Campylobacter Infections in Children

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Education Gap

Campylobacter is one of the 2 most common causes of foodborne illness in the United States. It most commonly occurs in children younger than 5 years of age. *Campylobacter* species can cause a wide range of syndromes, from asymptomatic infections to severe systemic infections.

Objectives

After completing this article, readers should be able to:

1. Recognize that *Campylobacter* is a common cause of foodborne illness in the United States and internationally.
2. Understand the indications for testing and the treatment of *Campylobacter* infection.

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ABBREVIATIONS

CDC	Centers for Disease Control and Prevention
CIDT	culture-independent diagnostic testing
GBS	Guillain-Barré syndrome
HIV	human immunodeficiency virus
IBD	inflammatory bowel disease
Ig	immunoglobulin

INTRODUCTION

Campylobacter species are an important cause of infection throughout the world, especially in young children. *Campylobacter jejuni* is the most common *Campylobacter* species in the United States, where it is 1 of the top 2 causes of foodborne illness. (1) Other important species include *Campylobacter upsaliensis*, *Campylobacter lari*, and *Campylobacter fetus*, which can cause serious systemic infections in all age groups, particularly in immunocompromised patients.

Campylobacter species infections cause gastroenteritis and typically present with diarrhea that may or may not be bloody, emesis, and abdominal pain. *Campylobacter* infection in children can mimic intussusception, appendicitis, or inflammatory bowel disease (IBD). Immune-mediated complications of *Campylobacter* infection include reactive arthritis and Guillain-Barré syndrome (GBS). *Campylobacter* infection is a nationally notifiable disease, and confirmed cases should be reported through the Centers for Disease Control and Prevention (CDC) Foodborne Diseases Active Surveillance Network.

Most *Campylobacter* infections are mild and self-limited and require only supportive care, although some may lead to severe dehydration. Serious infection or infections in immunocompromised hosts may benefit from treatment with macrolide antibiotics. Increasing rates of fluoroquinolone resistance have been observed in *Campylobacter* isolates. (2)

The largest source of *Campylobacter* infections is animals, especially wild and domestic birds. Strategies to prevent *Campylobacter* infection include thoroughly cooking poultry, preventing cross-contamination with other foods, properly

chlorinating water and pasteurizing milk, and practicing hand hygiene after interacting with animals.

MICROBIOLOGY

The *Campylobacter* genus is composed of gram-negative, motile, curved or spiral-shaped bacilli. The name *Campylobacter* is derived from Greek and Latin and means "curved rod." As of 2014, the genus included 26 species, approximately half of which cause disease in humans. (3) The organisms that are most commonly associated with infection in humans are *C jejuni* and *Campylobacter coli*. In 2015, 88% of all isolates that were speciated were *C jejuni*. (1) *C jejuni* contains 2 subspecies: *C jejuni* subspecies *jejuni* (typically referred to as *C jejuni*), which is the most commonly isolated cause of *Campylobacter* infection in the United States, and *C jejuni* subspecies *doylei*, which is much rarer. Other *Campylobacter* species that cause infection in humans are *C fetus, C upsaliensis, C lari*, and *Campylobacter hyointestinalis*. This review focuses on *C jejuni* and *C coli*, with a brief discussion of other, less common species at the end.

PATHOGENESIS

There are multiple organisms and host factors that enable *Campylobacter* infection in humans. Studies in healthy volunteers as well as observational data from outbreaks have demonstrated that the inoculum required to cause *Campylobacter* enteritis in humans can be as low as 500 organisms. (4)(5)(6) Disruption of the gastric acid barrier allows for pathogenic flora, such as *Campylobacter*, to survive and flourish. Therefore, patients with reduced gastric acidity, including those receiving proton pump inhibitors, may be at higher risk for infection with *Campylobacter*. (7)(8)(9)(10) The incubation period is relatively short, ranging from 1 to 7 days, with an average of 3 days. Higher inocula may result in shorter incubation periods. Infection is typically established in the distal ileum and colon and initially causes a noninflammatory diarrhea. This is followed by a locally invasive stage that leads to cell damage and inflammation that can present with dysentery, which is occasionally followed by translocation across the intestinal epithelium, causing lymphadenitis and extraintestinal infection. Histologically, infection is identical to salmonellosis or shigellosis and is marked by acute mucosal inflammation and edema, which can include infiltration of the lamina propria and crypt abscess formation. (11)(12)

The exact mechanisms of infection are not completely understood, but several virulence factors have been identified, including flagella, plasmids, adhesins, and chemotactic factors. (13) Initial infection is established when bacteria attach to intestinal epithelial cells via fimbriaelike filaments. Colonization of the gastrointestinal tract is then facilitated by flagella and chemotactic factors. *Campylobacter* flagellins do not seem to provoke proinflammatory cytokines, which may allow *Campylobacter* to evade innate immune response, differentiating it from other intestinal pathogens, including *Salmonella*. (14) There are a variety of other surface proteins and adhesins that facilitate colonization and invasion of intestinal epithelial cells. Some isolates of *Campylobacter* contain a high-molecular-weight plasmid, pVir, that has been associated with bloody stools and is thought to contribute to invasiveness. (15)

The immune response to *Campylobacter* infection seems to be primarily humoral. *Campylobacter*-specific serum immunoglobulin (Ig) A level rises rapidly during the first 2 weeks after infection and then declines slowly over the next month. The IgM and IgG antibody levels rise more slowly and peak 2 to 3 weeks after development of symptoms. (16) Prolonged and severe infection has been reported in patients with hypogammaglobulinemia. (17) The role of cellular immunity in *Campylobacter* infection is less well defined, but it does seem to be important because patients with human immunodeficiency virus (HIV) are also at risk for more severe and prolonged disease as well as higher rates of extraintestinal infection. (18) In immunocompetent patients, infection with *Campylobacter* provides some immunity against future infection, especially infection with the same strain.

EPIDEMIOLOGY

Campylobacter is a leading cause of acute diarrhea domestically and internationally. In the United States, *Campylobacter* and *Salmonella* are the 2 most common causes of foodborne illness. In 2016 the incidence of *Campylobacter* enteritis confirmed by culture or culture-independent diagnostic tests (CIDTs) was 17.4 infections per 100,000 persons, making it the most commonly identified cause of foodborne illness in the United States. *Campylobacter* has been tracked through the Foodborne Diseases Active Surveillance Network since 1996 and has been a notifiable disease since 2015. Most *Campylobacter* infections are mild and self-limited, but in 2016, 20% of reported infections resulted in hospitalization and 26 attributable deaths were identified (mortality, 0.3%). (19)

In the United States, the incidence of infection is highest in children younger than 5 years of age, but there is also a secondary peak in young adults. Infection rates are highest

in the summer, and most infections are domestically acquired, although *Campylobacter* enteritis is also seen in returning international travelers. (1) In developing countries, the infection is hyperendemic, and symptomatic infection occurs almost exclusively in infants and young children, who can be infected repeatedly. Subsequent infections tend to be asymptomatic, making symptomatic disease rare in older children or adults. (20)(21)

Campylobacter enteritis is usually sporadic; only 0.4% of identified infections in 2015 were associated with an outbreak. (1) Transmission to humans occurs through ingestion of contaminated food or water or by direct contact with fecal material from infected people or animals. Animal reservoirs, including wild and domestic birds and other animals, are the most common source of *Campylobacter* infections. Multiple studies have shown that most broiler chicken flocks, 60% to 80%, may be contaminated with *Campylobacter* at slaughter. (22) The main vehicle of transmission to humans is improperly cooked poultry; 1 study suggested that 48% of *Campylobacter* infections are attributable to poultry exposure. (23) Direct acquisition of infection from animals is less common and is usually associated with occupational exposure to poultry. Infection has been reported in association with domestic pets, especially puppies and kittens, that have *Campylobacter* diarrhea. (23) In the fall of 2017 an outbreak of multidrug-resistant *Campylobacter* was linked to puppies sold through a national pet store chain. (24) Person-to-person transmission is rarer, but caretakers of children or other individuals in diapers are at increased risk. Sexual transmission has also been reported, especially in men who have sex with men. (25)

Outbreaks have been reported in association with the distribution of contaminated water or milk. *Campylobacter* can survive in water or milk for many weeks at low temperatures. Because *Campylobacter* is nearly ubiquitous in the environment, decontamination is the most important strategy to prevent transmission. Waterborne outbreaks have occurred in association with unchlorinated water or failed chlorination systems, and milk-associated outbreaks have been connected to raw milk or failures in the pasteurization process. (26)(27)(28)(29) Importantly, milk-borne outbreaks have been associated with raw milk that had passed routine testing because *C jejuni* can sometimes be difficult to culture. (29)

CLINICAL PRESENTATION

The most common illness caused by *C jejuni* and *C coli* is gastroenteritis. Children present with diarrhea and abdominal pain, often accompanied by vomiting and fever. Infection can result in significant dehydration. Bloody stools are also seen in at least half of infected children. (30)(31) Fevers can be prominent, and seizures, meningismus, and encephalopathy have all been reported in association with *Campylobacter* infection. (26)(32) Bacteremia is rare and is seen primarily in patients who are immunocompromised. (33)(18)

Perinatal *C jejuni* infection is rare but has been described. It can result in abortion, premature labor, and/or neonatal septicemia and meningitis. Symptomatic gastroenteritis in neonates is also possible, which may present with only grossly bloody stools or fever. Infection is generally acquired perinatally from mothers that shed *Campylobacter* either symptomatically or asymptomatically. Outbreaks in neonatal nurseries due to nosocomial spread have been reported. (34)

Campylobacter infection can also mimic other gastrointestinal illnesses. Infants with *Campylobacter* enteritis may present with only bloody stools and vomiting without fever, which can be mistaken for intussusception. In older children, acute *Campylobacter* ileocecitis can cause severe right lower quadrant pain without diarrhea mimicking appendicitis. (35)(36) *Campylobacter* enteritis most commonly progresses from the small bowel distally, but rarely patients with severe infection may present with only colitis and bloody diarrhea that can be confused with IBD. (37) Imaging can be helpful in excluding other intra-abdominal processes, such as intussusception. The acute inflammatory changes of *Campylobacter* infection observed on histologic analysis should help differentiate it from the chronic changes of IBD. It has also been suggested that *Campylobacter* infection may play a role in the development of IBD. (38)

Although the manifestations can be variable, *Campylobacter* gastroenteritis is usually relatively mild and self-limited, with symptoms lasting 24 to 48 hours in most patients. Excretion of the organism can last much longer than clinical symptoms. On average, shedding lasts 2 to 3 weeks, but it can range from 3 days to several months. (31) Shedding tends to be prolonged in younger children and patients with immunodeficiencies. Rarely, symptoms may persist for several weeks or relapse may occur after initial resolution. Chronic or recurrent infection is more common in immunocompromised patients, who can develop a prolonged relapsing syndrome that mimics IBD. This has been described in particular in patients with HIV, in whom long-term carriage and recurrent enteritis can be associated with bacteremia, fever, and debilitating illness. (18)(33)(39)(40)(41) The incidence of *Campylobacter* infection remains higher in patients with HIV than in the general population, but it has decreased significantly since the introduction of highly active antiretroviral therapy. (41)

COMPLICATIONS

Early Onset

Extraintestinal manifestations without enteritis are rare, but septic arthritis, bursitis, osteitis, and soft tissue infections have been reported. Acute extraintestinal complications of enteritis include cholecystitis, peritonitis in patients with peritoneal dialysis, septic pseudoaneurysm, pericarditis, and myocarditis. Erythema nodosum, glomerulonephritis, hemolytic anemia, IgA nephropathy, postinfectious irritable bowel syndrome, and intestinal perforation have all been described as well.

Late Onset

Late-onset immune-mediated complications of *Campylobacter* infection, including arthritis and GBS, have been well described.

Reactive arthritis after *Campylobacter* enteritis can occur 3 to 40 days (mean, 11 days) after the onset of diarrhea. It is usually oligoarticular and asymmetrical, and it predominantly affects the knees. It is more common and possibly more severe in patients with HLA-B27 phenotype. The development of arthritis does not seem to be related to the severity of the initial illness. Symptoms generally last for up to 21 days, and most patients have spontaneous recovery within 6 months without long-term sequelae. The arthritis is reactive, not infectious; synovial fluid is sterile. Arthritis occurs in up to 7% of patients, but as many as 20% of patients report arthralgia. (42)

Both serologic studies and culture surveys suggest that *Campylobacter* infection is the most commonly identified cause of GBS. Thirty percent to 40% of cases of GBS develop in patients who were infected with *Campylobacter* 10 to 14 days earlier. In the United States, 1 in 1,000 patients with *Campylobacter* enteritis goes on to develop GBS. Some studies have suggested that GBS associated with *Campylobacter* infection may have a poorer prognosis than GBS associated with other etiologies. (43)

The primary mechanism for *Campylobacter*-associated GBS is molecular mimicry. The *C jejuni* lipopolysaccharide resembles GM1 ganglioside on peripheral nerve myelin. Infection can lead to the development of cross-reacting antibodies that cause nerve damage. The Miller Fisher variant of GBS, with more prominent cranial nerve involvement resulting in ophthalmoplegia, areflexia, and ataxia, is also more common in patients with *Campylobacter* infection. It is thought to be caused by antibodies that cross-react with *Campylobacter* lipopolysaccharide and ganglioside GQ1b in cranial nerve myelin. (44) Certain *Campylobacter* serotypes, especially Penner O19 and O41, seem to be more commonly associated with GBS. (43) There may be factors other than molecular mimicry that also play a role in the development of *Campylobacter*-associated GBS. Additional host factors seem to be important; some patients develop antibodies to GM1 ganglioside but do not develop neurologic symptoms. There may be an association between HLA types and the development of *Campylobacter*-associated GBS. (45)

DIAGNOSIS

Campylobacter enteritis is often clinically indistinguishable from other viral or bacterial gastroenteridites. Diagnostic testing is not always indicated for children who present with acute diarrheal illnesses, with or without fever or vomiting, because determining the cause often does not change clinical management. However, Infectious Diseases Society of America guidelines suggest testing patients with fever or bloody diarrhea or others in whom treatment may be indicated, including anyone with immunodeficiencies. In addition, organism-specific diagnosis can be valuable for the management of outbreaks. Blood cultures are recommended in infants younger than 3 months of age, those with signs of sepsis or systemic manifestations, and immunocompromised patients. (46)

Stool culture is the gold standard for the identification of *Campylobacter* species. Most laboratories specifically look for *Campylobacter* in standard stool cultures, but it can be difficult to isolate. *Campylobacter* grows best on media containing selective antibiotics and in microaerobic conditions with 5% to 10% oxygen, 1% to 10% carbon dioxide, and some hydrogen. *C jejuni* and *C coli* grow best at 107.6°F (42°C). *Campylobacter* is identified by its characteristic appearance as a comma- or spiral-shaped Gram-negative bacillus as well as oxidase and catalase production. Species-level identification is not typically performed, and differentiation of *C jejuni* from *C coli* is not usually necessary for management. Speciation and strain typing can be helpful for epidemiologic purposes or when species other than *C jejuni* or *C coli* are suspected. This typing is typically performed at reference laboratories.

Use of CIDTs, including nucleic acid amplification tests, is increasing. (19) These tests are generally more sensitive and have faster turnaround times than traditional culture-based diagnostics. Reverse transcriptase polymerase chain reaction identifies *Campylobacter* from stool 20% to 40% more frequently than culture-based methods. (47) However, because these tests identify the presence of nucleic acid rather than viable organisms, the clinical significance is not

always clear. The identification of multiple pathogens is not uncommon and can be difficult to interpret. In addition, CIDTs cannot be used to identify antibiotic susceptibility patterns. Unlike organisms such as *Shigella* and *Salmonella*, cultures of *Campylobacter* often are not performed automatically when the organism is detected by CIDT. Antibiotic resistance to quinolones and tetracyclines is common in *Campylobacter* isolates, so if treatment is warranted and there is concern for resistance, cultures can still be beneficial after identification by CIDT. (2)

Serologic testing can be used to detect recent *Campylobacter* infection in patients with reactive arthritis or GBS who have negative stool studies. Serologic studies are not helpful in the diagnosis of acute *Campylobacter* infection.

If *Campylobacter* infection is suspected or confirmed, it is important to attempt to identify the source of infection, mostly to prevent others from becoming infected. Families should be asked about exposures, including consumption of raw milk or contaminated drinking water, undercooked meats or poultry, contaminated fruits or vegetables, and contact with animals (wild and domesticated fowl, puppies, kittens) or their feces. Travel exposure may also be relevant, although most infections are acquired domestically.

MANAGEMENT

Campylobacter infection typically results in a mild, self-limited enteritis that requires only supportive care. Adequate rehydration, either orally or intravenously, is the mainstay of acute management. Antimotility agents should be avoided due to prolongation of symptoms. (48) Several trials have suggested that probiotics may decrease diarrhea duration and stool frequency in immunocompetent children with gastroenteritis of any cause, although the effect seems to be greater in viral than bacterial gastroenteritis, and the optimal formulation remains unknown. This benefit also has to be weighed against case reports of bacteremia or fungemia with molecularly matched isolates in critically ill or immunocompromised patients who received probiotics. (49) In countries with a high prevalence of zinc deficiency or malnutrition, zinc supplementation reduces the duration of diarrhea in children 6 months to 5 years of age with acute diarrhea. (50)

A meta-analysis of 11 randomized controlled trials of antibiotic treatment versus placebo for the treatment of *Campylobacter* infections in children and adults showed that antibiotic treatment reduced the duration of symptoms by 1.3 days and the duration of *Campylobacter* excretion in stool. However, given the self-limited nature of most *Campylobacter* infections, the relatively modest benefit to treatment with antibiotics, and the risk of development of antibiotic resistance, treatment of uncomplicated infection is usually not recommended. (51) Treatment is recommended in patients with severe disease, which includes those with bloody stools, high fever, extraintestinal infection, worsening or relapsing symptoms, or prolonged symptoms that exceed 1 week. It is also recommended for patients with uncomplicated infection who are elderly, pregnant, or immunocompromised due to their risk of severe disease. Both GBS and other late-onset complications of *Campylobacter* infections are immune-mediated and usually develop once symptoms of acute infection have resolved. Therefore, antibiotics are not routinely included in the management of these *Campylobacter*-associated syndromes unless patients otherwise meet the criteria for treatment with antibiotics, including prolonged or severe symptomatic infection or immunocompromise.

In patients with uncomplicated infection who merit antibiotic therapy, the recommended treatment is azithromycin 10 mg/kg per day for 3 days or erythromycin 40 mg/kg per day in 4 divided doses for 5 days. Treatment usually eradicates *Campylobacter* from the stool within 2 to 3 days. (52) A longer course (7–14 days) may be necessary in complicated infections or in patients who are immunocompromised. Carbapenems are appropriate empirical treatment in patients who are severely ill and unable to tolerate oral treatment, although it is important to complete susceptibility testing to verify carbapenem activity. Treatment for bacteremia should be tailored to available susceptibility testing. Although *C jejuni* and *C coli* are usually sensitive in vitro to clindamycin, tetracyclines, and chloramphenicol, data are lacking to support their use clinically. *Campylobacter* species are inherently resistant to trimethoprim and β-lactam antibiotics, including penicillin and cephalosporins, so these should be avoided.

Resistance

Fluoroquinolones may also be effective treatment for *Campylobacter* infection, but resistance is increasingly common, limiting the effectiveness of these agents for this indication. In the past, this resistance was thought to be, in part, attributable to quinolone use in poultry feeds, but this practice has been banned in the United States since 2005. United States data from 2014 indicated that ciprofloxacin resistance was present in 27% of *C jejuni* isolates and 36% of *C coli*. (2) Resistance is significantly higher outside the United States and is important to

consider in travelers. (53) Fortunately, macrolide resistance remains rare in the United States.

PREVENTION

Prevention of *Campylobacter* infection should focus on transmission from animals, milk or waterborne disease, and person-to-person transmission. Contaminated poultry is the most common source of *Campylobacter* infection, so thoroughly cooking poultry and avoiding cross-contamination with other foods should be emphasized. *Campylobacter* is killed by heat, so all meat, but especially chicken, should be cooked to proper temperature. Hand hygiene and thorough cleansing of cutting boards and utensils after contact with raw poultry as well as avoiding contact between raw poultry and other foods are also important. Hand hygiene is also important after contact with the feces of dogs and cats, especially puppies and kittens, where high rates of *Campylobacter* carriage are present. Unpasteurized milk should be avoided, and water should be chlorinated.

People with diarrhea should be excluded from food handling and the care of patients in hospitals and child care centers. They can resume work if infected but asymptomatic, as long as proper hand hygiene is practiced.

Infants and children in diapers who are found to have *Campylobacter* enteritis should be excluded from child care centers until symptoms have resolved. Treatment of these children with azithromycin or erythromycin can be considered to limit spread. Contact precautions should be used for children who are hospitalized with symptomatic *Campylobacter* enteritis. (52) Many institutions use syndromic contact isolation for any patient with diarrhea, regardless of the cause.

Previous infection with *Campylobacter* decreases the risk of future symptomatic infection but is not completely protective, so patients with previous infections should still use the prevention measures detailed previously herein.

OTHER *CAMPYLOBACTER* SPECIES

Campylobacter upsaliensis

Similar to *C jejuni* and *C coli*, *C upsaliensis* usually causes gastroenteritis, which may present as bloody diarrhea in 25% of patients, and occasionally bacteremia. It can also occasionally cause vomiting but is usually milder than *C jejuni* infection. (54) Bacteremia is primarily seen in malnourished or immunocompromised patients. (55) It is more common in children than adults. *C upsaliensis* was initially isolated from dogs, and they are considered to be its primary reservoir. Infection may be associated with close contact with dogs.

C upsaliensis is catalase negative or catalase weak and has a different antibiotic susceptibility pattern than *C jejuni*. It is susceptible to antibiotic agents usually used in *Campylobacter*-selective media and is, therefore, not detected in cultures using these media. (56) Consequently, the true prevalence of *C upsaliensis* is not clear, but it is the third most common *Campylobacter* species (after *C jejuni* and *C coli*) recovered in foodborne infections in the United States. (1)

Data are limited regarding treatment of *C upsaliensis*. Similar to *C jejuni* and *C coli*, it is usually treated with macrolides, although resistance has been reported in some studies. Some isolates are susceptible to gentamicin, azithromycin, erythromycin, meropenem, cephalosporins, fluoroquinolones, tetracyclines, and aminoglycosides. (57)

Campylobacter lari

C lari is the fourth most commonly isolated *Campylobacter* species in foodborne illnesses. It is common in chickens but relatively rare in humans. It accounts for less than 1% of *Campylobacter* isolates from humans and is predominantly seen in immunocompromised patients and neonates. (58) It can be grown on conventional *Campylobacter*-selective agars under similar conditions as *C jejuni*. Macrolides are generally effective for treating *C lari* infections.

Campylobacter fetus

C fetus is rare but tends to cause more serious illness than *C jejuni or C coli*. It causes bloodstream infections more commonly than gastroenteritis and is more common in patients at the extremes of age. In 2015 only 0.1% of laboratory-confirmed cases of *Campylobacter* in the United States were due to *C fetus*, but studies have shown that it is responsible for 19% to 53% of *Campylobacter* bacteremia. (1)(33)(59) It may not be identified in stool cultures because it grows best at 77.0°F (25°C) and 98.6°F (37°C) rather than at 107.6°F (42°C) like *C jejuni*. (60)

Three syndromes associated with *C fetus* bloodstream infection have been described: first, isolated bloodstream infection that is self-limited or rapidly responds to antibiotic therapy; second and most commonly, bloodstream infection associated with a focal infection such as meningitis, pneumonia, endocarditis, or thrombophlebitis; third, chronic relapsing/remitting bacteremia, seen most commonly in immunocompromised patients. Perinatal infection has also been described and can be associated with spontaneous abortion, preterm labor, and neonatal septicemia or meningitis. Perinatal infection with *C fetus* is rare but tends to be more severe than with *C jejuni*.

Although *C fetus* is often susceptible to ampicillin and gentamicin, carbapenems are preferred first-line agents pending susceptibility testing. *C fetus* is typically susceptible

to ampicillin, extended-spectrum cephalosporins, meropenem, imipenem, aminoglycosides, and erythromycin. (61)

Summary

- On the basis of strong evidence, *Campylobacter* is a common cause of acute diarrhea throughout the world, and *Campylobacter jejuni* is the most commonly isolated *Campylobacter* species. (1)
- On the basis of strong evidence, most *Campylobacter* infections result in a mild, self-limited enteritis, but *Campylobacter* can also cause invasive disease, including bacteremia. *Campylobacter* is also the most commonly identified cause of Guillain-Barré syndrome and can cause reactive arthritis.
- Based on strong clinical evidence, stool culture remains the gold standard for the diagnosis of *Campylobacter* infection, but culture-independent diagnostic testing is also sensitive. On the basis of consensus, species-level identification of *Campylobacter* isolates is usually not necessary for clinical management.
- *Campylobacter* is a nationally notifiable disease. Any confirmed cases should be reported through the Centers for Disease Control and Prevention Foodborne Diseases Active Surveillance Network.
- Based on strong evidence, antibiotic treatment of *Campylobacter* infections results in a modest reduction of duration of infection (1 day) and a reduced duration of shedding. However, based on consensus, treatment is not recommended for uncomplicated infection in immunocompetent hosts due to the mild nature of infection, modest benefit, and risk of development of antibiotic resistance. Treatment with azithromycin or erythromycin is recommended for severe infections or infections in immunocompromised hosts.
- Based on an abundance of evidence, important practices for the prevention of *Campylobacter* infection are thoroughly cooking poultry, avoiding cross-contamination between poultry and other foods, chlorination of water, pasteurization of milk, and observation of hand hygiene after contact with animals and their feces.

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1. A 7-year-old previously healthy girl presents to your office with a 3-day history of watery diarrhea. The mother reports that the child has also had several episodes per day of nonbloody, nonbilious emesis and some abdominal pain. Two other immediate family members have had similar symptoms for the past few days, including the father and the child's older brother. On further history, the mother tells you that they visited relatives in Maine approximately 2 weeks ago, swam in a lake, and went to a petting zoo. They have 2 healthy dogs in the home. The family members consume only pasteurized milk and cheeses but the child does drink a lot of milk both at home and at school. The family went to a new restaurant approximately 3 days before the onset of symptoms where they shared several dishes, including chicken and steak. Given the patient's recent exposures, you decide to test her stool for enteric pathogens, and testing returns positive for *Campylobacter*. Which of the following is the most likely mode of transmission of *Campylobacter* in this child?
   A. Adult pets such as dogs and cats.
   B. Exposure to lake water.
   C. Improperly cooked poultry.
   D. Pasteurized milk and cheeses.
   E. Petting zoos.
2. A 5-year-old girl is admitted to the hospital with dehydration after 2 days of nonbilious, nonbloody emesis, several episodes of bloody diarrhea, and fever today to 101.5°F (38.6°C) after drinking raw milk. During the hospital stay she is found to be positive for *Campylobacter jejuni*. Her parents are very concerned about her condition and ask you what some of the complications of this infection are. Which of the following would be considered a late-onset complication of *Campylobacter* infection?
   A. Encephalitis.
   B. Guillain-Barre syndrome (GBS).
   C. Hemolytic anemia.
   D. Immunoglobulin A nephropathy.
   E. Myocarditis.
3. A previously healthy 12-year-old girl is brought to the clinic with new-onset ataxia. She reports a gradual onset of the ataxia and lower extremity weakness over the course of the past 2 days. She is afebrile and her vital signs are stable. She is found to be areflexic in her lower extremities. A head computed tomographic scan is performed and the results are normal. She reports a history of nonbloody diarrhea, emesis, and abdominal pain that lasted 4 days approximately 2 weeks earlier after her family visited a farm. You are concerned that she might have *Campylobacter*-associated GBS. You discuss this entity with the family. Which of the following best describes *Campylobacter*-associated GBS?
   A. *Campylobacter fetus* is the most common type of *Campylobacter*-associated GBS.
   B. *Campylobacter* infection is the most commonly identified cause of GBS.
   C. Carries a better prognosis than GBS associated with other etiologies.
   D. Considered to be an early-onset complication of *Campylobacter*.
   E. Its primary mechanism is direct invasion of the nerve roots.
4. A 13-year-old boy is brought to the clinic with a 3-day history of watery, nonbloody diarrhea. The patient and his father recently returned from a camping trip in North Carolina 1 week earlier where they believe they may have had some undercooked poultry. The child has had 2 or 3 episodes of watery stools per day and 1 episode of nonbilious, nonbloody emesis this morning. He reports mild abdominal pain that is relieved after having a bowel movement. He has a poor appetite, decreased oral intake, and slightly decreased urine output. On physical examination the patient is in no apparent distress. His temperature is

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99.5°F (37.5°C); heart rate, 103 beats/min; respiratory rate, 20 breaths/min; and blood pressure, 90/60 mm Hg. His abdomen is mildly tender in the periumbilical area, but there are normal bowel sounds and no masses or hepatosplenomegaly. The remainder of his examination findings are within normal limits. Which of the following is the next best step in the management of this patient?

A. Antimotility agent.
B. Empirical treatment with azithromycin.
C. Stool sample for culture.
D. Trial of an oral rehydration solution.
E. Zinc supplementation until diarrhea resolves.

5. A 3-year-old child is brought to the emergency department (ED) with a 5-day history of watery stools that have now turned grossly bloody, with up to 12 stools reported on the day of admission. The parents report that the child had 4 or 5 episodes of emesis and complained of abdominal pain that worsened today along with a fever (to 103.6°F [39.8°C]), prompting them to come to the ED. On further history, the parents report that they went to a local farm recently and drank raw milk. You have seen several other patients in the past few days who visited this same farm with symptoms of gastroenteritis. Two of the patients had positive stool cultures for *Campylobacter*. On examination the child is ill appearing, pale, and lethargic. His temperature is 104.2°F (40.1°C), heart rate is 150 beats/min, respiratory rate is 40 beats/min, and blood pressure is 75/35 mm Hg. The child's lips are dry. His lungs are clear to auscultation and his heart sounds are normal, but he is tachycardic. On abdominal examination, he moans on palpation of his abdomen. Bowel sounds are sluggish throughout. His extremities are cool and his capillary refill is 4 to 5 seconds. Immediately, intravenous access was obtained and the child was given a normal saline bolus and was admitted to the PICU, where empirical antibiotics were started. Which of the following is the most appropriate choice of empirical antibiotic therapy?

A. Ampicillin.
B. Ceftriaxone.
C. Ciprofloxacin.
D. Meropenem.
E. Trimethoprim-sulfamethoxazole.

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