

852 Psoas Abscesses The psoas muscle is another location in which abscesses are encountered. Psoas abscesses may arise from a hematogenous source, by contiguous spread from an intraabdominal or pelvic process, or by contiguous spread from nearby bony structures (e.g., vertebral bodies). Associated osteomyelitis due to spread from bone to muscle or from muscle to bone is common in psoas abscesses. When Pott's disease was common, *Mycobacterium tuberculosis* was a frequent cause of psoas abscess. Currently, either *S. aureus* or a mixture of enteric organisms including aerobic and anaerobic gram-negative bacilli is usually isolated from psoas abscesses in the United States. *S. aureus* is most likely to be isolated when a psoas abscess arises from hematogenous spread or a contiguous focus of osteomyelitis; a mixed enteric flora is the most likely etiology when the abscess has an intra-abdominal or pelvic source. Patients with psoas abscesses frequently present with fever, lower abdominal or back pain, or pain referred to the hip or knee. CT is the most useful diagnostic technique.

TREATMENT PSOAS ABSCESSSES

Treatment includes surgical drainage and the administration of an antibiotic regimen directed at the inciting organism(s).


Pancreatic Abscesses See Chap. 371.

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160 Acute Infectious Diarrheal Diseases and Bacterial Food Poisoning

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 Acute diarrheal disease is a leading cause of illness globally and is associated with an estimated 1.4 million deaths per year. Among children <5 years of age, diarrheal disease is second only to lower respiratory infection as the most common infectious cause of death. The incidence rate of diarrheal disease among children in low- and middle-income countries is estimated to be 2.9 episodes per child per year, for a total of 1.7 billion episodes annually. The morbidity from diarrhea is also significant. Recurrent intestinal infections are associated with physical and mental stunting, wasting, micronutrient

deficiencies, and malnutrition. In short, diarrheal disease is a driving factor in global morbidity and mortality.

The wide range of clinical manifestations of acute gastrointestinal illnesses is matched by the wide variety of infectious agents involved, including viruses, bacteria, and parasites (Table 160-1). This chapter discusses factors that enable gastrointestinal pathogens to cause disease, reviews host defense mechanisms, and delineates an approach to the evaluation and treatment of patients presenting with acute diarrhea. Individual organisms causing acute gastrointestinal illnesses are discussed in detail in subsequent chapters.

PATHOGENIC MECHANISMS

Enteric pathogens have developed a variety of tactics to overcome host defenses. Understanding the virulence factors employed by these organisms is important in the diagnosis and treatment of clinical disease.

INOCULUM SIZE

The number of microorganisms that must be ingested to cause disease varies considerably from species to species. For *Shigella*, enterohemorrhagic *Escherichia coli*, *Giardia lamblia*, or *Entamoeba*, as few as 10–100 bacteria or cysts can produce infection, while 10⁵–10⁸ *Vibrio cholerae* organisms must be ingested to cause disease. The infective dose of *Salmonella* varies widely, depending on the species, host, and food vehicle. The ability of organisms to overcome host defenses has important implications for transmission; *Shigella*, enterohemorrhagic *E. coli*, *Entamoeba*, and *Giardia* can spread by person-to-person contact, whereas under some circumstances *Salmonella* may have to grow in food for several hours before reaching an effective infectious dose.

ADHERENCE

Many organisms must adhere to the gastrointestinal mucosa as an initial step in the pathogenic process; thus, organisms that can compete with the normal bowel flora and colonize the mucosa have an important advantage in causing disease. Specific cell-surface proteins involved in attachment of bacteria to intestinal cells are important virulence determinants. *V. cholerae*, for example, adheres to the brush border of small-intestinal enterocytes via specific surface adhesins, including the toxin-coregulated pilus and other accessory colonization factors. Enterotoxigenic *E. coli*, which causes watery diarrhea, produces an adherence protein called *colonization factor antigen* that is necessary for colonization of the upper small intestine by the organism prior to the production of enterotoxin. Enteropathogenic *E. coli*, an agent of diarrhea in young children, and enterohemorrhagic *E. coli*, which causes hemorrhagic colitis and the hemolytic-uremic syndrome, produce virulence determinants that allow these organisms to attach to and efface the brush border of the intestinal epithelium.

TOXIN PRODUCTION

The production of one or more exotoxins is important in the pathogenesis of numerous enteric organisms. Such toxins include *enterotoxins*, which cause watery diarrhea by acting directly on secretory mechanisms

TABLE 160-1 GASTROINTESTINAL PATHOGENS CAUSING ACUTE DIARRHEA

Mechanism	Location	Illness	Stool Findings	Examples of Pathogens Involved
Noninflammatory (enterotoxin)	Proximal small bowel	Watery diarrhea	No fecal leukocytes; mild or no increase in fecal lactoferrin	<i>Vibrio cholerae</i> , enterotoxigenic <i>Escherichia coli</i> (LT and/or ST), enteroaggregative <i>E. coli</i> , <i>Clostridium perfringens</i> , <i>Bacillus cereus</i> , <i>Staphylococcus aureus</i> , <i>Aeromonas hydrophila</i> , <i>Plesiomonas shigelloides</i> , rotavirus, norovirus, enteric adenoviruses, <i>Giardia lamblia</i> , <i>Cryptosporidium</i> spp., <i>Cyclospora</i> spp., microsporidia
Inflammatory (invasion or cytotoxin)	Colon or distal small bowel	Dysentery or inflammatory diarrhea	Fecal polymorphonuclear leukocytes; substantial increase in fecal lactoferrin	<i>Shigella</i> spp., <i>Salmonella</i> spp., <i>Campylobacter jejuni</i> , enterohemorrhagic <i>E. coli</i> , enteroinvasive <i>E. coli</i> , <i>Yersinia enterocolitica</i> , <i>Listeria monocytogenes</i> , <i>Vibrio parahaemolyticus</i> , <i>Clostridium difficile</i> , <i>A. hydrophila</i> , <i>P. shigelloides</i> , <i>Entamoeba histolytica</i> , <i>Klebsiella oxytoca</i>
Penetrating	Distal small bowel	Enteric fever	Fecal mononuclear leukocytes	<i>Salmonella typhi</i> , <i>Y. enterocolitica</i>

Abbreviations: LT, heat-labile enterotoxin; ST, heat-stable enterotoxin.