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Diarrhea, Infectious

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food-borne infection Infection acquired via contaminated food.
incidence Rate of occurrence of an event.
traveler's diarrhea Infectious diarrhea acquired while traveling in endemic areas.
waterborne infection Infection acquired via contaminated water.

"Diarrhea" derives from the Greek words δια ρεω, meaning "flow through." Diarrhea occurs when the volume of the colonic fluid is greater than the absorptive capacity of this segment, as a result of impaired absorption and/or increased secretion. In infectious diarrheas, the abnormal function is brought about by microorganisms that colonize the intestinal mucosa and subvert normal gut physiology either directly or via enterotoxins.

INTRODUCTION

Diarrheal diseases are a major cause of morbidity and mortality around the world, especially in developing countries where children suffer the greatest brunt of infectious diarrhea, malnutrition, and death. Annually, approximately 5 million children and infants die worldwide due to diarrheal diseases. In North America, the rate per year is still 0.9 diarrheal episodes per child, and in special circumstances (daycare centers, institutions), the incidence is as high as 5 episodes per year. Fourteen hospital admissions per 1000 children younger than 12 months, per year, result from acute diarrhea. Among the adult population, most patients developing acute diarrhea are managed as outpatients or will not seek medical attention. However, 0.5 million hospital admissions per year, or 1.5% of all adult hospital admissions annually, are due to diarrhea. In developing countries, inadequate water supply, inefficient or nonexistent sewage removal systems, chronic malnutrition, and lack of access to oral rehydration are responsible for the high incidence of infectious diarrheal diseases. In the industrialized world, acute diarrhea is still one of the most frequent diagnoses in general practice and children, elderly, and immunocompromised patients are the most vulnerable individuals and account for the majority of these cases.

Regardless of the etiology, diarrhea is defined clinically as the occurrence of three or more episodes of loose stool or any loose stool with blood during a 24 h time period. Symptoms lasting less than 14 days represent acute diarrhea, whereas persistent diarrhea lasts more than 14 days but less than 4 weeks, and chronic diarrhea is defined by a duration of symptoms greater than 4 weeks.

Infectious diarrheas are miserable illnesses of overwhelming impact on the general survival of entire populations. Throughout history, thousands-strong armies have been defeated by raging diarrheal diseases: from the Greeks and Macedons under Alexander (Tucidides), to the Romans in the campaigns against the Gauls (Julius Caesar), to the Hundred Years War in 13th century Europe, to Napoleon, the Civil War in America, World War II, and the Vietnam War. Scores of previously healthy men suffered and died from the scourge of diarrhea and dysentery in all of these conflicts.

EPIDEMIOLOGY

Twenty years ago, 800 million to 1 billion episodes of infectious diarrhea and nearly 5 million deaths occurred per year worldwide, primarily in developing countries. Ten years later, survival had improved, but the incidence was virtually unchanged despite greater knowledge of the pathophysiology of diarrhea and greater intervention by the World Health Organization (WHO). Approximately 100 million episodes of acute diarrhea occur in the United States yearly, with an incidence of 1.2 to 1.5 diarrheal episodes per person-year. Medical costs/analyses show that 8.0 million Americans sought physician care for diarrhea yearly and 250,000 required hospitalization. Hospitalization and medical costs approached $560 million, whereas lost productivity totaled $200 million. Approximately another 8 million people sought physician care but were not hospitalized. These patients incurred $690 million in medical costs and $2 billion in loss of work hours. An estimated 90 million cases occurred in people who

did not seek physician care, costing nearly $20 billion in lost productivity. Approximately 90% of all these cases were presumably of infectious origin. Thus, the total cost estimate for diarrheal diseases exceeds $23 billion annually in the United States alone.

Although the elderly have an increased risk for death from diarrhea, death from diarrhea is rare among young children in industrialized countries. In fact, of all pediatric admissions for diarrhea, 0.05% resulted in death, compared with 3% in patients older than age 80. Increased age was the most important risk factor for death with an odds ratio of 52.6 (95% confidence interval, 37.0 to 76.9) for age 70 or older versus children >5 years. The national mortality figures for the 9-year period 1979–1988 in the United States show 51% of diarrheal deaths occurring in individuals older than age 74.

Acute infectious diarrhea is transmitted mostly through the fecal–oral route and by ingestion of contaminated water and food. Infection via the fecal–oral route occurs by direct contact with index cases, especially under conditions of crowding, such as daycare centers or nursing homes. Waterborne and foodborne outbreaks are another important source of disease transmission and result from general and/or individual failures in proper standards for the safe handling of foods. In most developing nations, acute diarrhea is endemic due to poor sanitation. Furthermore, epidemics of significant proportions often result from natural disasters in areas where water and food supplies are already chronically jeopardized. In some areas of the world, such as Asia, Africa, and Latin America, certain infectious diarrheas (e.g., cholera) have become ongoing pandemics lasting several decades, notwithstanding WHO efforts at eradication.

In most parts of the world, a definite seasonality is recognized in the incidence of acute diarrhea. In industrialized nations, the highest incidence of hospital admissions for diarrhea occurs in August and September and in the winter months. In developing nations with warmer climates and endemic conditions, variations in incidence occur from year to year in relation to precipitation indices and crop failures.

PATHOPHYSIOLOGY

Infectious diarrheas may be classified according to various criteria: duration, underlying mechanism, clinical presentation, etiology, and history. Table I summarizes the various criteria for classifying diarrheas in general and infectious diarrheas in particular.

In this section, infectious diarrheas are described according to the duration of the main gastrointestinal symptom.

Acute Infectious Diarrhea

Acute diarrheas last, by definition, less than 4 days and the majority are due to infectious agents. Most of these infections are self-limited and generally do not require medical intervention, unless severe dehydration and toxicity develop. However, immunocompromised patients, the elderly, and the very young may develop complications from enteric pathogens that warrant prompt and decisive medical intervention. A list of the major organisms involved in the etiology of acute infectious diarrheas is presented in Table II. Not listed is a type of acute enteritis, waterborne and of presumed infectious origin, that has been responsible for several outbreaks of traveler's diarrhea, known as Brainerd diarrhea. The etiologic agent of this disease still escapes definition.

Many of the acute infectious diarrheas observed worldwide are diagnosed in the course of local or epidemic outbreaks. Three major situations may be encountered: (1) waterborne infections; (2) Food-borne diarrhea; and (3) traveler's diarrhea. Whereas foodborne diarrhea is often associated with residual microbial toxins, waterborne and traveler's diarrheas are more often caused by active infection via the fecal–oral route. Table III summarizes the most common causes in these epidemiological situations.

A successful enteric pathogen possesses well-developed abilities to colonize, grow, and compete for nutrients in a crowded environment and to interact effectively with the host's enterocytes, inducing changes in the balance between absorption and secretion of water and electrolytes. In most gut infections, a pathogen enters via the oral route and colonizes an area of the

TABLE I Classification of Infectious Diarrheas According to Various Criteria

Duration	Mechanism	Clinical findings	Etiology	Patient's History
Acute	Secretory	Inflammatory	Bacteria	Age
Persistent	Nonsecretory	Noninflammatory	Viruses	Travel
Chronic			Unicellular parasites	Immunocompetence
			Worms	Food-/waterborne
				Postinfectious

TABLE II Common Causes of Acute Infectious Diarrhea

Bacteria	Viruses	Unicellular parasites	Worms
Salmonella	Rotavirus	*Giardia lamblia*	*Strongyloides stercoralis*
Shigella	Norwalk virus	*Entamoeba histolytica*	*Anchilostoma duodenalis*
Escherichia coli[a]	Calicivirus	*Cryptosporidium parvum*	*Necator americanus*
Yersinia enterocolitica	Adenovirus	*Cyclospora cayetanensis*	*Hymenolepsis nana*
Vibrio spp.	Astrovirus	*Microsporidia*[b]	*Heterophyes heterophyes*
Campylobacter	Coronavirus	*Isospora belli*	
Staphylococcus aureus	Herpes simplex virus	*Blastocystis hominis*	
Bacillus cereus	Cytomegalovirus	*Balantidium coli*	
Listeria monocytogenes		*Dientamoeba fragilis*	
Clostridium perfringens			
Clostridium difficile			
Aeromonas			
Plesiomonas			

[a] EIEC, enteroinvasive *E. coli*; ETEC, enterotoxigenic *E. coli*; EPEC, enteropathogenic *E. coli*; EAEC, enteroadhesive *E. coli*.
[b] The phylum includes *Microsporidium, Encephalitozoon, Pleistophora, Trachipleistophora, Nosema, Vittaforma, Brachiola.*

intestine. Exceptions to this paradigm are the ingestion of preformed toxins. Pathogens produce diarrhea by three basic mechanisms: (1) enterotoxins that induce active intestinal secretion (*Vibrio cholerae*, *Staphylococcus aureus*, *Bacillus cereus*, *Clostridium botulinum*, rotavirus); (2) cytotoxic mediators (most bacteria, parasites); and (3) invasins promoting endocytosis, with subsequent tissue invasion and mucosal injury [*Shigella, Salmonella*, enteroinvasive *Escherichia coli* (EIEC)]. In addition to direct effects by microorganisms and their products, enteropathogens induce intestinal damage indirectly via the mucosal inflammatory response, which involves secretion of various powerful mediators of secretion and apoptosis. A summary of the current knowledge about the pathogenesis of the most common acute infectious diarrheal syndromes is shown in Fig. 1.

TABLE III Agents Associated with Outbreaks of Acute Infectious Diarrheas

Waterborne	Foodborne	Traveler's
Vibrio cholera	*Campylobacter*	*Escherichia coli*
Campylobacter	*Salmonella*	*Campylobacter*
Salmonella	*E. coli*	*Salmonella*
Shigella	*Shigella*	*Shigella*
E. coli	*Staphylococcus aureus*	*Aeromonas/ Plesiomonas*
Giardia	*Clostridia*	*Giardia*
Entamoeba hystolytica	*Vibrio parahaemolyticus*	*Cryptosporidium*
Cryptosporidium	Caliciviruses	*Cyclospora*
Cyclospora	Norwalk virus	*Isospora*
Microsporidia	*Giardia*	*Rotavirus*
Enteroviruses	*Cryptosporidia*	

On the basis of these three mechanisms, acute infections present as watery, noninflammatory diarrheal syndromes or inflammatory diarrheal syndromes. The majority of watery, noninflammatory diarrhea cases are self-limited diseases characterized by low-grade fever, nausea, vomiting, large-volume diarrhea, and the absence of blood and leukocytes in the stools. This presentation is typically reported in patients infected with enterotoxigenic *Escherichia coli*, *V. cholerae*, clostridial and staphylococcal food poisoning, rotavirus, Norwalk virus agent, *Giardia lamblia*, and *Cryptosporidium*. On the other hand, the inflammatory diarrheal syndrome is characterized by frequent, small-volume stools that may contain blood and leukocytes, tenesmus, fever, and severe abdominal pain. The most common microorganisms causing this syndrome include *Salmonella*, *Shigella*, *Campylobacter*, enterohemorrhagic *E. coli*, EIEC, *Clostridium difficile*, *Entamoeba histolytica*, and *Yersinia*. Table IV describes the basic biologic, pathophysiologic, and clinical characteristics pertinent to the most common enteric pathogens.

Persistent Infectious Diarrhea

Persistent diarrhea is emerging as a major world health problem. Children are more likely to develop persistent diarrhea and suffer malnutrition, wasting, and immunocompromise as a consequence. Persistent diarrhea is defined by loose–soft stools occurring at increased frequency and lasting for more than

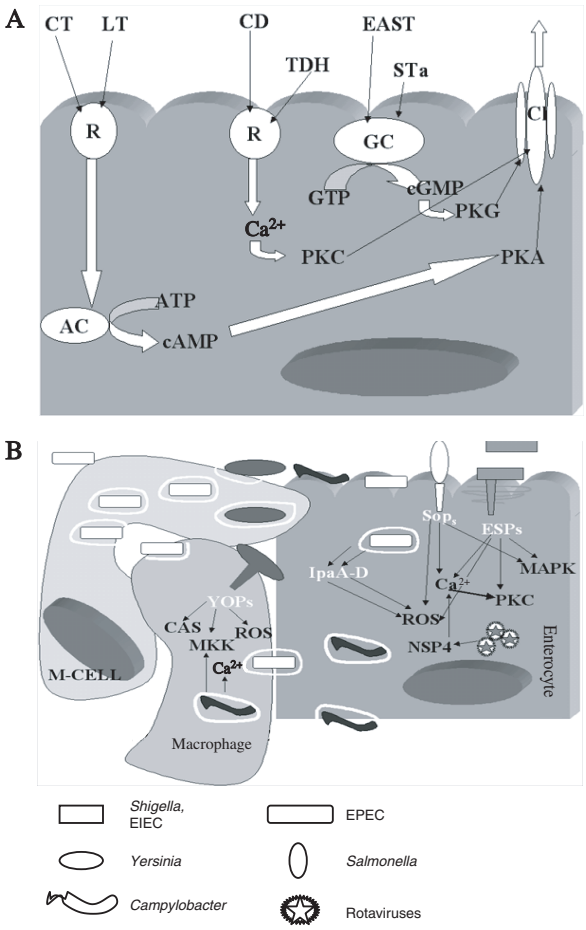

FIGURE 1 Infectious diarrhea: mechanisms of action of major enteric bacteria and viruses. Enteric pathogens can induce intestinal injury with consequent diarrhea in three ways: (1) by producing enterotoxins that interact with receptors located on the gut epithelial cells and evoke anion secretion, such as *V. cholera*, EPEC, EAEC, STEC, *C. difficile*, and *S. aureus* (A); (2) by invading the gut epithelium and M cells, thus altering the cell cytoskeleton and activating intracellular pathways through virulence factors. Organisms that lead to diarrhea through these mechanisms include EIEC, *Shigella*, EPEC, *Salmonella*, and rotaviruses (B); (3) by invading mucosal macrophages and inducing inflammatory responses leading to intestinal epithelial damage and anion secretion. *Campylobacter* and *Yersinia* use this mechanism (B).

2 weeks after the end of an acute episode of gastroenteritis. Persistent infectious diarrhea may result from multiple repeated infections, or persistent infection by the original organism, or as the so-called postgastroenteritis syndrome. Overall, the incidence of persistent infectious diarrhea is equally distributed in industrialized countries, including the United States, and developing nations. Table V lists the most common infectious agents associated with persistent diarrhea.

Postinfectious persistent diarrhea is a poorly defined syndrome that occurs as a sequela of an acute episode with definite infectious etiology. Patients may develop mild to severe degrees of malabsorption, from lactose intolerance to inability to absorb proteins, fat, and sugars, as well as permanent blunting of villi as assessed by histopathology. The condition is characterized by watery, malodorous stools and progressive wasting.

Chronic Infectious Diarrhea

Chronic infectious diarrhea occurs mostly in immunocompromised patients. After an acute infectious episode, patients sometimes develop chronic symptoms that are independent of the etiologic agents of acute diarrhea (irritable bowel syndrome with diarrhea, or, occasionally, ulcerative colitis). Table VI lists the most common agents isolated from cases of chronic infectious diarrhea.

By definition, chronic diarrhea lasts more than 4 weeks and patients developing this syndrome quite often are hospitalized and have undergone antibiotic therapy for other reasons. Elderly, human immunodeficiency virus (HIV)/acquired immunodeficiency syndrome (AIDS), transplant, and cancer patients are easy targets for reinfections or reactivation of only partially subdued infectious organisms. In addition to the causes listed above, bacterial overgrowth can occur in areas of bowel stasis or impaired bowel motility. Postsurgery patients, diabetics, posttrauma patients, and intensive care patients are more likely targets of chronic infectious diarrheas from bacterial overgrowth.

SPECIAL HOSTS

The Elderly

Infectious diarrhea causes high morbidity and mortality among the aging population worldwide. Multiorgan complications from an acute episode of infectious diarrhea are also more frequent among the elderly. Life expectancy in the United States has risen from an average of 45 years in the 20th century to 75 years at present. By the year 2025, 22% of the U.S. population will be older than age 65. Gastrointestinal physiology and gut colonization change constantly with aging and contribute in a significant way to increasing the susceptibility of elderly people to enteric infections. Furthermore, the gastric acid barrier in the elderly is impaired. The most frequently isolated organisms and most deadly in elderly patients with diarrhea are *C. difficile*, *Salmonella*, and toxigenic *E. coli*. These three agents top the list of

TABLE IV Acute Infectious Diarrhea: Biology, Pathophysiology, and Clinical Findings by Etiologic Agent

Organism	Microbiology	Pathophysiology	Epidemiology	Clinical findings
Bacteria				
Salmonella: S. enteritidis; S. typhimurium; S. typhi	Invasive, gram-negative rod; 2000 serotypes	Nontoxigenic; fimbriae, SPI-1 gene-encoded effectors (inv, spa, sic, sip, TTSS, etc.), plasmid-encoded effectors (sop A-E2, hsp)	Salmonellosis in USA: 1.4 million cases/year; >500 death/year; Typhoid fever in USA: 400 cases/year; worldwide: 16 million cases with 600,000 death/year	Salmonellosis: fever, abdominal cramps, diarrhea; typhoid fever: fever, headache, malaise, vomiting; uncommon diarrhea
Shigella	Invasive, gram-negative rod; 4 species	Toxigenic; pili, flagella, TTSS, Mxi-Spa, IpaA-C, IpgD effectors	USA: 450,000 cases/year (*S. sonnei*), worldwide: *S. flexneri* and *S. dysenteriae*; fatality rate 5–15%	Fever, abdominal pain, malaise, watery or bloody diarrhea
Escherichia coli	Gram-negative rod			
EIEC	Invasive	Pili, TTSS, IpaC, Esps, adhesin		Watery or bloody diarrhea
EPEC	Noninvasive or limited invasion; typical and atypical strains	TTSS, Bfp, intimin, EspF	USA: unknown Worldwide: unknown	Watery diarrhea, nausea, vomiting
EAEC	Adherent, limited invasion	Fimbriae, TTSS, EAST, cytotoxin		Watery diarrhea
ETEC	Noninvasive, adherent	TTSS, Cfs, LT, ST	USA: 80,000 cases/year	Watery diarrhea
EHEC	Noninvasive, adherent	Toxigenic; Intimin, Stx 1 and 2	USA: 70,000 cases/year; 61 deaths	Bloody diarrhea
Yersinia enterocolitica	Gram-negative rod	TTSS, Ysc, Yop effectors	USA: 1 case/100,000/year (culture-confirmed)	Fever, abdominal pain, bloody diarrhea
Vibrio:	Non invasive, gram-negative sickle-shaped			
V. cholera		CtxA, ctxB, zot, ace, tcpA effectors, toxR, tcpP	USA: 0–5 cases/year; pandemic in Asia, Africa, Latin America	Profuse watery diarrhea, vomiting
V. parahaemolyticus/ V. vulnificus		TxA/B	USA: 3000/95 cases/year; 7/35 deaths/year	Watery diarrhea, abdominal cramps
Campylobacter jejuni	Invasive, gram-negative	Type IV secretion	USA: 2.4 million cases/year; 124 deaths/year	Fever, abdominal cramps, diarrhea (often bloody)
Staphylococcus aureus	Noninvasive, gram-positive cocci	Staph enterotoxin	USA: true incidence unknown	Nausea, vomiting, watery diarrhea
Bacillus cereus	Rod-shaped, spore-forming	Stable emetic toxin, heat- and acid-labile enterotoxin	USA: 2% food-borne outbreaks/year;	Emetic syndrome and diarrheal syndrome
Listeria monocytogenes	Invasive, gram-positive	LLO, ActA	USA: 2500 cases/year; 500 deaths/year	Fever, abdominal pain, watery diarrhea
Clostridium perfringens	Noninvasive, gram-positive, spore-forming	CpA, cpE	USA: 2% of all acute infectious diarrheas	Nausea, vomiting, diarrhea

Clostridium difficile	Noninvasive, gram-positive, spore-forming	NeuroTx A, cytoTxB	USA: 25% of all antibiotic-associated diarrheas	Watery diarrhea, fever, anorexia, abdominal pain
Aeromonas/Plesiomonas	Gram-negative rod	Cytotoxic enzymes	USA: rare	Watery or bloody diarrhea, abdominal cramps
Viruses				
Rotavirus	*Reoviridae*, dsRNA	NSP4 enterotoxin	USA: 3 million cases/year; worldwide: l million deaths/year	Vomiting, watery diarrhea
Norwalk virus	*Caliciviridae*, ssRNA	Unknown	30% of all cases of diarrheas in children >1 year	Nausea, vomiting, diarrhea
Calicivirus	*Caliciviridae*, ssRNA	Unknown	1.5% of all cases of viral gastroenteritis	Nausea, diarrhea
Adenovirus	*Adenoviridae*, dsDNA, type 40 and 41	Unknown	<1% of all cases of viral gastroenteritis	Fever, vomiting, diarrhea
Astrovirus	*Astroviridae*, ssRNA	Unknown	1.5% of all cases of viral gastroenteritis	Watery diarrhea
Coronavirus	*Coronaviridae*, ssRNA	Unknown	<1% of all cases of viral gastroenteritis	Vomiting, diarrhea
Herpes simplex virus	*Alphaherpesvirinae*, dsDNA	Unknown		Fever, tenesmus, watery or bloody diarrhea
Cytomegalovirus	*Betaherpesvirinae*, dsDNA	Unknown	Rare in immunocompetent; 16% in solid organ transplants; 5% in HIV/AIDS	Fever, malaise, abdominal tenderness, diarrhea
Parasites, unicellular				
Giardia lamblia	Diplomonadida, cysts and throphozoites, 5 chromosomes (5K genes)	VSP (analogy with sarafotoxins)	USA: 2.5 million cases/year; endemic in developing countries	Diarrhea, flatulence, abdominal cramps, malabsorption
Entamoeba histolytica	Entamoebidae, cysts and throphozoites, 14 chromosomes	Cysteine proteinases	USA: infrequent; worldwide: 400 million infections/year, 100,000 deaths/year	Asymptomatic, mild gastroenteritis, or bloody dysentery
Cryptosporidium parvum	Alveolata, oocysts and throphozoites, ongoing genome sequencing	Peptidases	USA: 2% of the general population; worldwide: unknown	Asymptomatic or watery diarrhea
Cyclospora cayetanensis	Alveolata, oocysts and sporozoites, ongoing genome sequencing	Unknown	USA: unknown, outbreak related to contaminated berries; worldwide: unknown, endemic in Guatemala and Peru	Fever, watery diarrhea, fatigue
Microsporidia	Microsporidia, spores and schizontes	Unknown	Unknown	Asymptomatic or watery diarrhea
Isospora belli	Alveolata, oocysts and throphozoites	Unknown	Unknown	Asymptomatic or watery diarrhea
Blastocystis hominis	Stramenopiles	Unknown	Unknown	Asymptomatic or watery diarrhea
Balantidium coli	Alveolata, cysts and throphozoites	Unknown	USA: rare	Asymptomatic to bloody diarrhea

continues

TABLE IV Acute Infectious Diarrhea: Biology, Pathophysiology, and Clinical Findings by Etiologic Agent (*continued*)

Organism	Microbiology	Pathophysiology	Epidemiology	Clinical findings
Dientamoeba fragilis	Parabasalidea, throphozoites, no cysts	Peptidases	USA: infrequent	Nausea, malaise, mucous diarrhea, abdominal pain
Worms				
Strongyloides stercoralis	Helminths, nematodes, filariform larvae, can complete life cycle in humans	Organism effectors and host responses	USA: 4% prevalence in Appalachian States; worldwide: 100 million cases/year, 60% prevalence in tropical countries	Mild to severe diarrhea, malaise, fatigue, malnutrition
Anchilostoma duodenalis, Necator, americanus	Helminths, nematodes, filariform larvae and eggs	Organism effectors and host responses	USA: uncommon; worldwide: in tropical countries prevalence is increasing due to climate changes	Mild to severe diarrhea, abdominal pain, weight loss
Hymenolepsis nana, H. diminuta	Cestodes, cysticercoids and adult worms in humans (fleas and beetles intermediate hosts)	Organism effectors and host responses	USA: rare; worldwide: Latin America	Abdominal cramps, mucous diarrhea upon rupturing of villus by cysticercoids
Heterophyes heterophyes	Trematodes, metacercariae and eggs (fish and snails intermediate hosts)	Organism effectors and host responses	USA: rare; worldwide: endemic in Egypt, Middle East, and Far East	Asymptomatic to severe mucous diarrhea, intestinal wall granulomas

Note. SPI, *Salmonella* pathogenicity island; TTSS, type 3 secretory system; Tx, toxin; Cfs, cytotoxic factors; EAST, enteroaggregative heat-stable toxin; LT, heat-labile toxin; ST, heat-stable toxin.

TABLE V Agents Associated with Persistent Infectious Diarrhea

Bacteria	Parasites
Salmonella	*Cryptosporidium parvum*
Shigella	*Cyclospora cayetanensis*
Escherichia coli[a]	*Giardia lamblia*
Yersinia	*Entamoeba hystolytica*
Campylobacter	*Balantidium coli*
Clostridium	*Dientamoeba fragilis*

[a] EIEC, enteroinvasive *E. coli*; EAEC, enteroadherent *E. coli*; ETEC, enterotoxigenic *E. coli*; EPEC, enteropathogenic *E. coli*.

outbreaks in long-term and short-term care facilities and *Salmonella* by itself accounts for more than 50% of cases and more than 80% of deaths in food-borne outbreaks in nursing homes.

HIV/AIDS

More than 50% of HIV/AIDS patients in the United States experience infectious diarrhea and this estimate may approach 100% in developing countries where the HIV epidemic is currently raging unchecked. These patients are more likely to develop persistent or chronic diarrhea after an acute episode because of their impaired immunity, with a significant increase in morbidity and mortality. Table VII lists the most common causes of infectious diarrhea in AIDS patients.

The American Gastroenterological Association (AGA) has published a set of general guidelines for the management of chronic diarrhea in AIDS patients. At least three sets of stool samples should be secured for common enteric bacteria and parasites, including microsporidia, cryptosporidia, and *C. difficile*. Febrile patients with diarrhea should have blood cultures for common enteric bacteria. Patients with CD4 lymphocyte counts of <100 cells/mm are at high risk for disseminated mycobacterial infection.

TABLE VI Agents Associated with Chronic Infectious Diarrhea

Bacteria	Parasites
Campylobacter	*Amoeba*
Mycobacterium tuberculosis	*Cryptosporidium*
Aeromonas	*Giardia lamblia*
Plesiomonas	*Isospora*
Salmonella	*Cyclospora*
Clostridium difficile	*Strongyloides*
	Trichuris
	Schistosoma

CLINICAL AND LABORATORY FINDINGS

The most important finding in patients presenting with acute diarrhea is the degree of volume depletion, i.e., dehydration. Postural changes in blood pressure are a reliable sign of dehydration. Fever, abdominal tenderness, increased bowel sounds, or blood on rectal examination should alert the physician to acute infectious diarrhea.

Microscopic examination of a stool sample or rectal swab is a traditional and helpful tool in the rapid, bedside investigation of diarrheal illness. The specimen is placed on a glass slide and mixed thoroughly with two drops of methylene blue. The presence of ova, cysts, and/or leukocytes may point directly to a diagnosis. The AGA guidelines on managing acute diarrhea indicates empiric antimicrobial therapy in the case of positive fecal leukocytes in a febrile patients.

Endoscopy has limited utility in the investigation of acute infectious diarrhea and is not cost-effective. It may have a place, however, in cases of persistent or chronic diarrhea.

PREVENTION

Preventative measures against infectious diarrhea must include improvements in sanitation (water supply, sewer systems, housing), education of the general population and, where applicable, vaccination campaigns. Unfortunately, no effective vaccines are available for the organisms that cause infectious diarrheas, with the exception of typhoid fever.

TREATMENT

Most acute diarrheal illnesses are self-limited and no specific therapy is required. Water and electrolyte loss can be prevented or treated with oral fluid–electrolyte solutions. Intravenous saline–glucose solutions are recommended in cases of moderate to severe dehydration. Glucose in the intestinal lumen facilitates the absorption of sodium and the cotransport mechanism for these solutes appears to be unhampered by infection with microorganisms or by their toxins.

Antimotility therapy should be reserved for severe cases and chronic diarrheas and avoided in infants and children. Antibiotic or antiviral treatment should be considered in moderate to severe cases in which a microbiological diagnosis is obtained or strongly

TABLE VII Agents Associated with Diarrhea in AIDS Patients

Bacteria
Shigella
Salmonella
Escherichia coli
Campylobacter
Yersinia enterocolitica
Clostridium difficile
Clostridium perfringens
Staphylococcus aureus
Aeromonas
Plesiomonas
Bacillus cereus
Vibrio parahemolyticus
Mycobacterium avium complex
Treponema
Viruses
Cytomegalovirus
Adenovirus
Herpes simplex virus
Fungi
Hispolasma capsulatum
Blastocystis hominis
Parasites
Giardia lamblia
Entamoeba histolytica
Cryptosporidium
Isospora
Cyclospora
Enterocytozoon bieneusi
Encephalitozoon intestinalis
Balantidium coli

suspected. In immunocompromised patients with febrile diarrheas, empirical antibiotics should be promptly initiated after securing adequate culture specimens to define an etiology.

See Also the Following Articles

AIDS, Gastrointestinal Manifestations of • Anti-Diarrheal Drugs • *Campylobacter* • Cholera • *Cryptosporidium* • Cytomegalovirus • Diarrhea • Foodborne Diseases • Food Poisoning • Food Safety • Giardiasis • Rotavirus • *Salmonella* • *Shigella* • Traveler's Diarrhea

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