomiting, myalgia, and grossly bloody stools may also be present.[6,10-12,15,18-20,25-32] The diarrheal illness is similar to the enteritis caused by *Salmonella* or *Shigella* species. Death in association with *C jejuni* enteritis is rare.[33]

In experimental situations, infection has occurred after as few as 500 organisms were ingested.[34] The mean incubation period was estimated in one study to be 68 hours[29] and in another was 4 days.[35] The mean duration of excretion has been reported as 10.1 days,[36] and the duration of excretion is almost always less than 6 weeks in untreated subjects.

On presentation, some cases of *C jejuni* infection are indistinguishable from those of ulcerative colitis[6,30,37] or Crohn's disease,[6] and the illness has presented as a massive lower gastrointestinal tract hemorrhage. A number of patients have been taken to surgery because of abdominal pain with presumed appendicitis; only rarely has the appendix been inflamed and *C jejuni* thought to be the causative organism, however.[38,39]

The pathogenesis of *C jejuni* on the gut mucosa is incompletely understood. Some strains of *C jejuni* have been shown to produce a variable amount of enterotoxin that has similarity to the cholera toxin, but the pathogenesis of *C jejuni* on the gut mucosa is probably caused in large part by bacterial invasiveness. Immunohistochemical and ultrastructural evidence of invasion into the colonic mucosa has been found in a series of 22 patients, and an increased number of immunoglobulin (Ig) A- and IgM-containing plasma cells were noted in the colonic mucosa of infected patients.[40]

TABLE 1.—*Common Manifestations of* Campylobacter jejuni *Enteritis**

Symptom or Sign	*Patients Affected, %*
Diarrhea	75-100
Abdominal pain or cramps	55-97
Malaise	73-95
Fever	52-91
Anorexia	83-90
Headache	12-82
Nausea	30-76
Chills and sweats	29-75
Myalgia	19-69
Vomiting	19-55
Gross blood in stool	6-57

*From Drake et al,[6] Blaser et al,[10-12] Deming et al,[15] Vogt et al,[18] Taylor et al,[19] Kornblatt et al,[20] Stehr-Green et al,[25] Mentzing,[26] Tiehan and Vogt,[27] Porter and Reid,[28] Korlath et al,[29] Goodman et al,[30] Nolan et al,[31] and Gray and Pedler.[32]

On radiologic examination of patients with enteritis, findings vary from normal small bowel or colon to edema and ulceration of the small bowel and of the colon.[37,41,42] On sigmoidoscopy, there may be mucosal edema, exudate, granularity, and contact bleeding.[42-46] The findings of a rectal biopsy may be normal[40,44] or mimic ulcerative colitis, but most cases have histologic features of a nonspecific infectious colitis.[40,43,44,46,47]

Immunocompromised patients appear predisposed to chronic or recurrent *C jejuni* enterocolitis. Recurrent or chronic infection has been reported in patients with hypogammaglobulinemia and in persons with the acquired immunodeficiency syndrome (AIDS). Not surprisingly, a blunted or impaired antibody response to *C jejuni* has been noted in patients with recurrent or persistent infection.[47-49]

Other Gastrointestinal Manifestations

In a few cases, *C jejuni* has been isolated from the bile of patients who have undergone surgical therapy for cholecystitis. Acute pancreatitis has also been occasionally associated with *C jejuni* enteritis. The pathogenesis of acute pancreatitis associated with *C jejuni* enteritis is not understood, but as mentioned with the cases of cholecystitis, *C jejuni* can be found in the biliary tree of some patients.

Campylobacter jejuni has been reported as the cause of spontaneous bacterial peritonitis in a patient with cirrhosis and massive ascites due to alcoholism.[50] In one study, six of the authors' patients and seven others from the literature had *C jejuni* or *C coli* peritonitis associated with continuous ambulatory peritoneal dialysis.[51] These patients frequently had associated diarrhea that preceded the peritonitis in some cases.

Bacteremia and Recurrent Illness

Although a number of reports describe bacteremia in patients with *C jejuni* infection, bacteremia is generally not reported. In a summary of 8,320 human cases of infection with *Campylobacter* species, it was noted that only 0.4% of *C jejuni* isolates were from blood whereas 63% of *C fetus* isolates were from blood.[2] Others have shown in vitro that *C jejuni* is sensitive to normal human serum with an intact complement pathway whereas *C fetus* is relatively resistant.[52]

In normal subjects with *C jejuni* enterocolitis, serum IgA, IgM, and IgG antibodies to *C jejuni* rise during the second week after infection. A number of the reported cases of *C jejuni* bacteremia have occurred in immunocompromised patients such as persons with hypogammaglobulinema, recipients of corticosteroid therapy, patients with AIDS, and patients with malignant neoplasms. Bacteremia is substantially more common in AIDS patients with *C jejuni* infection than in otherwise healthy controls with *C jejuni* infection,[53] and several authors have noted that most cases of extraintestinal infection with *C jejuni*, including bacteremia, occur in patients at the extremes of age.

Rheumatic Manifestations

As much as 3% of cases of *C jejuni* enteritis may be followed by Reiter's syndrome or by a reactive arthritis.[54] The reactive arthritis is similar to that which follows venereal infection with *Chlamydia trachomatis* or enteric infection with *Shigella flexneri, Shigella dysenteriae, Salmonella typhimurium,* or *Yersinia enterocolitica.* The reactive arthritis associated with *C jejuni* is usually an oligoarticular arthritis that is asymmetric and most frequently involves the knees, ankles, or wrists.[54] The mean interval between the start of the enteritis and the arthritis is ten days.[54]

Campylobacter jejuni infection has only rarely been reported as the cause of septic arthritis, osteomyelitis, or prosthetic joint infection, and these cases all occurred in compromised hosts.

As listed in Table 1, nonspecific myalgias occur in 19% to 69% of cases of *C jejuni* enteritis.[26,32]

Neurologic Manifestations

An association is increasingly seen between *C jejuni* infection and some cases of the Guillain-Barré syndrome. In 1982, a case of the Guillain-Barré syndrome occurred in a 45-year-old man two weeks after an episode of culture-proven *C jejuni* enteritis.[55] Since then, a number of such cases have been reported in adults. These reports include several variants of the Guillain-Barré syndrome, and the severity of disease varies greatly.

In a series of 106 patients with the Guillain-Barré syndrome, 4 patients had known *C jejuni* infection preceding the development of their disease.[56] In a retrospective study of 56 Australian patients with the Guillain-Barré syndrome, 38% were noted to have serologic evidence of preceding *C jejuni* infection, but there was no evidence of previous infection in 27 control patients with other neurologic disorders or in 30 healthy controls.[57] More recently, it was reported that serologic evidence of recent infection with *C jejuni* was considerably more frequent in a series of 118 patients with the Guillain-Barré syndrome than in 56 controls with other neurologic diseases or in 47 healthy controls.[58] Finally, of 46 patients with the Guillain-Barré syndrome, 19 (41%) had evidence of *C jejuni* infection, and the majority of the *C jejuni* organisms identified in these patients belonged to Penner serogroup 19.[59]

The relationship between cases of the Guillain-Barré syndrome and preceding *C jejuni* enteritis is not well understood. High titers of IgG against GM_1 ganglioside were reported in two patients with acute axonal polyneuropathy following *C jejuni* infection, although no elevation of anti-GM_1 antibodies was noted in a subsequent but less severe case. Other investigators found that the serum of a patient who acquired the Guillain-Barré syndrome after *C jejuni* enteritis reacted to PO myelin-specific protein.[60]

Campylobacter jejuni meningitis has been reported in an adult who had previously undergone craniotomy for neuroblastoma. The illness was successfully treated with chloramphenicol.

Dermatologic Manifestations

An erysipelas-like cellulitis from *C jejuni* has been reported in three patients with hypogammaglobulinemia

and recurrent *C jejuni* bacteremia,[61] and cellulitis was reported in a patient with Bruton's (X-linked) agammaglobulinemia and in a patient with vascular disease.[62] Erythema nodosum is occasionally reported in patients with *C jejuni* enteritis.

Septic Abortion and Miscarriage

Campylobacter jejuni has been isolated from fetuses and from maternal blood in cases of septic abortion as well as from maternal blood in cases of early miscarriage. A patient with agammaglobulinemia suffered recurrent abortions that were thought to be due to *C jejuni* infection.[63]

Other Manifestations

Myocarditis has been reported in association with *C jejuni* enteritis. In the reported cases thus far, the disease has been self-limited.

Cases of the hemolytic-uremic syndrome have rarely been associated with *C jejuni* infection in adults.

Campylobacter jejuni is a rare cause of urinary tract infection. Blaser and co-workers have noted that the survival of *C jejuni* is brief in urine at 37°F.[64]

Diagnosis

A presumptive diagnosis of *C jejuni* enteritis may be made on the basis of an appropriate history and the presence of darting organisms on phase-contrast microscopy of a fresh stool specimen.[7] Fecal leukocytes have been reported in 66%[6] to 100%[65] of fecal wet mounts, and erythrocytes are also frequently present on a wet mount of stool.[65]

Campylobacter jejuni may be isolated from stool by passing the stool specimen through a filter (0.45 μm Millipore filter), which removes larger bacteria present in the stool and allows the relatively small campylobacters to pass onto the medium, as originally described in 1972.[66] In 1977 the use of selective media for isolating *Campylobacter* species, which inhibits the growth of other bacteria and yeast, was described.[67] Other isolation media have been outlined and include Preston, modified CCDA,* and Butzler media.[68] One author noted a nearly twofold increase in *Campylobacter* isolation rates when using the filtration method rather than standard plating on selective media.[69] DNA probes have been developed for *C jejuni* and *C coli* that may become clinically important.

Serotyping has been epidemiologically useful in some outbreaks of *C jejuni* enteritis. Methods of serotyping *C jejuni* strains that rely on heat-stable and heat-labile antigens have been described.[70,71]

Therapy

Because *C jejuni* enterocolitis is generally a self-limited illness, most patients require no more than supportive treatment. Antimotility agents have been shown to impede the resolution of infection and to increase the need for antimicrobial therapy.[31] When antibiotic treatment is necessary, several authors have suggested erythromycin as the drug of choice for *C jejuni* enterocolitis. Most authors believe that erythromycin therapy does not alter the clinical course of enteritis caused by *C jejuni*; unlike with *Salmonella* species enteritis, antibiotic treatment shortens the fecal excretion of the organism rather than prolonging it.

Erythromycin resistance in *C jejuni* is chromosomally mediated and involves the alteration of ribosomes so that erythromycin binding is not favored. Resistance has been noted in 0.5% to 8.4% of organisms from developed countries; however, 65% of those isolated from a series in Thailand were erythromycin-resistant.[69] Erythromycin resistance is substantially more common with *C coli* than with *C jejuni*.

Campylobacter jejuni is usually sensitive to gentamicin, tetracycline, doxycycline, furazolidone, nitrofurantoin, and ciprofloxacin. Ciprofloxacin, gentamicin, doxycycline, and tetracycline have been suggested as alternative agents to erythromycin, but quinolone resistance has emerged. The role that newer macrolide drugs might play in the treatment of *C jejuni* infection is not yet known. Intraperitoneal aminoglycoside administration and oral erythromycin have been suggested for the treatment of *C jejuni* peritonitis associated with continuous ambulatory peritoneal dialysis.[51]

Preventive measures such as careful preparation and thorough cooking of poultry, pasteurization of milk, and chlorination of water are important in preventing infection with *C jejuni* and *C coli*.

Conclusions

Campylobacter jejuni and *C coli* are common causes of self-limited enterocolitis, which usually presents with diarrhea and abdominal pain. Outbreaks have been associated with the consumption of raw milk, surface water, and poultry. Extraintestinal manifestations such as reactive arthritis, Reiter's syndrome, pancreatitis, or the Guillain-Barré syndrome may occur with *C jejuni* infection, and recurrent or chronic infection may be found in immunocompromised patients. Erythromycin is regarded as the antibiotic of choice when supportive therapy alone is inadequate; erythromycin-resistant strains occur, however, and gentamicin or ciprofloxacin have been suggested as alternative agents.

*Modified CCDA is a selective blood-free agar base containing nutrient broth, charcoal, casein hydrolysate, sodium desoxycholate, ferrous sulfate, sodium pyruvate, and agar.

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