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Vomiting, Diarrhea, Constipation, and Gastroenteritis

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KEYWORDS

• Vomiting • Diarrhea • Constipation • Gastroenteritis

VOMITING

Vomiting is a reflex composed of the coordinated series of motor and autonomic responses that results in the forceful expulsion of gastric contents through the mouth activated by humoral or neuronal stimuli.[1] Vomiting should not be confused with regurgitation or retching. Regurgitation is the return of esophageal contents to the hypopharynx with little effort, whereas retching is unsuccessful vomiting due to absence of gastric contents or closure of the upper esophageal sphincter. Vomiting continues to be a major problem throughout the world. An analysis of the 2006 National Hospital Ambulatory Medical Care Survey (NHAMCS) found nausea and vomiting as the chief complaint for 3.7% of Emergency Department (ED) visits.[2] Pregnant women are particularly afflicted, as 56% of women experience vomiting during their pregnancies.[3] The cost of nausea and vomiting to society is high, with a 2002 study estimating a yearly cost of $3.4 billion for food-related and gastrointestinal infections.[4]

Differential Diagnosis and Initial Approach

Nausea and vomiting are symptoms of a wide variety of underlying conditions that may involve almost any organ system. Accordingly, when faced with a patient with vomiting, the differential is broad and includes gastrointestinal, infectious, central nervous system, drug reaction, and cardiac origins (**Table 1**). While the most common causes of nausea and vomiting are acute gastroenteritis, febrile systemic illness, and drug effects,[5] one must also consider certain critical and emergent diagnoses such as Boerhaave syndrome, intracranial bleed or raised intracranial pressure, meningitis, diabetic ketoacidosis, myocardial ischemia, sepsis, gonadal torsion, abdominal inflammatory processes, bowel obstruction, adrenal insufficiency, and toxic ingestions.

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Table 1
Differential diagnosis of vomiting

System	Disease
Gastrointestinal	Gastroenteritis (viral or bacterial), gastric outlet obstruction, small bowel obstruction, gastroparesis, cyclic vomiting syndrome, irritable bowel syndrome, neoplasm, peptic ulcer disease, gastritis, gastroesophageal reflux disease, hepatitis, cholecystitis, biliary colic, appendicitis, mesenteric ischemia, Crohn disease, pancreatitis, diverticulitis, volvulus, intussusception, pyloric stenosis, intestinal perforation
Central nervous system	Migraine, tumor, hemorrhage, infarction, congenital malformation, abscess, meningitis, demyelinating disorders, hydrocephalus, pseudotumor cerebri, seizure, Meniere disease, labyrinthitis, motion sickness, anxiety, depression, psychogenic vomiting, anorexia nervosa, bulimia nervosa, postconcussive syndrome
Drugs (only most common offenders listed)	Chemotherapy, nonsteroidal anti-inflammatory drugs (NSAIDs), opioids, antiarrhythmics, antihypertensives, diuretics, antibiotics, hormonal preparations, anticonvulsants, oral hypoglycemics, vitamins, ethanol
Metabolic and endocrinologic	Pregnancy, diabetic ketoacidosis, uremia, hyperparathyroidism, hypoparathyroidism, Addison disease, porphyria, uremia, alcoholic ketoacidosis
Cardiac	Cardiac ischemia, myocardial infarction, hypotension, hypertension, congestive heart failure
Other	Pain, gonadal torsion, renal colic, postoperative, overdose and toxins, emotional response, sepsis

Despite the heterogeneity in potential etiology, a thorough history and physical examination can often focus the approach and narrow the differential diagnosis. The American Gastroenterological Association (AGA) recommends a pragmatic 3-step approach to the management of the patient with nausea and vomiting.[6] After a complete history and physical examination, the clinician should first correct any complications of vomiting such as hypokalemia, metabolic alkalosis, hypovolemia, ketosis, or vitamin deficiencies. Second, the underlying cause should be sought with the intention of initiating targeted therapy. The third step is the initiation of treatment strategies to suppress the symptoms. Although there are no clinical guidelines in the initial workup of the patient with vomiting, pregnancy testing should be considered in women of reproductive age. Serum electrolytes, complete blood count, liver function tests, lipase, urinalysis, and electrocardiogram may also be considered depending on the clinical situation.

Antiemetics

Dopamine receptor antagonists

When a specific etiology of vomiting is diagnosed, targeted intervention toward the underlying process is important. For the empiric treatment of undifferentiated vomiting, the pharmaceutical armamentarium consists of many different drug classes primarily directed at 5 neurotransmitter receptor sites (**Table 2**). Before the recent increase in use of the serotonin 5-hydroxytryptamine-3 (5-HT_3) antagonists, the mainstay of therapy had been dopamine receptor antagonists. The literature on this class of

Table 2
Antiemetics

Drugs	Mechanism of Action	Special Considerations
Prochlorperazine, promethazine, chlorpromazine	Phenothiazines: predominantly D2-dopamine antagonism, also M1-muscarinic and H1-histamine antagonism	High incidence of extrapyramidal reactions, may cause hypotension, promethazine black box warning for children <2 y
Metoclopramide, domperidone	Benzamides: D2-dopamine antagonism, weak 5-HT_3 antagonism at higher doses, enhances acetylcholine at neuromuscular junction	Prokinetic properties, domperidone does not cross blood-brain barrier, metoclopramide pregnancy category B
Droperidol, haloperidol	Butyrophenones: D2-dopamine and α antagonism	Second-line agents, droperidol black box warning due to QT prolongation and torsades
Diphenhydramine, dimenhydrinate, cyclizine	H1-histamine antagonism	Primarily used for motion sickness, sedating
Ondansetron, granisetron, dolasetron, palonosetron	Selective 5-HT_3 antagonism	Favorable toxicity profile, high cost
Aprepitant, fosaprepitant	Selective NK1-substance P antagonism	Used for chemotherapy, synergistic effect with serotonin receptor antagonists and corticosteroids
Dexamethasone, methylprednisolone	Corticosteroid: inhibits inflammatory cytokines, produces glucocorticoid and mineralocorticoid effects	Prophylaxis for chemotherapy-induced vomiting
Lorazepam, alprazolam	Binds to benzodiazepine receptors, enhances GABA effects	Sedating, often used as adjunctive agent
Dronabinol, nabilone	Cannabinoids: exact mechanism unknown, possible interaction with vomiting control center	Multiple other effects, most studied in cancer patients

Abbreviations: 5-HT_3, 5-hydroxytryptamine-3; GABA, γ-aminobutyric acid.

medications is vast and at times contradictory. One of the most extensively studied drugs is metoclopramide (Reglan). Metoclopramide has been compared with prochlorperazine (Compazine), with some investigators finding similar efficacy[7,8] and others finding a modest benefit of one over the other.[9–11] Metoclopramide is often recommended for pregnant patients, as it is the only medication in this class with a pregnancy category B rating.

Similar inconclusive findings have been found when comparing promethazine (Phenergan) and prochlorperazine. Recently, however, one randomized, double-blind

study of 84 ED patients found subjects who received promethazine had a treatment failure rate of 31% versus just 9.5% in the prochlorperazine group.[12] Of interest, both groups had a similar rate of akathisia although the promethazine group experienced increased drowsiness. Other studies have shown a higher rate of akathisia and dystonia with prochlorperazine compared with other antiemetics.[13,14] In an ED-based study of 229 patients receiving prochlorperazine, 16% developed akathisia and 4% developed acute dystonia.[15] Diphenhydramine (Benadryl) is the first-line choice to treat these reactions. A study of 82 patients with akathisia found diphenhydramine was effective in reducing akathisia from 9.8 to 1.2 on a scale of 0 to 17.[16] These findings have led some clinicians to administer diphenhydramine concurrently with prochlorperazine in an effort to prevent akathisia; however, this practice has not been validated with a randomized, placebo-controlled study.

As the literature does not consistently support one dopamine receptor antagonist over another, it is not surprising that practice patterns vary. A 2000 ED-based analysis found antiemetics to be used with the frequencies promethazine (55%), prochlorperazine (25.3%), metoclopramide (5.2%), and ondansetron (Zofran) (1.3%), reflecting the choice of antiemetic remains one of clinician preference.[17]

Serotonin 5-HT$_3$ antagonists

Over the past decade, there has been an increase in the use of 5-HT$_3$ antagonists due to a lower incidence of side effects. Although controversial, the 2001 black box warning by the Food and Drug Administration (FDA) of droperidol (Inapsine)[18] may also have contributed to shifting practice patterns. Of the currently approved drugs in the United States, ondansetron (Zofran), granisetron (Granisol, Kytril), and dolasetron (Anzemet) have all been shown to be equally effective and tolerated.[19–21] Palonosetron (Aloxi) differs from the others in this class by its longer half-life, and has been shown to reduce delayed chemotherapy-induced vomiting when compared with dolasetron.[22] Although the bulk of the research on this class of drugs comes from the oncology literature for chemotherapy-induced vomiting,[23,24] studies examining undifferentiated ED patients have recently been published. A randomized, placebo-controlled, double-blind trial found ondansetron was not superior to metoclopramide and promethazine in 163 patients presenting to the ED with undifferentiated nausea.[25] Furthermore, a 2008 study of 120 ED patients found no difference in the reduction of nausea between ondansetron and promethazine, however, the group that received ondansetron experienced less sedation.[26] A recent review article examined the evidence supporting the use of droperidol, promethazine, prochlorperazine, metoclopramide, and ondansetron for the treatment of nausea or vomiting in the ED. The investigators concluded that "based on the safety and efficacy of ondansetron, it may be used as a first-line agent for relief of nausea or vomiting for most patient populations in the ED."[27]

Pediatric Patients

Vomiting in the pediatric patient is an extremely common complaint in children presenting to the ED.[28] While most patients have a self-limiting disease process, vomiting may also be the presenting symptom for severe life-threatening conditions, and a thorough history and physical examination are therefore required to guide management. Clinicians have historically been cautious to prescribe antiemetics for children, a practice reinforced by the American Academy of Pediatrics' (AAP) recommendation to use oral rehydration as first-line therapy for both mildly and moderately dehydrated children with gastroenteritis.[29] In a recent study, 73 children ranging in age from 8 weeks to 3 years with moderate dehydration from viral gastroenteritis were

randomized to either oral rehydration therapy (ORT) or intravenous fluids. While nearly 50% of both groups were successfully rehydrated at 4 hours, subjects receiving ORT had shorter ED stays and were hospitalized less often. Based on their findings, the investigators opined that ORT was the preferred treatment option.[30]

Despite these recommendations, a survey found 36% of pediatricians believed vomiting was a contraindication to ORT.[31] The FDA's 2006 black box warning on promethazine for pediatric patients younger than 2 years may have contributed to the hesitancy to use antiemetics and subsequently avoid ORT in children. Nonetheless, the past decade has also seen an increase in 5-HT_3 antagonists in the pediatric population. A double-blind, placebo-controlled study from 2002 on intravenous ondansetron for gastroenteritis found a reduction in admission rates for pediatric patients presenting with vomiting and an initial serum carbon dioxide level of 15 mEq/L or more.[32]

Pharmacologic data demonstrating that orally administered ondansetron tablets are equivalent to its intravenous formulation have led to further investigations exploring whether intravenous access could be avoided. In 2006, investigators enrolled 215 children aged 6 months to 10 years treated in the ED for gastroenteritis-related dehydration. Compared with placebo, subjects who received ondansetron orally were less likely to vomit (14% vs 35%), had greater oral intake (239 mL vs 196 mL), and were less likely to require intravenous fluids (14% vs 31%).[33] Another pediatric study replicated these results, finding subjects who received oral ondansetron had a decreased need for intravenous fluids than those who received a placebo (21.6% vs 54.5%).[34] These results reinforce the practice of using oral ondansetron and ORT to treat pediatric patients with mild to moderate dehydration.

DIARRHEA

Diarrhea is classically thought of as a physical sign of a disease rather than a disease in itself; therefore, much of the pertinent literature focuses on its etiology and the supportive, empiric treatment of diarrhea. Nevertheless, while the majority of cases of diarrhea in the United States are self-limited, diarrhea continues to pose an enormous health challenge worldwide. The World Health Organization (WHO)[35] estimates approximately 4 billion cases of diarrhea worldwide per year, with such episodes responsible for a staggering 2.2 million deaths annually. Overall, in the United States, there are an estimated 211 million to 375 million cases of acute diarrhea, resulting in 900,000 hospitalizations.[36] Furthermore, diarrhea remains the most common and incapacitating symptom of patients with ulcerative colitis.[37] Diarrhea is an ailment that can be particularly severe in children, with the majority of the deaths worldwide caused by diarrheal illness occurring in children younger than 5 years old. A recent study found ED visits for pediatric patients with diarrhea nearly doubled from 1995 to 2004, with 25% of those presentations being due to rotavirus.[38,39] Diarrhea is also common in the military. More than three-quarters of troops deployed to Iraq and Afghanistan reported at least one diarrhea episode during their deployments, with 45% noting a decreased work performance for a median of 3 days.[40]

Although definitions vary, diarrhea is typically characterized as a change in normal bowel movements with the passage of 3 or more stools per day or at least 200 g of stool per day.[41] Acute diarrhea is defined as episodes lasting 14 days or less; persistent diarrhea lasts more than 14 days; and chronic diarrhea lasts for more than 30 days. Furthermore, diarrhea is broadly categorized as either secretory or osmotic. Osmotic diarrhea occurs when a nonabsorbable solute exerts an osmotic pressure effect across the intestinal mucosa, a process that produces excessive water output.

Secretory diarrhea, commonly caused by bacterial toxins or neoplasms that disrupt epithelial crypt cells in the gastrointestinal tract, is extremely difficult to control.

Differential Diagnosis

The differential diagnosis for diarrhea is broad, with several causes displaying overlapping signs and symptoms. A focused history including the onset, frequency, and character of the diarrhea (eg, presence of blood or mucus) as well as associated symptoms (eg, fever, vomiting), medical history (eg, human immunodeficiency virus [HIV], inflammatory bowel disease), medications, and travel history may aid in narrowing the differential. Nevertheless, there are several clinically noteworthy causes of diarrhea that have exceptional treatment regimens as well as important clinical ramifications to consider.

Clostridium difficile, which affects approximately 3 million patients yearly in the United States with a mortality rate of 1% to 2.5%,[42] is caused by a disruption of normal intestinal flora,[43] and is responsible for 15% to 20% of antibiotic-related cases of diarrhea.[44] Severe *C difficile* infection may result in life-threatening complications such as toxic megacolon, intestinal perforation, sepsis, or death. Furthermore, diarrhea caused by *C difficile* may present with severe abdominal pain, high fever, and more than 10 watery stools per day; however, as it is common among elderly patients many or all of these signs and symptoms may be absent. One study found 15% of patients with diarrhea hospitalized at an academic center tested positive for *C difficile,*[45] while during times of outbreak more than 50% of patients in an affected ward may become colonized.[46] Of interest, although *C difficile* historically has not been thought of as a pediatric illness, recent evidence suggests the contrary. A pediatric ED-based study found that of specimens that underwent complete testing, 12.4% tested positive for *C difficile* toxin,[47] and nearly 3% of children tested positive for *C difficile* toxin in another similar study from France.[48] Recently a new disease pattern, community-onset *C difficile*–associated diarrhea, has emerged, and may occur without exposure to the typical risk factors including antibiotic usage.[49]

Several agents have been implicated in the increased incidence of *C difficile*, including usage of antibiotics and proton pump inhibitors (PPIs). In one recent study, *C difficile* diarrhea among hospital inpatients was associated with the use of PPIs (9.3% of patients receiving PPIs vs 4.4% who did not receive PPIs) and receipt of 3 or more antibiotics.[50] Removing the inciting antibiotic treats up to 25% of cases of *C difficile* diarrhea.[51] Antibiotic treatment regimens have traditionally used oral metronidazole (Flagyl) or vancomycin (Vancocin) for 14 days; however, for recurrent *C difficile* infection some experts recommend oral tapered-pulsed vancomycin (125 mg once a day for 1 week, 125 mg 3 times a day for 1 week, 125 mg every day for 1 week, 125 mg every other day for 2 weeks, 125 mg every third day for 2 weeks).[52]

Traveler's diarrhea, which affects 20% to 50% of individuals traveling from developed to developing countries and 4% to 9% of individuals traveling from developing to developed countries, is typically caused by enterotoxigenic *Escherichia coli* (ETEC) and enteroaggregative *E coli*, which bind to the intestinal mucosa to cause diarrhea typically without fever.[53] Incubation periods for ETEC last between 10 hours and 3 days followed by 3 to 5 days of illness.[54] ETEC produces a noninvasive toxin that causes severe watery diarrhea, abdominal cramps, nausea, and (infrequently) fever.[55] *Shigella* species and *Salmonella* species are other important bacterial pathogens. *Campylobacter jejuni*, a bacteria that poses additional hazards because it has been implicated with acute cases of myocarditis, has emerged as another important cause of traveler's diarrhea.[56] Electrolyte disturbances for patients with traveler's diarrhea are rare, and therefore laboratory work is usually unnecessary.[57] Treatment of

traveler's diarrhea is centered on antibiotic therapy, such as ciprofloxacin (Cipro), trimethoprim-sulfamethoxazole (Bactrim; resistance common), azithromycin (Zithromax), and rifaximin (Xifaxan).[58] If the patient with suspected traveler's diarrhea has more than 2 unformed stools per day, bloody stools, or fever (>37.8°C), treatment with antibiotics is advised.[59] A short, single-day course of ciprofloxacin, 500 mg twice a day, is usually successful at stopping the illness within 24 hours,[60] although other sources recommend a 3-day course of ciprofloxacin or rifaximin.[61] Prophylactic administration of antibiotics for those traveling to developing countries is not typically recommended. However, in one placebo-controlled trial performed on United States travelers in Mexico, subjects who took rifaximin prophylactically had significantly reduced rates of diarrhea (53.70% for those taking placebo vs 14.74% for those taking rifaximin).[62] Nonetheless, *C difficile* and *Helicobacter pylori* must also be on the differential among the traveler afflicted with diarrhea. Noninfectious origins need to be considered if there is no response to antimicrobials or antiparasitics and there is a protracted course. Nonbacterial causes include enteric viruses, viral hepatitis, influenza, giardia, *Cryptosporidium*, *Cyclospora*, *Entamoeba*, *Strongyloides*, and other less common parasites.

Cryptosporidiosis, which typically affects immunocompromised individuals (particularly HIV-infected persons) and may also affect immunocompetent persons (usually children younger than 5 years), causes diarrhea lasting 1 to 2 weeks, and may develop into life-threatening illnesses. Although it appears that nitazoxanide (Alinia) reduces the load of parasites and may be useful in immunocompetent persons, a recent review found there is no evidence for effective agents in the management of cryptosporidiosis.[63]

Pediatric Patients

The differential diagnosis for the pediatric patient with diarrhea is broad and includes pathogens such as *E coli*, *Campylobacter*, *Shigella*, *Salmonella*, and viruses. However, in recent years a newer strain of *E coli* has emerged in the pediatric population: enteroaggregative *E coli*. In a study of 1327 children younger than 1 year with acute gastroenteritis, enteroaggregative *E coli* was isolated significantly more often in inpatients (4.7%) and ED patients (10.0%) than from well children (1.4%).[64] Viral gastroenteritis caused by rotaviruses is another concern in the pediatric population. Among middle- and low-income countries, it is estimated rotaviruses are responsible for 600,000 to 870,000 pediatric deaths per year, resulting in up to 6% of all mortality in children younger than 5 years.[65] The majority of these deaths were due to dehydration, underscoring the importance of rehydration therapies for children. Implementation of the rotavirus vaccine shows promise. A 2004 review of 64 trials conducted on 21,070 children found the vaccine's effectiveness at preventing diarrhea caused by rotavirus ranged from 22% to 89%.[66]

Evaluation of hydration status often dictates the treatment of pediatric patients with diarrhea. While acute appendicitis must always remain on the differential diagnosis of the child with diarrhea, digital rectal examinations and nasogastric tubes rarely provide additional actionable information for pediatric patients.[66] Treatment of the pediatric patient with diarrhea centers on supportive care, with encouragement of fluids for mild to moderate cases, and in severe cases intravenous or nasogastric fluid replacement. Educating parents in the appropriate treatment of their child's diarrhea is crucial. Whereas 52% of parents treated their child's diarrhea with appropriate rehydration fluids and solutions, 13% of parents used treatments not recommended in the current Centers for Disease Control (CDC) guidelines, typically using antidiarrheal agents and fluids high in simple sugars.[67]

Developed in 1975, the WHO standard oral rehydration solution consists of a high content of sodium (90 mmol/L) and has been found to be effective in the treatment of dehydration from acute gastroenteritis regardless of the etiology of the diarrhea.[68] Several newer products with lower sodium levels, including the WHO revised formula (75 mmol/L) and Pedialyte (45 mmol/L), may be better tolerated among pediatric patients. However, these products may not be appropriate for patients suffering from diarrhea caused by cholera, one of the most serious types of diarrheal disease that can cause rapid electrolyte loss. In an analysis of 7 trials of patients with cholera, the investigators found an increased number of patients with hyponatremia treated with hypoosmolar solutions compared with standard oral rehydration solutions, although the outcomes were similar.[69]

Elderly Patients

Elderly patients afflicted with diarrhea tend to have longer hospital stays (7.4 days in patients older than 75 years versus 4.1 days in those patients 20 to 49 years old) and a higher mortality.[70] Age greater than 65 years is also considered an independent *C difficile* risk factor.[71] In one ED-based study of 174 patients with diarrhea, it was found that age greater than 40 years with constant abdominal pain and diarrhea was predictive of a surgical etiology for their symptoms.[72] Taken together, these factors should prompt the clinician to at times take a more aggressive and perhaps more comprehensive approach in attempting to search for the origin of diarrhea in the elderly patient.

Treatment

Rehydration and electrolyte replacement remain cornerstones of treatment for patients with diarrhea. To accomplish this, the "BRAT" diet (bananas, rice, apple sauce, and toast) is often recommended, although evidence supporting its practice is limited. Loperamide (Imodium) has been shown to be efficacious in reducing the symptoms of diarrhea in undifferentiated patients with mild symptoms[73]; however, there is scant evidence regarding its safety profile in patients with moderate or severe diarrhea.[74] A recent review did not find conclusive evidence supporting or refuting the usage of antimotility agents and adsorbents in controlling diarrhea in people with HIV/AIDS,[75] thus reinforcing the need for adjunct treatments such as fluid replacement. Nevertheless, one meta-analysis found that when combined with antibiotic therapy, loperamide was more efficacious than antibiotics alone in decreasing illness duration for adult patients with traveler's diarrhea.[76] Antidiarrheal agents are not recommended in the treatment of pediatric patients with diarrhea, as they have potentially serious side effects in this population.[77]

Antibiotics are the mainstay of treatment for patients with a suspected bacterial cause for their diarrheal symptoms. A study of 139 patients presenting with severe diarrhea characterized by one of either profuse watery diarrhea with dehydration, passage of stools containing mucus and blood, temperature greater than 38.4°C, passage of more than 6 soft stools in 24 hours, duration of illness of more than 48 hours, severe abdominal pain in a patient older than 50, or diarrhea in the elderly, found single-dose quinolone therapy shortened the duration of symptoms and was equally efficacious when compared with a 5-day antibiotic regimen.[78]

Probiotics, which are found in yogurts, fermented milks, and dietary supplements, may help treat diarrheal diseases. In one randomized, double-blind, placebo-controlled study, consumption of a 100-g drink containing *Lactobacillus casei*, *Lactobacillus bulgaricus*, and *Streptococcus thermophilus* twice daily during a course of antibiotics and 1 week after the antibiotic was finished resulted in an absolute risk

reduction of 21.6% for the occurrence of antibiotic-associated diarrhea.[79] Another study noted probiotic organisms may be beneficial for 3 problems common in the elderly: undernutrition, constipation, and the capacity to resist infection.[80] A systematic review of the literature on probiotics, which examined 23 studies with 1917 participants, found probiotics reduced the risk of diarrhea at 3 days and the mean duration of diarrhea by 30 hours.[81]

Prevention

Most preventive measures aimed at limiting the spread of diarrheal diseases focus on improving the quality of available water sources. Two studies found the addition of household-based water filters reduced the prevalence of diarrhea by 60% in Columbia[82] and by 70% in rural Bolivia.[83] Further, one study found treating turbid water in rural Kenya with a disinfectant resulted in a 19% absolute reduction in the prevalence of diarrhea.[84] Communicable and diarrheal diseases are also major concerns for disaster-affected populations in camp settings. In treated households in Liberia, disinfectants reduced diarrheal prevalence by 83% compared with control households.[85] Other developing countries have instituted hand-washing campaigns. One, in urban squatter settlements in Pakistan, found campaigns promoting hand-washing reduced the incidence of diarrhea by 53% among children younger than 15 years.[86] A recent review, which examined 33 trials with more than 53,000 participants, found that interventions focused on improving the quality of drinking water were effective in preventing diarrhea, with interventions aimed at the household level more effective than those aimed at the source.[87]

CONSTIPATION

Constipation is the most common digestive complaint in the United States, affecting up to 27% of the North American population. Although constipation tends to be associated with increasing age,[88] children also may experience this problem. One review of 4157 children younger than 2 years found the prevalence of constipation was 2.9% in the first year of life and 10.1% in the second year of life. While the majority of these cases were diagnosed as functional constipation, in 1.6% of cases underlying disease was responsible.[89] Constipation is also the most common cause of acute abdominal pain in children. A study of 962 children seen in a pediatric office found 9% had a complaint of abdominal pain; chronic constipation was diagnosed in 35% of those patients and acute constipation was diagnosed in 13%. A surgical condition was found in only 2% of the children with abdominal pain.[90] Furthermore, pregnant women are also disproportionately afflicted with constipation, with 25% of healthy women experiencing symptoms during their pregnancy and up to 3 months postpartum.[91]

Etiology

The cause of constipation is often multifactorial. While a 2007 review revealed a small number of publications addressing the etiologic factors of this very common problem,[92] it was suggested that insufficient dietary fiber intake, inadequate fluid intake, decreased physical activity, side effects of drugs, hypothyroidism, sex hormones, and colorectal cancer obstruction may be responsible for constipation.[92] Furthermore, the cause of constipation may also be related to abnormal bowel motility, anatomical rectal disorders, neurological disorders, or psychosocial issues (**Table 3**). A thorough review of the patient's medications is advisable, as there are many different medications that secondarily contribute to constipation, such as calcium and iron supplements, opioids, anticonvulsants, antipsychotics, and

Table 3
Etiology of constipation

Category	Cause
Abnormal motility	Slow-transit constipation, irritable bowel syndrome
Anatomic disorders	Anal fissure, hemorrhoids, rectal polyps, rectocele, rectal stenosis, fistulas, colonic or rectal neoplasm
Drugs	Calcium, iron, opioids, anticonvulsants, antipsychotics, antihistamines
Neurologic	Hirschsprung disease, spinal cord injury, multiple sclerosis, diabetes mellitus, Parkinson disease
Endocrine	Hypothyroidism, pregnancy, hypercalcemia, diabetes mellitus
Psychosocial	Depression, anxiety
Systemic	Scleroderma, amyloidosis, lupus

antihistamines.[88] Constipation resistant to simple measures may be caused by painful anorectal conditions, irritable bowel syndrome (IBS), slow transit constipation, or obstructive defecation. Obstructed defecation is a broad term used to describe the inability to empty stool from the rectum, which may result from functional, metabolic, mechanical, or anatomical problems.[93] Mechanical and anatomical disorders causing obstructive defecation include Hirschsprung disease, rectocele, rectoanal intussusception, enterocele, sigmoidocele, and rectal prolapse.[94] Studies have found that obstructive defecation is a significant problem for middle-aged women. Obstructive defecation was self-reported and defined by difficulty in passing stool, hard stool, straining for more than 15 minutes, or incomplete evacuation, occurring at least weekly. In this study of 2109 subjects, 12.3% of women reported obstructive defecation at least once weekly. Risk factors correlated with patients developing obstructive defecation included a history of IBS, vaginal or laparoscopic hysterectomy, unemployment, use of 3 or more medications, symptomatic pelvic organ prolapse, history of urinary incontinence surgery, or other pelvic surgeries.[95]

IBS, characterized by chronic abdominal pain and altered bowel habits without a clearly defined organic cause,[96] affects 10% to 15% of North Americans.[97,98] Attempts to standardize the diagnosis of IBS have been made, and the American Gastroenterology Association recommends clinicians use the Rome III criteria, last revised in 2005, to diagnose IBS.[99] These criteria require the presence of recurrent abdominal pain or discomfort at least 3 days per month as well as 2 or more of the following: improvement with defecation, onset associated with a change in form of stool, or onset associated with a change in frequency of stool. The Rome III diagnostic criteria for IBS must be fulfilled for 3 consecutive months with symptom onset at least 6 months before diagnosis.[100] Other symptoms that are not part of the Rome criteria but support the diagnosis of IBS include defecation straining, urgency, a feeling of incomplete bowel movement, passing mucus, and bloating. Four subtypes of IBS are recognized: IBS with constipation (hard stools $\geq$25% and loose stools <25% of bowel movements), IBS with diarrhea (loose stools $\geq$25% and hard stools <25% of bowel movements), mixed IBS (hard stools $\geq$25% and loose stools $\geq$25% of bowel movements), and unsubtyped IBS (insufficient abnormality of stool consistency to meet the other subtypes).

Pharmacologic intervention must be tailored to the specific subtype of IBS. While antidepressant therapy has been explored as treatment for IBS, a trial of 51 patients

randomized to placebo, imipramine, or citalopram found none of these agents significantly improved global IBS end points.[101] Antibiotics, specifically rifaximin, have also been tried in the treatment of IBS. A recent study of 80 patients randomized to rifaximin or placebo for 10 days found the group that received rifaximin had a greater improvement of IBS symptoms and a lower bloating score.[102]

Presenting Signs and Symptoms

Studies show a discrepancy among how physicians and patients define constipation, although they have a similar understanding of the symptoms.[88] Patients typically describe constipation as straining to have bowel movements, lumpy or hard stools, incomplete evacuation, anorectal obstruction, and a decreased frequency of bowel movements. To establish a standard for defining constipation, the Rome III criteria were created by a consortium of representatives from 18 countries.[88,100] These criteria include the aforementioned signs and symptoms frequently described by patients, while requiring at least 2 of the following over 3 months' duration: fewer than 3 bowel movements per week, at least 25% of bowel movements involving manual maneuvers to disimpact, straining, passing hard stools, or a sensation of anorectal obstruction. The Rome criteria excludes patients with loose stools as well as those meeting diagnostic criteria for IBS, given that there are separate Rome criteria for the diagnosis of IBS as mentioned previously.

Diagnostic Evaluation

The physical examination may include a digital rectal examination to determine presence of stool impaction or blood in the stool. The clinician should be aware of what the American College of Gastroenterology (ACG) refers to as "alarm" signs or symptoms, which include fever, nausea, vomiting, weight loss of more than 10 pounds, anorexia, blood in stool, anemia, family history of colon cancer, onset of constipation after the age of 50 years, or acute onset of constipation in the elderly.[88,103] If any of these symptoms are present a workup is advised, including a complete blood count, basic metabolic panel, thyroid tests, and possibly colonoscopy. If these signs are absent, the ACG recommends empiric treatment of the constipation.[88,103]

Radiographic studies are sometimes used to help determine the etiology of constipation. However, in a 2005 review article of the pediatric literature, the investigators found conflicting evidence for an association between a clinical and a radiographic diagnosis of constipation. The investigators therefore do not recommend performing routine abdominal films on pediatric patients presenting with constipation.[104]

Management and Treatment

The management of constipation depends on the degree to which the symptoms affect the patient's daily life, patient preference for type of treatment, efficacy of treatments tried in the past, and the provider's clinical judgment. If, for example, the patient is impacted then an enema or manual disimpaction is indicated. The ACG Chronic Constipation Task Force guidelines state exercise may help patients with constipation by reducing gastrointestinal transit time. Further, increasing water and fiber in the diet can increase frequency of bowel movements.[92,103,105] However, patient satisfaction surveys show dissatisfaction with initial treatment regimens of lifestyle and dietary changes for chronic constipation,[105] which highlights the importance of a multipronged approach in treating constipation.

When lifestyle changes fail, the options for medical treatment include bulking agents, osmotic agents, stimulant laxatives, and enemas (**Table 4**). Stool softeners are surface-acting agents that function as detergents, allowing water to interact

more effectively with stool. Docusate sodium (Colace) is a stool softener that is frequently prescribed for the treatment of chronic constipation; however, there are insufficient data to support its use.[106] One placebo-controlled crossover trial of docusate calcium (Surfak) versus placebo demonstrated no differences in stool consistency or frequency between the 2 groups.[107] Another trial, which was a multicenter, randomized, double-blind study of 170 patients with chronic constipation, found psyllium was superior to docusate sodium for increasing the stool water content and frequency of bowel movements.[108]

Nevertheless, the ACG recommends osmotic laxatives to treat constipation if an increase in water and dietary fiber fails.[103,109] One double-blind, multicenter study randomized 100 patients who presented with chronic medication-induced constipation to receive either polyethylene glycol (PEG) 3350 (Miralax) or placebo for 28 days. The standard dosing of PEG, 17 g mixed with 8 ounces of water daily, was given to patients in the treatment group. PEG 3350 was found to be superior to placebo (78.3% vs 39.1%) in relieving constipation. Diarrhea and flatulence occurred more frequently with PEG treatment, although not to a statistically significant extent from placebo.[110] Based on this study and others supporting PEG's efficacy at improving stool frequency and consistency, the ACG Task Force gave PEG as well as lactulose (Cholac) grade A recommendations.[103,105]

Nevertheless, when osmotic laxatives fail to provide relief of symptoms of constipation, stimulant laxatives may be prescribed. Stimulant laxatives include compounds containing senna or bisacodyl, and are thought to act by stimulating the sensory nerve endings of the colonic mucosa. The FDA has approved these agents for treatment of occasional constipation; however, they should be used only as needed and for a brief time (<1 week), due to concerns regarding side effects with chronic use such as

Table 4
Medications used to treat constipation

Type	Agent	Mechanism
Bulking	Psyllium (Metamucil)	Increases stool bulk and intestinal motility, shortens transit time
	Methylcellulose (Citrucel)	Same as above
	Polycarbophil (FiberCon)	Same as above
	Docusate sodium (Colace)	Facilitates mixture of stool fat and water, softens stool
Osmotic	Lactulose	Osmotically active nonabsorbable sugars pull fluid into the gut
	Sorbitol	Same as above
	Polyethylene glycol (Golytely, Miralax)	Same as above
Stimulants	Bisacodyl (Dulcolax)	Stimulates the myenteric plexus, increasing intestinal motility
	Anthraquinones (Peri-Colace)	Same as above
	Senna (Senokot, Ex-lax)	Same as above
	Magnesium (milk of magnesia, magnesium citrate)	Shortens colonic transit time
	Glycerin suppository	Local rectal stimulation
Enemas	Tap water	Colonic distention prompts defecation
	Soap suds	Same as above, bowel wall irritant
	Monophosphate (Fleets)	Same as above, osmotic effect in small intestine, stimulates peristalsis

abdominal cramping, fecal incontinence, electrolyte imbalances, and reduced colonic motility.[88,105] One study, which evaluated sennosides (Sennakot) alone versus sennosides plus docusate sodium in the treatment of hospitalized oncologic patients, found that the sennosides group required fewer alternative laxative therapies (40% in the sennosides group versus 57% in the sennosides plus docusate sodium group) to treat constipation.[111] As there are no placebo-controlled trials of stimulant laxatives, insufficient data exist to make a recommendation about the effectiveness of stimulant laxatives in patients with chronic constipation.[106] A drug that has recently emerged for the treatment of chronic constipation is lubiprostone (Amitiza), which works by activating chloride channels that in turn increase secretion of intestinal fluid.[105,109] Patients treated with lubiprostone in phase 3 clinical trials experienced a median increase of 3 or 4 spontaneous bowel movements per week after 1 month of treatment.[112]

Treating constipation in pediatric patients is also challenging, although studies have demonstrated superior efficacy of PEG in this population as well.[113–117] In one study of 100 children aged 6 months to 15 years with constipation who received PEG or lactulose for 8 weeks, the investigators found a significant increase in the mean number of defecations per week in both groups. In terms of complete relief of symptoms after 18 weeks, however, 56% of patients who received PEG 3350 were successfully treated compared with 29% of patients who received lactulose.[114] Other studies have been performed to establish the most effective dose of PEG.[108,109] The results showed that 95% of patients receiving a higher dose (1–1.5 g/kg/d) achieved disimpaction versus 55% of patients receiving a lower dose (0.25–0.50 g/kg/d). However, diarrhea and bloating were more common in the higher-dose group.[117] A second study on PEG for children with constipation found low-dose PEG (0.2 g/kg/d) was successful in 77% of patients, mid-dose PEG (0.4 g/kg/d) was successful in 74% of patients, and high-dose PEG (0.8 g/kg/d) was successful in 73% of patients. All were more successful than placebo (42% success rate).[118] Nevertheless, a recent review stated that even though PEG achieved more treatment success compared with other laxatives, the studies were not of high enough quality to suggest laxative treatment is better than placebo in children with constipation.[119]

GASTROENTERITIS

Gastroenteritis is defined as a syndrome of vomiting, diarrhea, or the combination of both, that begins abruptly in otherwise healthy individuals.[120] Although the symptoms of vomiting and diarrhea convey a broad differential, it is clinically important to consider the diagnosis of gastroenteritis in patients with these symptoms for public health reasons. Worldwide, infectious gastroenteritis is a leading cause of morbidity and mortality.[120] In the United States, the highest incidence of infectious gastroenteritis is in patients younger than 5 years, whereas severe disease leading to hospitalization and resulting in mortality is most frequently observed in patients older than 60 years.[121] Even so, approximately 10% of hospitalizations in children younger than 5 years are caused by gastroenteritis and dehydration, accounting for nearly 220,000 hospitalizations yearly.[122] Gastroenteritis has many causes, including viral, bacterial, parasitic, and noninfectious (**Table 5**).

Norwalk Virus

The most prominent cause of acute gastroenteritis is viruses. Noroviruses account for more than 90% of the outbreaks in the United States, and affect both children and adults.[123,124] Outbreaks occur more commonly in cold-weather climates and in places where people are closely confined, such as schools, nursing homes, hospitals, and

Table 5
Gastroenteritis etiology

Viral (50%–70%)	Bacterial (15%–20%)	Parasitic (10%–15%)	Others	Drug-Associated
Norovirus	*Shigella*	Giardia	Ciguatera	Antibiotics
Calicivirus	*Salmonella*	Amebiasis	Scombroid	Laxatives
Rotavirus	*Campylobacter*	Cryptosporidium	—	Colchicine
Adenovirus	*Yersinia*	Cyclospora	—	Quinidine
Parvovirus	*Escherichia coli*	—	—	Sorbitol
Astrovirus	*Vibrio cholera*	—	—	—
Coronavirus	*Aeromonas*	—	—	—
Pestivirus	*Bacillus cereus*	—	—	—
Torovirus	*Clostridium difficile*	—	—	—
—	*Clostridium perfringens*	—	—	—
—	*Listeria*	—	—	—
—	*Mycobacterium avium-intracellulare* (MAI)	—	—	—
—	*Providencia*	—	—	—
—	*Vibrio parahaemolyticus*	—	—	—
—	*Vibrio vulnificus*	—	—	—

cruise ships.[125] The primary mode of transmission is through fecal-oral spread, but the virus can also be transmitted by respiratory droplet contact or ingestion of contaminated food or water. Up to 30% of exposed individuals shed the virus before developing the illness, and patients with underlying illnesses or immunocompromised states may continue to do so long after the illness resolves.[126] Noroviruses survive in a variety of temperatures, remaining live on environmental surfaces, in recreational and drinking water, and on raw fruits and vegetables. Although patients may develop illness year-round, outbreaks tend to peak during periods of cold weather.[124] Norovirus illness usually presents with both vomiting and diarrhea, as well as abdominal cramps, malaise, myalgias, and chills. Symptom onset is sudden, with vomiting being more common in children and diarrhea more common in adults.[124]

Fever, which is typically low grade, is present in 50% of patients. Symptoms usually last 24 to 60 hours and are typically mild and self-limited, while severe disease may develop in debilitated, elderly, or immunocompromised individuals.[123] Of particular concern, noroviruses may be associated with necrotizing enterocolitis (NEC). In one study of an outbreak of 8 infants with NEC in a neonatal intensive care unit, investigators found 4 (50%) of the infants had stool samples that tested positive for norovirus.[127]

Rotavirus

Although norovirus infection is the most common cause of gastroenteritis outbreaks in people of all ages worldwide, group A rotavirus is the leading cause of diarrheal illness in children younger than 5 years.[128] A cohort study from Europe of 2928 children younger than 5 years with more than 3 loose stools per day for more than 2 weeks found 43.4% of stool samples were positive for rotavirus.[129] A similar incidence was

found in the United States. In a study of 516 children younger than 3 years with acute gastroenteritis, the investigators found 44% had rotavirus-positive stool samples.[130] Although rotavirus is more common in children, it can also affect adults. In a 4-year prospective study of 683 adults with acute diarrhea, 14% of subjects tested positive for rotavirus.[131] There is a wide spectrum of disease severity for adults presenting with rotavirus, from mild vomiting, diarrhea, or both, to dehydration and severe systemic disease. Vomiting is present in 90% of cases and 30% of patients have a fever (>39.0°C). Finally, while the illness is usually worst in the first 24 hours it is typically mild and self-limited in immunocompetent adults.[132]

Salmonella

Salmonella infection is the most common cause of bacterial gastroenteritis in the United States, with more than 95% of subjects infected by contaminated food. Usually the source is raw or undercooked eggs, but the bacteria may also be found in meats, unpasteurized dairy products, fruits, vegetables, and peanuts.[133–137] Transmission may also occur via contact with infected animals, such as turtles.[138] *Salmonella* infection has a short incubation period of approximately 6 to 48 hours. Symptoms typically persist for 24 hours to 1 week and may include vomiting, diarrhea, crampy abdominal pain, and fever. In patients infected with *Salmonella*, resistance can be a problem and susceptibility testing is recommended. While antibiotics are thought to increase carrier states in patients with *Salmonella* infection, in selected patients, or in patients with severe illness, recommended antibiotics include third-generation cephalosporins or fluoroquinolones.[139]

Campylobacter jejuni

C jejuni is the most common cause of bacterial gastroenteritis worldwide and the second most common in the United States after *Salmonella* infection. The CDC reported an incidence of *C jejuni* infection of 13.02 per 100,000 persons in 2009.[140] The highest incidence of disease is among children younger than 5 years. Although *C jejuni* infection may be acquired from contaminated drinking water or exposure to infected farm or domestic animals, 50% of cases are associated with the handling and consumption of undercooked poultry.[141] *Campylobacter* infection develops 1 to 10 days postexposure. The illness may start with a prodrome of fever, malaise, chills, and headache before the onset of abdominal symptoms, which include watery but sometimes bloody diarrhea, abdominal pain, nausea, and vomiting. Symptoms typically resolve within 5 days, but in some cases may persist for several weeks.[141] For patients infected with *C jejuni*, treatment with erythromycin (Erythrocin) or azithromycin (Zithromax) is recommended. Fluoroquinolones are no longer advised as there have been increasing resistance patterns, possibly resulting from the usage of fluoroquinolones for farm animals.[141,142]

Campylobacter infection has also been associated with the development of postinfectious Guillain-Barré Syndrome (GBS), with an incidence of 1 per 1000 individuals. Serological surveys have found anti–*C jejuni* antibodies in patients with GBS, a finding consistent with recent infection. Further, a high proportion of patients have *C jejuni* in their stools when they develop GBS. Finally, GBS has been shown to be more severe and more likely to be irreversible when it is preceded by *C jejuni* infection.[143]

Vibrio parahaemolyticus

Whereas gastroenteritis caused by the organism *Vibrio parahaemolyticus* is common in Japan, the CDC reports a total of just 4500 cases per year in the United States.[140] The organism lives in oysters, clams, and crabs, and is transmitted by the ingestion of

contaminated saltwater seafood or direct exposure of an open wound to seawater. Cases in the United States have predominantly been linked to the consumption of raw oysters.[144] *V parahaemolyticus* has a 6-hour to 4-day incubation period and presents with the sudden onset of severe watery diarrhea, vomiting, abdominal cramping, and fever. Based on susceptibility testing of an outbreak of 10,000 patients infected with *V parahaemolyticus* in Chile in 2005, it is best treated with tetracycline (Sumycin), ciprofloxacin, or chloramphenicol. Of note, investigators found the organism was universally resistant to ampicillin.[145]

Shigella

Shigella infection primarily affects people in developing countries, and the majority of cases are in children younger than 5 years.[146] The incidence of *Shigella* in the United States in 2009 was 3.99 per 100,000.[140] The bacteria are transmitted mainly through person-to-person contact but may also be acquired from food, water, flies, and feces. *Shigella* invades the cells of the colonic epithelium, and the shiga toxin induces local inflammation, which in turn produces hemorrhagic colitis. Following an incubation period from 1 to 6 days, patients develop fever, crampy abdominal pain, and diarrhea, which often contains blood and mucus. Infants, on the other hand, present more often with nonbloody stool and lack of fever.[147] Although symptoms caused by *Shigella* infection are typically self-limiting and resolve within 2 to 3 days, most clinicians treat *Shigella* with antibiotic therapy. Fluoroquinolones are the mainstays of therapy, while azithromycin, trimethoprim-sulfamethoxazole (Bactrim), ampicillin, and ceftriaxone (Rocephin) are other options.

Yersinia

Yersinia is a prominent infection worldwide, but caused only 0.32 cases per 100,000 persons in the United States in 2009.[140] A primary risk factor for acquiring *Yersinia* is the consumption of contaminated foods, in particular raw pork.[148] *Yersinia* infection presents with a gradual onset of symptoms from several days to 1 week, which include bloody diarrhea, fever, vomiting, and severe right lower quadrant abdominal pain that may mimic appendicitis. Approximately 20% of patients also present with pharyngitis.[149] Treatment regimens for *Yersinia* include trimethoprim-sulfamethoxazole, fluoroquinolones, gentamycin, tobramycin, amikacin (Amikin), or cefotaxime (Claforan). *Yersinia*-infected individuals have a risk of developing bacteremia, liver or spleen abscesses, suppurative appendicitis, peritonitis, intussusception, and toxic megacolon.

Escherichia coli

E coli infections are categorized as enterohemorrhagic (O157:H7), enterotoxigenic (traveler's diarrhea), enteropathogenic (nontoxin mediated, uses an adhesin to attach and efface intestinal cells), or enteroinvasive. *E coli* 0157:H7 primarily affects children younger than 10 years and elderly patients, with an incidence of 0.99 per 100,000 persons in the United States in 2009.[140] Transmission has been linked to the consumption of undercooked beef, contaminated drinking water, unpasteurized milk,[150,151] and from fecal contamination of raw vegetables and unpasteurized apple juice.[152,153] *E coli* 0157:H7 bacteria cause a hemorrhagic colitis due to a shiga-like cytotoxin that destroys the colonic microvilli. Patients infected with *E coli* may develop an acute onset of watery diarrhea, which may progress to bloody diarrhea, abdominal cramps, and vomiting. Fever is typically absent or low grade. Approximately 6% of patients with *E coli* 0157:H7 infection, particularly those younger than 5 years and elderly patients, develop hemolytic uremic syndrome.[154] This risk is increased with bloody diarrhea, leukocytosis, fever, and possibly the use of antimotility agents.[155,156]

Outbreaks

Gastroenteritis outbreaks have been studied to determine their causes. One study examined patients hospitalized with community-acquired gastroenteritis in Berlin, Germany. The investigators found *Campylobacter* in 35% of specimens, norovirus in 23%, *Salmonella* in 20%, rotavirus in 15%, and a noninfectious cause in 8% of patients, supporting the need to remain diligent in looking for other causes of diarrhea even in an outbreak. In this study, length of hospital stay (median: 5.5 days) was independent of the pathogen, but was associated with patients who had underlying medical conditions.[157] Another study evaluated 29 acute gastroenteritis outbreaks in childcare centers. Stool specimens from symptomatic children and environmental surface swabs found offending pathogens included rotavirus (17% of outbreaks), norovirus (10%), astrovirus (10%), and sapovirus (7%). In 3 of the outbreaks, 10% of patients were found to have multiple viruses responsible for their infection, highlighting the importance of surveillance monitoring during these occurrences.[128]

Assessment and Diagnostic Evaluation

The duration and severity of the patient's diarrhea and vomiting should be assessed, along with their fluid intake, urine output, and overall mental status. Malnourished and immunocompromised patients are more likely to have serious outcomes. Stool cultures in the ED are typically reserved for patients with severe illness or for patients presenting with diarrhea in times of community-wide outbreaks. While fecal leukocytes are 70% sensitive and 50% specific for detecting inflammation in studies examining the infectious etiology of diarrhea, white blood cells in the stool may also be present in other conditions such as ulcerative colitis and Crohn disease.[158] Sensitivities can be increased to 83% by testing for fecal calprotectin.[159] Nonetheless, clinicians may consider sending a stool sample if there is blood or mucus in the stool, persistent diarrhea of more than 2 weeks' duration, or to help exclude an intestinal infection.

Laboratory testing for patients with vomiting and diarrhea caused by viral illnesses is typically not helpful, as these patients may not demonstrate markers of infection in their blood work or stool cultures. However, in one study of patients with known norovirus illness, investigators found leukocytosis with a neutrophil predominance was common. Furthermore, 64% of subjects tested positive for fecal leukocytes. This finding was surprising, as it had been thought that leukocytosis and stool leukocytes were rare in patients with norovirus-induced gastroenteritis.[160]

Management

The mainstay of therapy for acute gastroenteritis is supportive care. No specific antiviral therapy exists for viral gastroenteritis. Rehydration and electrolyte replacement are the most important aspects of treatment. Severely ill, immunocompromised, or very young children with suspected bacterial infections should receive empiric treatment with antibiotics.[161] To help estimate the degree of fluid loss in children, investigators in Canada have created the Clinical Dehydration Scale (CDS) to identify pediatric patients with severe dehydration. The scale assesses 4 characteristics: general appearance, eyes, mucous membranes, and tears. If all of these are normal, the score is zero, and the child is determined to have no dehydration. A score of 1 is given in each category if the child appears thirsty or restless, has slightly sunken eyes, sticky mucous membranes, or decreased tears. A score of 2 is given in each category if the child has a drowsy, limp, cold, or sweaty appearance, has very sunken eyes, dry

mucous membranes, or absent tears. A CDS score of 1 to 4 indicates some dehydration, whereas a score of 5 to 8 indicates moderate to severe dehydration.[162]

According to the CDC, children weighing less than 10 kg should receive 60 to 120 mL of oral rehydration solution per episode of vomiting or diarrheal stool, and those weighing more than 10 kg should receive 120 to 240 mL oral rehydration solution in addition to their daily requirements.[28,132] The original standard WHO oral rehydration solution had an osmolality of 311 mOsm/kg; however, in 2002 the formulation was changed to a lower osmolality solution (245 mOsm/kg) with lower concentrations of glucose (75 mmol/L) and sodium (75 mEq/L) based on several studies demonstrating a reduced osmolality solution, diminished stool volume, and the duration of diarrhea.[163] Most commercial oral rehydration solutions contain 2% to 3% carbohydrate. Common household fluids such as tea, fruit juice, sports drinks, and soft drinks have too little sodium along with a higher carbohydrate and osmolality content than suggested, and should therefore be avoided when attempting to hydrate a child with diarrhea.[28,29] In children with severe dehydration (more than 10% body weight loss), intravenous fluids are recommended. A 20 mL/kg bolus of intravenous fluid is suggested except in the case of a malnourished infant, where 10 mL/kg as a starting resuscitative amount is recommended to avoid overhydration or heart failure. After the initial intravenous therapy, 100 mL/kg oral rehydration solution over 4 hours or D5½NS (dextrose 5% in 0.45% normal saline) intravenously at a rate of twice maintenance may be administered.

Suggested indications for hospital admission for children with gastroenteritis include severe dehydration, neurological involvement, toxic state or shock, inability to tolerate oral rehydration, potential for surgery, failure of treatment despite oral rehydration therapy, or uncertain diagnosis. Providers should also consider admission for children younger than 2 months, febrile infants younger than 6 months with bloody stool, children with immunodeficiency or malnutrition, or if there is an inability to take care of the child at home.[132]

SUMMARY

Patients commonly present to the ED with symptoms of vomiting, diarrhea, constipation, and gastroenteritis. While management focuses largely on supportive care, the clinician needs to be aware that some patients, particularly infants, the elderly, and immunocompromised individuals, may need more aggressive care. New medications and treatment modalities continue to be developed for these conditions, with the latest pharmaceuticals offering promise in terms of their efficacy and side effect profiles.

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