Bacterial Colitis

Harry T. Papaconstantinou, M.D.[1] and J. Scott Thomas, M.D.[1]

ABSTRACT

Bacterial colitis results in an inflammatory-type diarrhea that is characterized by bloody, purulent, and mucoid stool. These diseases have been designated as bacterial hemorrhagic enterocolitis. Associated symptoms include fever, tenesmus, and severe abdominal pain. The pathologic changes range from superficial exudative enterocolitis to a transmural enterocolitis with ulceration. Common pathologic bacteria causing bacterial colitis include *Campylobacter*, *Salmonella*, *Shigella*, *Escherichia*, and *Yersinia* species. The primary source of transmission is fecal-oral spread and ingestion of contaminated food and water. Although detailed history and identification of specific risk factors assist in the diagnosis, definitive diagnosis requires bacterial identification. Therefore, the physician must be familiar with the disease pathophysiology, epidemiology, and specific diagnostic modalities for clinical diagnosis and management. Specific tests are used to detect enteric pathogens and include stool and rectal swab culture, histology, and identification of specific bacterial toxins. Although many of these bacterial colitis infections are self-limiting, antibiotics should be used for high-risk patients and patients with complicated disease.

KEYWORDS: Bacteria, colitis, diarrhea, dysentery

Objectives: On completion of this article, the reader should be able to diagnose and treat bacterial colitis caused by common organisms.

Acute infectious bacterial diarrhea is a common presenting problem in general practice and is a significant health problem in both developing and developed regions of the world. Children, elderly persons, and immunocompromised individuals are especially susceptible to these infections. Common modes of transmission include the fecal-oral route, animal hosts, ingestion of contaminated food and water, and close human-to-human contact. Infection through direct contact is common in areas where people are housed together with potential exposure to compromised hygiene (i.e., day care centers and nursing homes).[1] The ingestion of water and food contaminated with pathogenic microorganisms is a significant source of disease transmission and has caused large outbreaks of disease in the United States.[2,3]

Bacterial diarrhea can be classified into noninflammatory diarrhea and inflammatory diarrhea. Noninflammatory diarrhea is caused by pathogenic bacteria (i.e., enterotoxigenic *Escherichia coli* and *Staphylococcus*) that alter normal absorptive and secretory processes of the bowel, leading to watery diarrhea without febrile illness. Inflammatory diarrhea is characterized by bloody and mucopurulent stool that is often associated with fever, tenesmus, and severe abdominal pain. Common pathogenic bacteria causing inflammatory diarrhea include *Campylobacter*, *Salmonella*, *Shigella*, enteroinvasive and enterohemorrhagic *Escherichia coli*, *Yersinia*,

[1]Department of Surgery, Division of Surgical Oncology, Section of Colon and Rectal Surgery, The Texas A&M University System Health Science Center, Scott and White Hospital, Temple, Texas.

Address for correspondence and reprint requests: Harry T. Papaconstantinou, M.D., Department of Surgery, Division of Surgical Oncology, Section of Colon and Rectal Surgery, The Texas A&M University System Health Science Center, Scott and White Hospital, 2401 South 31st St., Temple, TX 76508. E-mail: hpapaconstantinou@swmail.sw.org.

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Chlamydia, *Neisseria*, and tuberculosis. These organisms cause a bacterial hemorrhagic enterocolitis and are the focus of this article.

CAMPYLOBACTER

Campylobacter, a curved, highly motile microaerophilic gram-positive rod, has become one of the major causes of infectious diarrhea today.[4] The most important species found in human infections is *Campylobacter jejuni*. In the United States, 4% to 11% of all cases of diarrhea are caused by *C. jejuni*, and the isolation of *Campylobacter* species in these patients is two times more common than that of *Salmonella* and seven times more common than that of *Shigella*.[5]

Epidemiology

Transmission occurs most commonly through contaminated poultry and is acquired by eating undercooked chicken. The reservoir for this organism is enormous because many animals can be infected and includes cattle, sheep, swine, birds, and dogs.

Clinical Features

After ingestion, the incubation period is 24 to 72 hours. Clinical illness manifests as frank dysentery, with few patients exhibiting watery diarrhea or asymptomatic excretion.[4] The most common clinical symptoms are diarrhea and fever (90%), abdominal pain (70%), and bloody stool (50%). Localized infections of the terminal ileum and cecum can suggest a clinical picture of acute appendicitis. *Campylobacter* species possess oxidase and catalase activity that facilitates invasion and ulceration in the colonic mucosa, resulting in bloody stools. Most illnesses last less than 1 week, although symptoms can persist for 2 weeks or more and relapses occur in as many as 25% of patients.[6] In up to 16% of patients, prolonged carriage of the organism can occur for 2 to 10 weeks. Recurrent and chronic infection is generally reported in immunocompromised patients.

Complications of *Campylobacter* infections are rare and include gastrointestinal hemorrhage, toxic megacolon, pancreatitis, cholecystitis, hemolytic-uremic syndrome (HUS), meningitis, and purulent arthritis. Reiter syndrome and Guillain-Barré syndrome are conditions that may follow *C. jejuni* enterocolitis. Reiter syndrome is a reactive arthritis that is observed more frequently in patients who carry the HLA-B27 phenotype.[7,8] Guillain-Barré syndrome is found as a chronic sequel of *C. jejuni* infections with serotype HS:19. Cross-reactivity of antibodies to *C. jejuni* lipopolysaccharide and antigenic determinants of nerve gangliosides are speculated to contribute to the nerve damage in these patients that result in muscle weakness and sensory nerve abnormalities.[9]

Diagnosis

Stool examination reveals the presence of fecal leukocytes and erythrocytes supporting the diagnosis of colitis, and laboratory tests frequently indicate volume depletion and leukocytosis. Colonoscopic findings show segmental edema, loss of normal vascular pattern with ulceration, and patchy involvement of the colonic mucosa.[10] These tests, however, are nonspecific. Diagnosis can be established only by culture of organisms. The yield of *C. jejuni* is higher from colonic tissue culture than stool culture.[11] *Campylobacter* species grow much more slowly than other enteric bacteria; therefore, successful identification requires culture on Skirrow's selective medium incubated at 42°C under an atmosphere of 5% O_2 and 10% CO_2.

Treatment

Most patients with mild to moderate *C. jejuni* enterocolitis do not benefit from antibiotic therapy because this illness is usually self-limiting.[12] Treatment is reserved for patients with dysentery and high fever suggestive of bacteremia and debilitated or immunocompromised patients. Quinolone antibiotics should be used empirically because isolation and identification of the pathogen takes time and quinolone antibiotics are active against *Campylobacter*, *Shigella*, and other common enteric pathogens.

Resistance to fluoroquinolones is a major problem in parts of the developing world and has been identified in certain parts of the United States. In a large study from Minnesota, human isolates of *Campylobacter* species exhibited a rise in quinolone resistance from 1.3% to 10.2% between 1992 and 1998.[13] Resistance has been linked to foreign travel, local patterns of fluoroquinolone use, and antibiotic use in animal husbandry. In areas where fluoroquinolone resistance is common, azithromycin has proved effective and should be used.

Although *C. jejuni* is sensitive to erythromycin in vitro, therapeutic trials have shown no effect on the clinical course when compared with placebo.[14] However, fecal excretion of the organism is reduced by erythromycin.

SALMONELLA

Salmonella species are gram-negative, rod-shaped bacilli that are members of the Enterobacteriaceae family. *Salmonella typhi* and *Salmonella paratyphi* cause typhoid fever, and other *Salmonella* species are associated with gastroenteritis, enterocolitis, and focal infections

including meningitis, septic arthritis, cholangitis, and pneumonia.[15,16]

Epidemiology

Salmonella is considered primarily a food-borne infection. The major route of transmission is by the "5 Fs": flies, food, fingers, feces, and fomites. Large outbreaks of *Salmonella* species–induced enterocolitis are frequently derived from institutional dinners and contaminated food and water supply. In the United States, the two most common serotypes that result in enterocolitis are *Salmonella enteritidis* and *Salmonella typhimurium*.[4] The incidence of these infections is estimated as 20 cases per 100,000 population in the United States. Nonhuman reservoirs play a crucial role in transmission of this disease, with up to 80% of outbreaks being caused by animals or animal products. Poultry has the highest incidence of *Salmonella* contamination (40% turkeys, 50% chickens, and 20% of commercial egg whites). Household pets, especially turtles and lizards, have also been implicated in outbreaks of *Salmonella*. Infectivity of a specific strain is related to its serotype and inoculum quantity.

S. typhi is the primary cause of typhoid fever, with ~500 cases occurring in the United States each year.[15] This organism is unique among the *Salmonella* species in that its only natural reservoir is humans. Identification of an infection could indicate the presence of a carrier state; therefore, public health authorities should be notified so that chronic carriers can be registered and the microorganism typed so that outbreaks can be traced.

Clinical Features

Nontyphoidal *Salmonella* infections arise with nausea, vomiting, abdominal cramps, and diarrhea. The diarrhea can vary from loose stools to dysentery with grossly bloody and purulent feces. Symptoms arise 8 to 48 hours after ingestion of contaminated food. The illness lasts for 3 to 5 days in patients manifesting with gastroenteritis and 2 to 3 weeks in patients who develop enterocolitis. Toxic megacolon is a known complication of *Salmonella* colitis.[17] Bacteremia occurs in up to 10% of patients and can result in focal infections such as meningitis, arteritis, endocarditis, osteomyelitis, septic arthritis, and focal abscesses.[12] Predisposing factors that increase the risk of salmonellosis include sickle cell anemia, hemolytic anemias (malaria), immunosuppression (corticosteroids, chemotherapy, and acquired immunodeficiency syndrome [AIDS]), low gastric acidity (H_2 receptor blockers and resection of the stomach), and patients at extremes of age (infants < 1 year old and elderly patients > 60 years old).[4] A chronic carrier state is seen in less than 1% of infected individuals and is usually associated with structural abnormalities of the biliary tract, such as cholelithiasis, or the urinary tract, such as nephrolithiasis.[18]

Clinical symptoms of *S. typhi*, also known as typhoid fever, include sustained hectic fever, delirium, abdominal pain, splenomegaly, persistent bacteremia, and "rose spot" skin rashes.[4] Untreated, the illness lasts ~4 weeks. Typhoidal disease is not truly an intestinal disease and has more systemic than intestinal symptoms. Ingested organisms penetrate the small bowel mucosa and rapidly enter the lymphatics, mesenteric lymph nodes, and then the bloodstream. After this initial bacteremic event, the organism is sequestered in macrophages and monocytic cells of the reticuloendothelial system. These sequestered cells multiply and reemerge several days later in recurrent waves of bacteremia spreading throughout the host and infecting many organ sites. The liver, spleen, and lymph nodes (including Peyer's patches) become involved and may result in focal areas of liver and spleen necrosis, acute cholecystitis, and microperforations in the terminal ileum. Erosion into blood vessels may produce severe intestinal hemorrhage. After 6 weeks, ~50% of patients with typhoid fever still shed organisms in their feces. This declines with time to 1% to 3% shedding organisms at 1 year, which is defined as a chronic carrier state. Patients who are high risk for the carrier state are older patients, women, and patients with biliary disease.[12,18]

Diagnosis

Diagnosis of salmonellosis and typhoid fever is established by isolating the organism. Blood culture during episodes of bacteremia is positive in up to 90% of patients within the first week of symptoms with *S.typhi*. Cultures from stool, rectal swab, and endoscopic biopsy specimens are effective. Endoscopic evaluation of the colon in patients with nontyphoidal salmonellosis reveals hyperemia, friability of the mucosa, ulcerations, aphthous erosions, and deep fissures with segmental involvement of the colon.[19,20] In patients with *S. typhi* the involvement parallels the anatomic location of Peyer's patches (terminal ileum and proximal colon) with characteristic oval contour ulcerations with raised margins and a clear white base.

Treatment

Most cases of nontyphoidal *Salmonella* enterocolitis are self-limiting and do not require antibiotic therapy. Antibiotic therapy has no effect on duration of illness, diarrhea, or fever, and some studies have shown prolonged fecal excretion in antibiotic-treated patients.[21,22] Therefore, antimicrobial therapy should not be used in most cases of nontyphoidal *Salmonella* enterocolitis. Exceptions include patients with lymphoproliferative disorders, malignancy, AIDS, transplantation, prosthetic implants, valvular heart disease,

hemolytic anemias, extreme ages of life, and symptoms of severe sepsis. Amoxicillin, quinolones, or trimethoprim-sulfamethoxazole (TMP-SMX) are first-line antibiotics for uncomplicated disease; parenteral third-generation cephalosporin or quinolones are reserved for more severe infections.[12]

The antibiotic treatment for *Salmonella* typhoid and typhoid fever is chloramphenicol, TMP-SMX, and ampicillin. However, worldwide emergence of organisms that are resistant to these antibiotics has caused concern. A 10- to 14-day course of a quinolone is highly effective for the treatment of typhoid fever, and quinolone antibiotics have become the treatment of choice in eradicating the carrier state.[23]

SHIGELLA

Shigellae are a group of gram-negative enteric organisms that are included in the Enterobacteriaceae family and cause a broad spectrum of gastrointestinal illness ranging from mild diarrhea to life-threatening dysentery. There are four major subgroups: *Shigella dysenteriae* (group A), *S. flexneri* (group B), *S. boydii* (group C), and *S. sonnei* (group D).[4] Shigellosis is a worldwide endemic disease and is responsible for more than 650,000 deaths each year.[12] In the United States, *S. sonnei* is the most common serotype and is the cause of nearly 80% of bacillary dysentery.[24] *S. dysenteriae* and *S. flexneri* are the predominant species causing endemics and pandemics in developing countries.

Epidemiology

Shigella is highly contagious and requires only a small number of ingested inocula to yield clinical symptoms in infected volunteers.[25] The disease is spread readily through person-to-person contact with fecal-oral and oral-anal contacts. In developed countries, *Shigella* infection is most commonly seen in day care centers, nursery schools, and male homosexuals.[26,27]

Clinical Features

After ingestion, incubation periods range between 6 hours and 9 days. The classic presentation of bacillary dysentery is with crampy abdominal pain, rectal burning, and fever, associated with multiple small-volume bloody mucoid stools. All *Shigella* species are capable of elaborating Shiga toxin, a potent toxin that is enterotoxic, cytotoxic, and neurotoxic.[28] Initial diarrhea is watery without gross blood and is related to the action of enterotoxin. The second phase is associated with tenesmus and small-volume bloody stools that occur 3 to 5 days after onset and corresponds to invasion of the colonic epithelium and acute colitis. Toxic, highly febrile illness is associated with severe colitis; however, bacteremia is distinctly uncommon. Severe complications are relatively common and include intestinal perforation, megacolon, septic shock, HUS, profound dehydration, hypoglycemia, hyponatremia, seizures, and encephalopathy.[29] Arthritis, joint pain, and effusions may appear and are usually associated with HLA-B27. This clinical picture is a result of cross-reacting antigens with *Shigella* proteins resulting in circulating antibody-antigen complexes.[30]

The clinical course of shigellosis is variable with children exhibiting mild infections lasting no more than 1 to 3 days. Infections in adults last ~7 days, and severe cases may have persistent symptoms for 3 to 4 weeks. Untreated disease with a prolonged course may be confused with ulcerative colitis. Chronic carriers are uncommon and are susceptible to intermittent attacks of the disease.

Diagnosis

The diagnosis of shigellosis is suspected by the triad of lower abdominal pain, rectal burning, and diarrhea. Stool studies reveal multiple polymorphonuclear leukocytes and red blood cells. Stool culture is necessary to make a definitive diagnosis, and the yield is increased when fecal leukocytes and blood are present. Colonoscopy reveals erythema, edema, loss of vascular pattern, punctuate hemorrhagic spots, mucosal friability, aphthoid erosions, star-shaped ulcers, and adherence of grayish-white mucopurulent material.[31,32] The most common site of involvement is the rectum and sigmoid colon and can extend continuously toward the proximal colon. To distinguish this disease from idiopathic ulcerative colitis, colonic biopsies are required within 4 days of onset of symptoms. Otherwise, positive stool cultures and dramatic improvement on antibiotics are the only distinguishing factors in patients with prolonged shigellosis.

Treatment

Treatment is initiated with volume resuscitation and specific therapy for complicating conditions such as seizures, encephalopathy, and intestinal perforation. Antibiotic treatment is always indicated for *Shigella* infections because of its ease of transmission and propensity to cause life-threatening illness.[12] *Shigella* resistance to sulfonamides, tetracyclines, ampicillin, and TMP-SMX exists worldwide, and they are therefore not recommended as empirical therapy. Quinolones are the current drugs of choice for shigellosis in adults. In children, azithromycin is preferred because quinolone safety may be an issue.

ESCHERICHIA COLI SPECIES

Escherichia coli species are found as normal intestinal microflora in humans and animals. Most strains are

relatively harmless in the bowel; however, there are five major groups of *E. coli* that cause enteric infections, each with specific virulence factors that include toxin production, adherence to epithelial cells, and invasiveness. These groups include enteropathogenic *E. coli* (EPEC), enterotoxigenic *E. coli* (ETEC), enteroadherent *E. coli* (EAEC), enteroinvasive *E. coli* (EIEC), and enterohemorrhagic *E. coli* (EHEC).[33,34] Specific virulence factors for each group are encoded by specific genetic elements (plasmids or chromosomal genes) that determine pathogenicity. The EPEC strains are associated with diarrhea in hospitalized infants and nursery outbreaks, and EAEC strains cause persistent diarrhea in children. The ETEC strains are a major cause of outbreaks in travelers to tropical or subtropical areas. With these three infections, patients develop frequent bowel movements within 1 to 2 days after exposure, reflecting the action of enterotoxins on the intestinal mucosa. The pathogenic subtypes EIEC and EHEC cause hemorrhagic colitis with frequent bloody or mucoid diarrhea. *E. coli* O157:H7 is a specific and most common form of EHEC that was first identified in 1983 and has subsequently evolved into an important cause of frequent outbreaks of acute bacterial colitis. This EHEC subtype is the focus of the following discussion.

Epidemiology

E. coli O157:H7 is a virulent organism requiring a small inoculum of 10 to 100 organisms to produce illness.[35] It is estimated that over 70,000 cases of illness occur each year with ~60 deaths.[36] Cattle are the primary reservoir of infection, and the organism is routinely found in the intestinal tract of healthy animals with a 10% and 50% carriage rate.[33,34] Cattle lack the gut vascular receptors required for binding the Shiga toxins found within the O157:H7 organism, which may explain the lack of disease in these animals.[37] The majority of outbreaks (66%) are a result of consumption of contaminated food and are most commonly traced to inadequately cooked ground beef. Other common forms of transmission include human-to-human contact (19%) especially in child care centers, waterborne sources (12%), and direct animal contact (3%).[35,38] This organism can survive in the environment for months, making the risk for infection in contaminated areas higher. Risk factors for disease include young and old age, antibiotic therapy prior to infection, and prior gastrectomy. Young children in day care centers have been shown to shed the organism for up to 2 to 3 weeks after infection.

Clinical Features

After ingestion, the incubation period averages 3 to 4 days but can range between 1 and 10 days.[33,34] The clinical picture of *E. coli* O157:H7 infection is variable and can mimic that of other diseases such as inflammatory bowel disease, pseudomembranous colitis, or ischemic colitis. Symptoms range from an asymptomatic carrier state to diarrhea, either bloody or nonbloody. Vomiting occurs in over 50% of patients; however, fevers are rare. Symptoms usually last for 1 week, and admission to the hospital may be required in up to 40% of infected patients. The most dreaded complications are HUS and thrombocytopenia, which are caused by microangiopathic injury resulting from organism production of Shiga toxin.[36,37] *E. coli* O157:H7 is the most common cause of HUS in the United States, and serologic data suggest that it is responsible for the majority of patients with thrombocytopenic purpura.

Diagnosis

The majority of hospital-based laboratories are routinely testing for *E. coli* O157:H7 in stool cultures. Sorbitol-containing MacConkey agar is used to isolate the organism because growth in this medium requires the unique capacity of this organism to ferment sorbitol. Sorbitol-fermenting colonies are then tested with antisera against O157 and H7 antibodies.[36] Colonoscopic findings in infected individuals reveal mucosal hyperemia, shallow ulcerations, marked edema, hemorrhage, erosions, and longitudinal ulcer-like lesions throughout the colon.[39] Inflammatory changes in the mesenteric fat are common and most prominent in the right colon.[40] Computed tomography (CT) scan shows a target sign indicative of diffuse thickening of the colonic wall.[41]

Treatment

The severity of the hemorrhagic colitis and frequency of complications such as HUS and thrombotic thrombocytopenic purpura suggest that antibiotic therapy for *E. coli* O157:H7 is imperative. However, clinical data do not support the role of antibiotic use. In fact, a prospective randomized controlled trial found no effect of antibiotics on the progression of symptoms, excretion of the organism, or development of HUS.[42] Retrospective studies found that antibiotics prolong bloody diarrhea, increase fatalities, or have no effect.[43–45] Other studies have linked use of TMP-SMX and ciprofloxacin to increased production and increased extracellular release of Shiga toxin, respectively.[46,47] Specific therapies such as antimotility agents are associated with an increased risk of HUS.[48] Combined, these data indicate that antibiotics and antimotility agents should be avoided in patients with presumed EHEC infections.

YERSINIA

Yersinia enterocolitica and *Yersinia pseudotuberculosis* are gram-negative facultative anaerobic bacilli that closely

resemble *E. coli*.[4] These two species are pathogenic toward humans and infect Peyer's patches and mesenteric lymph nodes resulting in the potential to cause systemic infection. *Yersinia* enterocolitis is of particular importance to the surgeon because of its prevalence and capacity to mimic regional enteritis and appendicitis.

Epidemiology

Infection with *Yersinia* occurs through the fecal-oral route, by hand-to-mouth transfer following handling of contaminated animals or animal products, or by the ingestion of contaminated food or water. The ability of the organism to grow at 4°C means that refrigerated meats can be the source of infection. Undercooked pork products and contaminated milk products are common foods implicated in this infection.[12] Children are affected more frequently than adults. Other predisposing factors include cirrhosis, hemochromatosis, acute iron poisoning, transfusion-dependent blood dyscrasias, immunosuppressed patients, diabetics, and elderly and malnourished individuals.

Clinical Features

Y. enterocolitica and Y. *pseudotuberculosis* cause similar signs and symptoms. Typical complaints are fever, diarrhea, and abdominal pain lasting 1 to 3 weeks. Nausea and vomiting occur in 15% to 40% of cases. Fecal leukocytes, blood, or mucus may be present in stool specimens. Patients with mesenteric adenitis or ileitis may have a syndrome clinically indistinguishable from acute appendicitis.[12] Other symptoms that can occur include a migratory polyarthritis, Reiter syndrome (common in HLA-B27–positive patients), and erythema nodosum.[7,49]

Diagnosis

Routine laboratory testing is usually nonspecific. *Yersinia* can be isolated from the stool, mesenteric lymph nodes, peritoneal fluid, abscesses, or perhaps blood. Fecal isolation can be difficult because of normal flora overgrowth, and detection can be enhanced by cold incubation at 20°C to 25°C. Hemagglutination is a useful indirect test to detect *Yersinia* infection, and titers in the range of 1:128 in previously healthy individuals are suggestive of infection.

Barium enema typically demonstrates thickening of mucosal folds; round filling defects in the mucosa (indicating swollen lymphoid tissue), and fine luminal irregularities without narrowing in the terminal ileum.[50] Colonoscopic examination shows round or oval elevations with or without ulceration in the ileum and yellow oval aphthae of the colon, mimicking Crohn's disease.[51,52]

Treatment

The value of antimicrobial therapy in mesenteric adenitis and enterocolitis is unclear as these infections are usually self-limited. However, patients with prolonged enteritis, extraintestinal manifestations, or increased risk of septicemia should be treated with antibiotics such as aminoglycosides, TMP-SMX, doxycycline, or fluoroquinolones.[53] There has been no evidence of acquired resistance in recent *Yersinia* isolates.[54] However, in vivo treatment failures have been reported with third-generation cephalosporins and imipenem.[55] Septicemic patients have mortality rates in the 50% to 75% range despite appropriate antibiotic therapy. Prevention should be emphasized and focus on safe handling and preparation of all foods, especially pork and milk. Hand washing after toilet use or diaper changes as well as after the handling of animals or pets is mandatory.

TUBERCULOSIS

Gastrointestinal tuberculosis is widely prevalent in the developing world and continues to be a health hazard despite progress in prophylaxis and treatment. Clinical manifestations of this disease continue to challenge the diagnostic and therapeutic skill of treating physicians.

Epidemiology

In developing countries, the resurgence of tuberculosis has closely paralleled the AIDS epidemic.[56,57] Tuberculosis organisms that infect the gastrointestinal tract include *Mycobacterium tuberculosis* and *Mycobacterium bovis*. *M. tuberculosis* is primary to the lungs and can be carried to the intestinal tract by the swallowing of infected sputum. This is most commonly seen in patients with cavitary lung lesions. *M. bovis* is transferred through the ingestion of unpasteurized milk. The ileocecal region is the most common site for infection, but segmental and occasionally universal colitis can be observed.[58]

Clinical Features

Presenting symptoms are abdominal pain, weight loss, anorexia, and fever. Abdominal pain is located in the area of disease involvement. Three pathologic forms of intestinal tuberculosis are described and can be seen in the same patient; ulcerative, hyperplastic, and sclerotic.[59] This spectrum of pathologic presentation and its predilection for the ileocecal region are similar to findings in Crohn's disease, making the diagnosis difficult.

Physical examination may reveal the presence of a right lower quadrant mass. Stricture or ulceration may also occur and can simulate the appearance of a malignancy, prompting an oncologic resection of the involved segment of intestine.

Diagnosis

Definitive diagnosis is achieved by identification of *M. tuberculosis* or *M. bovis*. However, these organisms are difficult to culture and detect, and a definitive diagnosis is possible in a small number of patients. Therefore, a high index of suspicion must be maintained to diagnose these patients, especially in the absence of pulmonary disease. A positive tuberculin skin test is a helpful screening test but is not diagnostic, and rarely will acid-fast bacilli (AFB) be found in the stool.

Radiologic studies are helpful but not necessarily diagnostic of the condition. Barium contrast enema performed by enteroclysis has been the traditional method of evaluation and diagnosis. Classic radiologic features include contracted terminal ileum with a wide, open ileocecal valve (Fleischner sign) and a narrow ileum opening into a contracted cecum (Sterlin's sign).[59] Ultrasound examination in ileal disease has shown a nonspecific finding of the "pseudokidney sign," suggesting an echogenic center surrounded by a sonolucent rim, correlating with a thickened bowel wall. Findings on CT scan include ascites, adenopathy, abscess, and additional thickened bowel.[60,61] Asymmetric bowel wall thickening and enlarged necrotic lymph nodes are suggestive of the diagnosis of tuberculous colitis.

Colonoscopy has emerged as the diagnostic modality of choice and allows diagnostic procedures such as biopsy and fine-needle aspiration for histopathology, AFB staining, and culture.[59] Macroscopically, the disease can be difficult to differentiate from Crohn's disease. Transverse ulcerations can be helpful. Histopathologic examination may reveal the presence of granulomas, caseous necrosis, and submucosal Langhans giant cells that are strongly suggestive of the diagnosis. Pathologic diagnosis may not always be accomplished by culture, and clinical, radiologic, and colonoscopic evidence suggestive of gastrointestinal tuberculosis warrants initiation of a therapeutic trial.

Treatment

Medical therapy is the mainstay of treatment, and surgery should be avoided if possible to give maximal time for the results of chemotherapy to be assessed. Medical therapy consists of two phases.[59] The induction phase consists of isoniazid, rifampin, pyrazinamide, and ethambutol or streptomycin taken daily for 2 months. The patient is then switched to the continuation phase, consisting of isoniazid and rifampin daily for 4 months. Prolonged continuation phase antibiotics (9 to 12 months) with the addition of ethambutol are necessary for resistant organisms. Surgery is indicated with specific complications. The most common complication is acute intestinal obstruction, followed by perforation, malabsorption, fistulae, and bleeding from an ulcer. Appropriate protective equipment is required in the operating room to minimize risk of transmission to the operative team.

CHLAMYDIA SPECIES

Chlamydia species are obligate, intracellular organisms that consist of three major subgroups: *C. psittaci*, *C. trachomatis*, and *C. pneumoniae. C. trachomatis* represents the most common sexually transmitted disease and can be subdivided into lymphogranuloma venereum (LGV) and non-LGV strains.[62]

Epidemiology

Asymptomatic infected persons are the main reservoir of continued transmission.

C. trachomatis is a common cause of proctitis in homosexual males practicing anoreceptive intercourse.[63] Approximately 5% of gay men are asymptomatic carriers of *C. trachomatis*.

Clinical Features

The most common presenting features are bloody diarrhea and mucopurulent anal discharge followed by tenesmus and rectal pain. Severity can be mild to severe ulcerative proctocolitis. Confusion with Crohn's disease is common because of the chronic diarrhea and potential for perianal fistula formation.[64] Progressive involvement of the bowel wall and replacement with fibrotic tissue lead to stricture formation.

Diagnosis

Culture of *C. trachomatis* can be obtained by stool or rectal swab culture onto McCoy cells. Many lubricating products used in the office setting contain bacteriostatic substances that may result in decreased culture yield. Colonoscopy reveals normal to moderate inflammatory changes with mucosal friability and small erosions in the distal 10 to 15 cm of the rectum.[64] Histopathologic specimens reveal granulomatous inflammation (noncaseating), inflammatory cell infiltrates, and crypt abscesses.

Treatment

Tetracycline or doxycycline is the current treatment of choice for uncomplicated *C. trachomatis* proctitis.

Fluoroquinolones or azithromycin can be considered in patients unable to tolerate tetracyclines.[4] Counseling should also occur with regard to the mode of transmission and any potential contacts.

NEISSERIA GONORRHOEAE

Neisseria gonorrhoeae is a gram-negative coccus organism occurring in pairs or clumps; upon microscopic examination, the organism appears as intracellular gram-negative diplococci.

Epidemiology

Gonorrhea is a common sexually transmitted infectious disease that involves mucous membranes of the urethra, vagina, and cervix. Rectal infection is most commonly seen in homosexual males and is transmitted through anoreceptive intercourse. Women may become infected through similar practices; however, vaginal discharge of infected secretions can infect rectal mucosa everted or exposed at defecation.[65]

Clinical Features

Symptoms begin approximately 1 week after exposure and consist of pruritus, mucopurulent discharge, rectal bleeding, and diarrhea.[66] Symptoms suggesting sexually transmitted disease in other locations may also be present. Asymptomatic rectal infection is prevalent, and the acute proctitis of the lower rectum (most commonly involved region) may reflect inoculum size or trauma during anal intercourse. Anoscopy may reveal mucopurulent exudate and inflammatory changes of the rectal mucosa, although differentiation from *C. trachomatis* or herpes simplex virus infection can be difficult.

Diagnosis

Confirmation of *Neisseria* organisms can be obtained by rectal swab culture inoculated onto selective chocolate agar (Thayer-Martin) incubated in carbon dioxide.[65,66]

Treatment

Therapy is directed against β-lactamase–producing strains of *N. gonorrhoeae*. Single-dose ceftriaxone, 125 mg intramuscularly, cures 99% of uncomplicated anorectal gonorrhoeae.[67] Fluoroquinolone use is an acceptable alternative.

CONCLUSION

Bacterial colitis–associated bloody diarrhea is commonly encountered in medical practice. A thorough understanding of epidemiologic factors including bacterial reservoirs, modes of transmission, and virulence factors is required for identification and treatment of these disease processes. *Campylobacter*, *Shigella*, *Salmonella*, *Escherichia coli*, and *Yersinia* are commonly encountered pathogens causing bacterial hemorrhagic enterocolitis as a result of fecal-oral or food and water contamination. *Chlamydia trachomatis* and *Neisseria gonorrhoeae* are sexually transmitted bacterial organisms that can cause proctitis. Stool studies are frequently positive for fecal leukocytes and red blood cells. Although nonspecific, this finding increases the yield for bacterial stool culture of enteric pathogens. Many of these illnesses are self-limiting, requiring only supportive care. Antibiotic-directed therapy is always indicated in high-risk individuals such as those at the extremes of age and those with immunosuppression and complicating diseases such as toxic megacolon, intestinal obstruction, perforation, and septicemia. Many of these organisms acquire antibiotic resistance; therefore, careful review of susceptibility is required to ensure adequate coverage and effective treatment.

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