Acute Infectious Gastrointestinal Disorders

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Patients with acute gastrointestinal (GI) infections can present with a number of symptoms, including odynophagia, dysphagia, dyspepsia, nausea, vomiting, diarrhea, and abdominal pain as well as systemic symptoms of fever, chills, and general malaise. However, these symptoms are among the most common presenting complaints encountered in the emergency department (ED) and are frequently seen in many other medical and surgical problems. The challenges for the emergency medicine physician are to recognize acute GI infections and to identify which patients require diagnostic testing and disease-specific treatment versus supportive care. Most patients with GI infections require only supportive care and appropriate counseling regarding ways to avoid spread of infection (eg, food-handling and hand-washing).

Acute GI infections may have a viral, fungal, bacterial, or parasitic etiology. GI infectious illness may result following consumption of contaminated food or water, fecal-oral transmission of pathogenic agents, overgrowth of normal bacterial flora due to antibiotic use, or colonization by *Helicobacter pylori*. Patients with compromised immune systems are particularly susceptible to GI infections.

**INTRODUCTION**

**ESOPHAGITIS**

Viruses, fungi, or bacteria can infect the esophagus. The symptomatology for each of these infections may be similar. History and physical examination findings may help to differentiate between the causative agents.

**VIRAL**

Viral esophagitis is most commonly caused by herpes simplex virus (HSV), varicella-zoster virus (VZV), or cytomegalovirus (CMV). Esophagitis due to HSV-1 or VZV may be seen in immunocompetent patients, while HSV-2 and CMV esophagitis are rarely seen in these patients. HSV-2, VZV, and CMV infections occur more frequently in the immunocompromised. Symptoms of viral esophagitis include odynophagia, dysphagia, nausea, vomiting, fever, chills, and acute chest pain. In severe cases of all types of esophagitis, hematemesis can occur. The physical examination may be normal, or there may be disease-specific symptoms such as herpetic vesicles on the nose, lips, and mouth. If external vesicles are not seen, diagnosis can usually be confirmed by endoscopy through tissue sampling and microscopic testing. Antiviral agents such as acyclovir, ganciclovir, and foscarnet are used to treat viral esophagitis. The specific agent and dosages vary depending on the type and extent of viral infection.1,2

**FUNGAL**

*Candida* species are the major cause of fungal esophagitis. This condition is mostly seen in immunocompromised patients, such as those with HIV infection, cancer, or diabetes. Patients typically present with odynophagia and dysphagia, but these symptoms are sometimes absent. It is rare for patients to have significant systemic symptoms or bleeding. Examination may reveal clues such as classic mucocutaneous findings or white patches of oral thrush, although the absence of these external findings does not exclude fungal infection. Endoscopy can confirm the diagnosis through tissue sampling and microscopic testing. Treatment options include nystatin, clotrimazole, ketoconazole, fluconazole, miconazole, or amphotericin.1,2

**BACTERIAL AND PROTOZOAL**

Bacterial and parasitic esophagitis are uncommon. In the immunocompromised patient, *Lactobacillus*, β-hemolytic streptococci, *Cryptosporidium*, *Pneumocystis carinii*, and *Mycobacterium tuberculosis* have been reported as causative agents. Bacterial or protozoal esophagitis are usually a co-infection with a virus or fungus.2 Treatment should be guided by culture results.

**GASTRODUODENITIS AND GASTRITIS**

Gastritis is inflammation of gastric mucosa, while gastroduodenitis involves the duodenum in addition to the stomach. Inflammatory changes can have infectious,
autoimmune, or chemical etiologies, and gastritis can be acute or chronic in nature. By far, the most common infectious agent causing gastritis is *H. pylori*, which induces either acute or chronic inflammation. Most other infectious agents cause acute gastritis; these include other bacterial (eg, *Helicobacter heilmannii* or *Trep- onema pallidum*), mycobacterial, viral, parasitic, and fungal organisms. Patients with gastritis or gastroduodenitis usually present with complaints of dyspepsia, nausea, abdominal pain, and decreased appetite. Most patients with *H. pylori* infection are asymptomatic.

**HELICOBACTER PYLORI**

*H. pylori* gastritis begins as an acute infection and may progress into chronic active gastritis. In the United States, the prevalence of *H. pylori* gastric colonization increases with age. Healthy 30-year-old adults have a 25% prevalence of gastric colonization, while those over age 50 years have rates equal to their age.3 *H. pylori* gastric colonization is reported to be higher in developing countries and lower socioeconomic areas and among African-Americans and Hispanics.3,4 Most patients with *H. pylori* gastric colonization remain asymptomatic. Only a very small percentage of these asymptomatic colonizations will progress to gastric or duodenal ulcer formation and thus require treatment and eradication of the infection.

Symptomatic patients with *H. pylori* infection may describe dyspepsia symptoms similar to those seen in reflux, ulcer, and/or dysmotility. The symptoms of gastritis or gastric ulcers may be indistinguishable from those of duodenitis or duodenal ulcers. Patients may describe abdominal pain as sharp, burning, gnawing, boring, or aching, as a hunger sensation, or as pressure or fullness. Most will localize pain to the epigastric area, and some may indicate the area to the right of the epigastrium. Pain usually occurs 1 to 3 hours after a meal and frequently awakens the patient at night; it is usually relieved quickly by food or antacids. Physical examination findings reveal epigastric tenderness or right upper quadrant tenderness. Stool may test positive for occult blood. Hemorrhage is a serious complication.

The American College of Gastroenterology (ACG) recommends testing for *H. pylori* infection only in patients who will benefit from eradication of the infection, including those with active documented peptic ulcer disease, a documented history of peptic ulcer disease, or certain types of gastric lymphoma.3,5 The ACG guidelines strongly emphasize that testing should not be performed unless treatment is intended.3 Noninvasive tests include serologic tests and the urea breath test. Serologic testing is the least expensive method and is used most often in ED and office-based testing. Its value is limited, however, because serology only diagnoses *H. pylori* infection but does not provide information on the degree of involvement or associated conditions such as ulcers, mucosa-associated lymphoid tissue lymphoma (MALT), and cancer. In the emergent setting of an active upper GI bleed, serologic tests offer the most reliable results. As part of an outpatient workup, the urea breath test is the most accurate noninvasive test for confirming the diagnosis and is effective for monitoring efficacy of treatment; unfortunately this test is not available in the ED setting. Endoscopy provides the best methods of testing through histology, culture, and detection of urease.

Treatment of gastric and duodenal ulcers traditionally focused on pain relief and ulcer healing. However, since a causal link between *H. pylori* infection and gastroduodenal inflammation and ulcers was established, the major treatment objective is to eradicate the infection. Treatment is recommended only if the patient has an active ulcer or a history of documented ulcers, regardless of nonsteroidal anti-inflammatory agent use. Eradication dramatically decreases the rate of ulcer recurrence and its complications. Treatment regimens are aimed at maximizing patient compliance and eradication of the *H. pylori* infection while minimizing risks of therapy. A variety of treatments can be used in combination, including bismuth compounds, amoxicillin, tetracycline, clarithromycin, metronidazole, omeprazole, and H₂-receptor antagonists.2,5 Consensus has not been reached regarding the merit of one combination therapy over another.

**OTHER BACTERIAL GASTRITIDES**

Bacterial gastritis may progress to a rare, life-threatening infection of the gastric wall leading to necrosis that often presents as sepsis. Causative bacteria include streptococci, staphylococci, *Proteus*, and *Escherichia coli*. Infection with these bacteria can result in the rare condition known as phlegmonous gastritis. Management includes rapid diagnosis intraoperatively or by computed tomography, administration of intravenous antibiotics, and conservative fluid management. Gastrectomy may be required.

**VIRAL**

Generally, infectious viral gastritis is only found in immunocompromised patients. These infections may be caused by HSV or CMV. Symptoms are similar to other gastritides, and diagnosis is made by endoscopy. Antiviral therapy is guided by type of virus, extent of infection, and comorbid conditions.
SMALL AND LARGE BOWEL INFECTIONS

Most patients with small and large bowel infections present with nausea, vomiting, and/or diarrhea. These patients are classified as having 1 of 2 clinical syndromes based on symptoms and clinical findings: the toxigenic or the invasive/cytopathic clinical syndrome (Table 1). Diagnostic and treatment strategies vary accordingly. In general, patients presenting with the toxigenic syndrome require supportive therapy alone, while those with the invasive/cytopathic syndrome may require antibiotics.

TOXIGENIC CLINICAL SYNDROME

Toxigenic clinical syndrome is characterized by a short incubation period (1 to 12 hours), abrupt onset of vomiting and/or diarrhea without fever or systemic symptoms, minimal physical examination findings, and rapid resolution (less than 24 hours). Patients with this syndrome generally do not appear toxic, but they may be dehydrated. The toxigenic syndrome follows ingestion of preformed toxins (such as those produced by Staphylococcus aureus, Bacillus cereus, and Clostridium perfringens), toxins produced after colonization (as with Clostridium perfringens), or alteration of the intestinal mucosa without invasion. The toxins cause hypersecretion of fluid and electrolytes into the bowel lumen without causing cellular destruction. The infections that can result in a toxigenic syndrome, including their clinical presentation and specific treatment, are discussed in the following sections. Food poisoning by Staphylococcus aureus, B. cereus, and Clostridium perfringens will not be discussed here since they are not true infections. The incubation period and duration may be substantially longer for some of the infections listed below, particularly with enterotoxigenic E. coli, V. cholerae, and protozoal infection.

Viral

Norwalk virus. Norwalk virus (and related caliciviruses) may be transmitted via poorly cooked or raw shellfish that have been contaminated with untreated fecal waste.6 Outbreaks occur year round in which older children and adults are affected but infants and younger children are spared.7 Patients usually present with a typical toxigenic syndrome (Table 1), although headache, malaise, fever, and leukocytosis may be present. Symptoms generally appear 24 to 48 hours after ingestion and resolve within 24 to 48 hours; bismuth subsalicylate has been shown to reduce symptom duration.8

Rotavirus. Rotavirus infection occurs throughout the world. It is the most prominent cause of dehydrating diarrhea in children under age 3 years, and it accounts for 30% to 50% of all cases of diarrhea requiring hospitalization for rehydration in this age-group. This virus kills intestinal villi cells, leading to a loss of absorptive area and osmotic diarrhea. Vomiting precedes diarrhea in 80% of cases and may be accompanied by fever. Symptoms generally last 2 to 6 days. Treatment consists of rehydration alone.

Bacterial

Aeromonas. Aeromonas species are found in both freshwater and saltwater and are common food contaminants in the United States. Aeromonas species account for 10% to 15% of all childhood diarrheal illness.9 They can be pathogenic in adults as well, particularly in the immunocompromised.10 Predisposing factors include extremes of age, underlying GI illness, and recent hospitalization or antibiotics. Most Aeromonas infections result from drinking untreated well or spring water.9 The pathophysiology of Aeromonas diarrhea remains unclear. Patients usually present with a toxigenic syndrome (Table 1), occasionally have fever, and rarely present with invasive characteristics. Children tend to have a more acute, severe illness. If untreated, diarrhea lasts for 2 to 10 weeks and tends to be persistent in adults. Treatment with ciprofloxacin results in rapid cure.
*Escherichia coli* is the most common cause of traveler’s diarrhea, affecting 30% to 50% of travelers from industrialized nations going to developing countries. Ingestion of food or beverages contaminated with feces results in infection. Common sources include unpeeled fruits, leafy vegetables, impure drinking water, and ice cubes. Bacteria colonize the small intestine and elaborate 2 types of enterotoxins—heat-labile and heat-sensitive. Both toxins cause fluid and electrolyte secretion into the bowel lumen and subsequent diarrhea. Supportive treatment with fluids and possibly antimotility agents is generally sufficient.

**Vibrio cholerae.** Humans are the only species infected with *Vibrio cholerae*, which is transmitted through food or water contaminated with feces. There have been 7 world pandemics of cholera since 1817. Cholera is rare in industrialized countries but remains commonplace in other parts of the world, particularly the Indian subcontinent and sub-Saharan Africa. Over the past 15 years, sporadic endemic infections due to consumption of contaminated shellfish have occurred in the Gulf Coast states. The organism colonizes the upper small bowel and elaborates cholera toxin. This exotoxin causes a persistent activation of adenylate cyclase, which leads to fluid and electrolyte secretion. The resultant massive, watery diarrhea (up to 15 L/day) is referred to as “rice-water” stool due to its thin, gray-white appearance. Severe dehydration, acidosis, and hypokalemia are responsible for a mortality rate of 40% without treatment. Mortality is dramatically decreased to 1% with appropriate treatment. Although prevention of dehydration is the primary therapy, treatment with ciprofloxacin or doxycycline has been shown to reduce duration of illness, volume loss, and duration of bacterial shedding.

**Vibrio vulnificus.** *Vibrio vulnificus* is found naturally in the waters of the U.S. Gulf Coast. Outbreaks commonly occur in the summer and fall when the water is warmer and the organism multiplies more rapidly. An average of 40 culture-proven cases of this infection along with 35 hospitalizations and 12 deaths occur each year in the Gulf Coast region. Illness follows consumption of raw or undercooked oysters. Food contaminated by *V. vulnificus* unfortunately does not appear, smell, or taste abnormal. *V. vulnificus* can cause illness through 2 routes, namely, the production of an enterotoxin or via direct mucosal invasion. Symptoms range from typical toxigenic syndrome to bloody diarrhea and may be accompanied by fever, chills, and headache. High-risk patients (Table 2) may develop sepsis; patients in whom sepsis develops have a 50% mortality rate. High-risk patients should be advised against eating raw or undercooked shellfish as they are 80 times more likely to develop illness from infection. Invasive illness should be treated with doxycycline or a third-generation cephalosporin.

### Protozoal

**Cryptosporidium and Isospora belli.** *Cryptosporidium* and *Isospora belli* generally cause illness in the immunocompromised host and result in indistinguishable illness. Both are intestinal parasites that cause diarrhea in the young of numerous species. *Cryptosporidium* was first reported as a cause of human diarrhea in 1976 and is now recognized to be the most common cause of chronic diarrhea in AIDS patients. Patients with congenital immunodeficiency and those receiving treatment with chemotherapeutic agents or immunosuppressive medications also are predisposed to infection. Several large outbreaks have been reported worldwide, including a waterborne outbreak in Milwaukee, WI, in 1993 that affected more than 400,000 persons. *Cryptosporidium* spores are not reliably removed by standard water filtration systems. Transmission occurs through oral-fecal contamination of food, surfaces, and toys.

*I. belli* generally affects only AIDS patients. Diarrhea results from alteration of the intestinal mucosa. The incubation period is 1 week, which is followed by a typical toxigenic syndrome that generally lasts 1 to 3 weeks in immunocompetent patients, although chronic diarrhea is the rule in immunocompromised patients. Examination of stool for ova and parasites is the primary mode of diagnosis. Treatment is generally supportive, including antimotility agents and fluids.

**Cyclospora cayetanensis.** *Cyclospora cayetanensis* was
identified as a human pathogen in 1994. The average incubation period for *Cyclospora cayetanensis* is 1 week. This protozoal parasite invades the intestinal epithelial cells, causing watery diarrhea (through malabsorption), anorexia, weight loss, abdominal pain, nausea, vomiting, malaise, myalgias, low-grade fever, and fatigue. Symptoms may continue for more than 1 month if left untreated, although some infections are asymptomatic. Treatment is trimethoprim-sulfamethoxazole.

**Giardia lamblia.** *Giardia* is the most common cause of waterborne diarrhea in the United States. Ingestion of water contaminated with cysts from human or animal feces (particularly from beavers, muskrats, dogs, and raccoons) produces illness. *G. lamblia* is responsible for “backpacker’s diarrhea,” which is transmitted by drinking stream water in the mountainous West. Trophozoites adhere to duodenal, jejunal, and upper ileal mucosa, causing symptoms primarily through malabsorption. Most patients infected with *G. lamblia* are asymptomatic carriers. Acute infection presents with abdominal distention, colicky pain, flatulence, and frequent, explosive diarrhea with pale, loose, foul-smelling stools. Symptom onset is abrupt following a 1 to 3 week incubation period. Stool examination for ova and parasites is the primary mode of diagnosis, although small bowel aspiration or biopsies also can be used to diagnose this disease. All patients, even asymptomatic carriers, should be treated with metronidazole.

**INVASIVE/CYTOPATHIC CLINICAL SYNDROME**

Invasive/cytopathic clinical syndrome is characterized by a prolonged incubation period (1 to 3 days), gradual symptom onset, fever, abdominal pain and tenderness, and duration of 1 to 7 days. Systemic symptoms may be present, including headache, nausea, vomiting, malaise, and myalgias. This syndrome follows direct mucosal invasion and subsequent inflammation, which often results in bloody or mucoid stools and fecal leukocytes. The infections that can result in an invasive/cytopathic syndrome, including their clinical presentation and specific treatment, are discussed in the following sections.

**Bacterial**

*Campylobacter.* *Campylobacter* is the most common cause of infectious bacterial food poisoning, with more than 2 million cases and 500 deaths occurring each year in the United States. Most cases are sporadic, and children under age 4 years have the highest incidence. Chickens are the primary source of infection, although water, unpasteurized milk, red meat, and sick pets can transmit disease. Direct invasion of the colonic epithelium causes illness. Patients may present with a disease spectrum from mild to the typical fulminant invasive syndrome. Complications are rare but serious. Bacteremia occurs in 1% of cases, primarily in the immunocompromised patient and those at the extremes of age. Bacteremic patients are at risk for meningitis, cholecystitis, and urinary tract infections. Late complications include Guillain-Barré syndrome, which complicates 1 in 1000 infections, and reactive arthritis. Up to 40% of Guillain-Barré syndrome cases are estimated to be due to *Campylobacter* infection.

*Clostridium difficile.* *Clostridium difficile* is the most common cause of infectious diarrhea affecting hospitalized patients in the United States. Colonization of healthy, nonhospitalized adults is rare, but colonization rates greater than 20% for hospitalized adults have been reported. There is a clear association with recent antibiotic use and development of symptomatic *Clostridium difficile* colitis, especially use of clindamycin, ampicillin, and cephalosporins. For illness to develop, patients must be colonized with a toxigenic strain of *Clostridium difficile* and subsequently be exposed to antibiotics, which suppress the normal colonic flora, allowing these bacteria to proliferate. However, most patients fitting the aforementioned profile remain asymptomatic. Many physicians believe that a third condition must be met in order for patients to demonstrate symptoms, such as impaired host immunity or type and duration of antibiotic exposure.

*Clostridium difficile* produces 2 toxins that damage colonic mucosa, leading to inflammation. Patient presentations range from asymptomatic to pseudomembranous colitis with hemorrhage to toxic megacolon. The ACG recommends testing a single stool sample for *Clostridium difficile* or its toxin only in patients with diarrhea who have received antibiotics within the previous 2 months or if the diarrhea began 72 hours or more after hospitalization. If the stool sample testing is negative but symptoms persist, 1 or 2 more samples should be tested. If the diagnosis needs to be known urgently or if no stool is available, endoscopy should be performed to look for pseudomembranes and to collect stool for toxin assays. Stool testing for the toxin is the most specific method of diagnosis, and culture for the organism is the most sensitive, but both tests may take a minimum of 2 days. Fecal leukocyte testing is neither sensitive nor specific and is therefore of little value. Oral metronidazole is the treatment of choice, and vancomycin is an alternative. The preferred route of antibiotic administration is oral, since this provides a higher intestinal antibiotic concentration and is therefore
more effective. Antimotility agents are contraindicated since they predispose to the development of toxic megacolon. Surgery may be necessary for those with toxic megacolon or those with suspected perforation.15

*Escherichia coli* O157:H7. Each year, *Escherichia coli* O157:H7 infects 73,000 people in the United States and results in 2100 hospitalizations and 61 deaths.8 This organism was first isolated in 1982 in Michigan and Oregon, where it was traced to contaminated hamburgers.18 Most reported cases occur in the United States and Canada. *Escherichia coli* O157:H7 is found in the intestines of healthy cattle and meat contaminated during slaughter. Most infections are traced to eating contaminated ground beef, although transmission through water supplies and lakes contaminated with feces as well as person-to-person transmission have been reported. The organism is noninvasive but attaches to the intestinal mucosa where it produces 2 cytotoxins that damage intestinal vascular endothelial cells and cause hemorrhagic colitis, thus distinguishing itself from other *Escherichia coli* strains. Symptoms are typical of an invasive/cytopathic syndrome, although fever is uncommon. Between 2% and 7% of those infected will develop hemolytic uremic syndrome (HUS), and 5% of these patients will die.6 HUS consists of microangiopathic hemolytic anemia, thrombocytopenia, and renal failure; it is the most serious complication of *Escherichia coli* O157:H7 infection. Risk factors for HUS include age under 2 years, severe gastrointestinal symptoms, fever, leukocytosis, and antibiotic treatment.19 Special cultures (sorbitol-MacConkey agar) must be requested if there is any suspicion of *Escherichia coli* O157:H7. Treatment of infection is supportive, and antimotility agents are not recommended. Use of antibiotics remains controversial since they have been associated with an increased risk of HUS.19

*Plesiomonas shigelloides*. *Plesiomonas shigelloides* infection is transmitted by consumption of raw oysters and has been associated with travel to Mexico. The bacteria directly invade the intestinal mucosa, producing typical invasive syndrome. Patients with *Plesiomonas shigelloides* should be evaluated for immunodeficiency, although up to 17% of immunocompetent patients with diarrhea may have positive cultures.20 The laboratory must be notified if *Plesiomonas shigelloides* is suspected, since special testing is required to identify the organism.

*Salmonella*. *Salmonella* is the second most common cause of infectious bacterial food poisoning in the United States, resulting in 1.4 million cases and causing 1000 deaths each year.6 The primary reservoir is the intestinal tracts of infected or colonized animals, including cattle, pigs, and chickens. Contaminated eggs are responsible for 75% of all *Salmonella enteritidis* outbreaks. Patients may not realize that raw eggs are ingredients in many common food items such as hollandaise sauce, Caesar dressing, tiramisu, homemade ice cream, homemade mayonnaise, cookie dough, and frostings. Pet turtles are another common source of childhood infection, but the sale of turtles was banned in 1975.6 Half of all human *Salmonella*-related illnesses are due to infection with *Salmonella typhimurium* and *Salmonella enteritidis*. A large inoculum is required to cause illness since *Salmonella* species are acid and heat sensitive. Once ingested, organisms invade the mucosa of the distal ileum and proximal colon, producing localized inflammation. Reduced gastric acidity and recent antibiotic use predispose an individual to infection. Most *Salmonella* infections occur at the extremes of age (ie, in children less than 5 years old or the elderly). Illness presents as a typical invasive syndrome. Fever lasts 1 to 2 days, except with *Salmonella typhi* (typhoid fever), which lasts for weeks. High-risk patients (Table 2) should receive antibiotics within 48 hours for therapy to be most effective. Antimotility drugs are contraindicated because their use prolongs fever and diarrhea and increases the incidence of bacteremia and the carrier state. Complications include bacteremia (5% to 10% in high-risk patients),21 reactive arthritis (5%),22 chronic carrier state, and osteomyelitis (which most commonly affects patients with sickle cell disease). Health care workers and food handlers should not return to work until their stool cultures are negative.

*Shigella*. *Shigella* infections in the United States have modestly declined since 1995, with an incidence of 450,000 cases per year.6 Outbreaks occur in confined populations and areas with inadequate sanitation, including mental and penal institutions or nursing homes. Transmission occurs through oral-fecal contamination, and humans are the only host. *Shigella sonnei* is responsible for 75% of *Shigella* illness in the United States, followed by *Shigella flexneri*; *Shigella dysenteriae* rarely causes disease.25 *Shigella* is a highly efficient pathogen, requiring just 10 to 200 organisms to cause illness.6 Bacteria invade the bowel mucosa and produce an exotoxin that causes fluid and electrolyte secretion. Bacteremia is rare. Patients predominantly present with an invasive syndrome. Neurologic symptoms such as seizures and altered mental status are common in children.20 Symptoms generally resolve with conservative therapy alone within 1 week, although most patients will continue to shed bacteria for 2 or more weeks. The relapse rate is approximately 10%. Treatment with ampicillin, trimethoprim-sulfamethoxazole, or ciprofloxacin will shorten the illness. However, antibiotic
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treatment may lead to resistance. High-risk patients (Table 2), severely ill patients, and all patients with *Shigella dysenteriae* should receive antibiotics. Antimotility agents may prolong fever, diarrhea, and bacterial shedding. Follow-up stool cultures are required for all cases of *Shigella dysenteriae*. Reiter’s syndrome has been associated with prior *Shigella flexneri* infection.6

**Vibrio parahaemolyticus**. Vibrio parahaemolyticus is found in the coastal waters of the United States and other temperate zones.10 The Centers for Disease Control and Prevention (CDC) reports 30 to 40 cases per year in the United States, with 1 to 3 deaths in the Gulf Coast states.6 In the United States, most infections follow consumption of inadequately cooked seafood, especially shrimp or crab. Contaminated food appears, smells, and tastes normal. Of those exposed to contaminated food, a high percentage become infected. *V. parahaemolyticus*, unlike other *Vibrio* species, is invasive, causing an intense inflammatory response. Symptoms are similar to those produced by other invasive pathogens. Antibiotics have failed to reduce symptom duration or pathogen excretion. *Vibrio* species require special culture media for growth (TCBS agar), which should be requested if clinical suspicion exists.

**Yersinia enterocolitica**. Yersinia enterocolitica is found worldwide.10 Infection is transmitted via food or drink contaminated with feces, especially milk or raw pork. Bacteria directly invade the intestinal mucosa of the terminal ileum and mesenteric lymph nodes. The clinical presentation is typical of an invasive pathogen. Abdominal pain and diarrhea last 10 to 14 days when untreated. Patients may shed bacteria for up to 6 weeks. Infection is most common in young children. *Yersinia* requires special techniques and prolonged incubation for culture. Illness is generally self-limited. Antibiotics have not been shown to reduce symptom duration or shedding, but they are recommended for patients who remain symptomatic when culture results return. Late complications include erythema nodosum or polyarthritis in 2% to 5% of patients, mostly in adults.6

**Anisakis lumbricoides**

Anisakis lumbricoides larvae are transmitted through consumption of infected raw seafood. The larvae penetrate the gastrointestinal submucosa and produce heme-positive diarrhea and eosinophilia. Sushi and sashimi are the most common vectors in the United States. Mebendazole may be helpful, but surgical removal is often required.14

**Diphyllobothrium latum**

Humans acquire *Diphyllobothrium latum* by eating raw or undercooked fish containing larvae. The larvae attach to the intestinal wall with minimal damage and develop into worms. Most infections are asymptomatic, but some patients develop abdominal discomfort and diarrhea. The parasite preferentially consumes vitamin B12, producing a megaloblastic anemia. Praziquantel is the treatment of choice.14

**Entamoeba histolytica**

Entamoeba histolytica is a ubiquitous protozoan organism. Approximately 10% of the world’s population is infected with this organism, and 10% of these are symptomatic. The prevalence in the United States is estimated at 4%.10 Consumption of food or water contaminated with feces is the primary mode of transmission. Trophozoites invade the colonic mucosa, forming ulcerations. Symptoms range from asymptomatic cyst passing to amebic dysentery. Acute amebic dysentery presents with abrupt onset of fever, severe abdominal cramping, profuse bloody diarrhea, and tenesmus. Chronic amebic dysentery is more common, and consists of intermittent bloody or mucus-tinged diarrhea. Diagnosis is confirmed by identifying the organism in stool or via serologic testing. Complications include hepatic abscesses and amebomas, which may cause colonic obstruction. Paromomycin or iodoquinol are the drugs of choice.14

**Taenia saginata**

*Taenia saginata* causes infection in humans who consume raw or undercooked beef containing larvae. Larvae attach to the gut wall and develop into adult worms. Most patients with adult tapeworms are asymptomatic, although worms may appear in the stool or protrude from the anus. The treatment of choice is niclosamide or praziquantel.14

**WORKUP AND MANAGEMENT**

An approach to the diagnosis and management of bowel infections in the emergent setting is presented in the Figure. Patients who appear well, do not meet high-risk criteria (Table 2), are able to tolerate oral fluids, and have a history consistent with a typical toxigenic exposure should receive oral rehydration and antiemetics. Antibiotics are rarely indicated. These patients should be advised to adopt a lactose-free diet and avoid caffeine for up to 1 week after symptom resolution. Health care providers and food handlers should remain home from work until complete resolution of symptoms.

Oral hydration solutions should contain water, glucose, and electrolytes. Glucose present in the solution
stimulates absorption of electrolytes and water via a glucose-sodium transport mechanism that is unaffected by toxin-induced diarrhea. The World Health Organization recommends rehydrating patients by adding the following to 1 L of water: 20 g of glucose, 3.5 g of sodium chloride, 2.5 g of sodium bicarbonate, and 1.5 g of potassium chloride. Rehydralyte and Pedialyte RS also are acceptable. Pedialyte, Lytren, Gatorade, and other commercial electrolyte solutions are less effective because they contain insufficient electrolytes or have excessive glucose concentrations.

Patients demonstrating invasive/cytopathic symptoms should have stool testing for fecal leukocytes. If fecal leukocytes are not present and the patient appears well and is not high risk, treatment is supportive therapy alone. Patients with fecal leukocytes, those appearing systemically ill, or those meeting high-risk criteria should have stool sent for culture. The presence of fecal leukocytes has a 45% positive predictive value of a positive stool culture and a 93% negative predictive value. The laboratory should be notified if infection with Escherichia coli O157:H7, Plesiomonas shigelloides, or V. parahaemolyticus is suspected because special culture media are required for their growth. Patients with invasive/cytopathic syndromes who appear well should receive supportive therapy alone. Empiric antibiotics may be prescribed for prolonged or severe cases (ie, those with symptoms lasting longer than 7 days or those with high fever, dehydration, or toxic appearance), but antimotility agents should be avoided. Recommended antibiotics include ciprofloxacin 500 mg twice daily by mouth or 1 trimethoprim-sulfamethoxazole DS tablet twice daily by mouth for 3 to 5 days. Patients with severe dehydration or signs of systemic toxicity should be admitted. Patients who are released without treatment with antibiotics but whose stool cultures are returned as positive should be contacted for a follow-up examination. Antibiotics should be prescribed if the patient remains symptomatic at follow-up. Stool testing for ova and parasites should be considered if the history suggests possible parasitic infection (eg, travel to endemic regions), if symptoms have persisted beyond 2 weeks, or if symptoms persist despite antibiotic treatment.

The pathogens that emergency medicine physicians must report to their local public health departments are listed in Table 3. The CDC monitors these pathogens for epidemiologic purposes. Finally, emergency
Table 3. Pathogens Reportable to the Centers for Disease Control and Prevention

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<td>Campylobacter</td>
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<td>Cryptosporidium</td>
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<td>Cyclospora</td>
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<td><em>Escherichia coli</em> O157:H7</td>
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<td><em>Listeria monocytogenes</em></td>
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<td><em>Salmonella</em></td>
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<td>Shigella</td>
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<tr>
<td><em>Vibrio</em> species</td>
</tr>
<tr>
<td><em>Yersinia</em> enterocolitica</td>
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</tbody>
</table>

medicine physicians should educate their patients to practice good hygiene (eg, frequent hand-washing) and safe food preparation (ie, wash all utensils and surfaces before and after food preparation, keep raw animal products separate from ready-to-eat foods, thoroughly cook and chill foods) in order to prevent further illness. Patients who work in the food preparation industry or with the immunocompromised should not return to work until all symptoms have resolved and repeat cultures are negative.

**CONCLUSION**

Emergency medicine physicians have an important role in the recognition and early treatment of GI infections. Although most patients require supportive care alone, the emergency medicine physician must identify patients who require further diagnostic studies and specific therapy. Physicians also must educate their patients in order to reduce the spread of disease as well as report pathogens tracked by the CDC to local public health departments.

**REFERENCES**


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