TEXTBOOK
OF
ENVIRONMENTAL
EMERGENCIES

Editor-in-Chief
AC Anand
Professor and Head, Department of Internal Medicine
and Senior Adviser, Medicine and Gastroenterology

Editors
AS Narula
Professor, Department of Internal Medicine
and Senior Adviser, Medicine and Nephrology

R Kakkar
Professor, Department of Internal Medicine
and Senior Adviser, Medicine and Rheumatology

Executive Editor
Ravi Kalra
Associate Professor, Medicine and Cardiology

Associate Editors
S Johri, Associate Professor, Medicine & Neurology
VP Singh, Associate Professor, Medicine
Rajan Kapoor, Assistant Professor, Medicine
R Ananthakrishnan, Assistant Professor, Medicine

Published by
Department of Internal Medicine
Armed Forces Medical College
Pune
3. Clinical Aspects and Management of Diarrheal Diseases

BS Ramakrishna

CLINICAL ASSESSMENT OF A PATIENT WITH DIARRHEA

Diarrhea clinically manifests as an increase in stool weight, increase in stool frequency and/or abnormal looseness of the stool. The duration of illness, nature of diarrhea, and the presence of associated symptoms are all important in diagnosis. Acute diarrhea refers to an illness of less than two weeks duration, while chronic diarrhea refers to illness of greater than four weeks. Persistent diarrhea is used to denote an acute infective diarrheal illness lasting longer than two weeks. Dysentry refers to diarrhea with blood and mucus in the stool. Acute diarrhea is usually infective in origin, while the cause of chronic diarrhea can range from infective causes to malabsorption syndromes and inflammatory bowel disease. This chapter confines itself to the discussion of acute diarrheal illness, which is usually the result of environmental causes.

The presence of severe vomiting in a patient with diarrhea must lead one to suspect the diagnosis of either food poisoning or viral gastroenteritis [1]. Viral gastroenteritis such as that caused by norovirus (earlier called Norwalk agent) or calicivirus are characterised by vomiting as a very prominent or sometimes the sole symptom. The diagnosis is often suspected if the disease affects a cluster of patients. While viral gastroenteritis typically affects children, adults are also affected, especially the elderly living in hostels or institutions. Food poisoning also is characterised by vomiting as a prominent symptom. The diagnosis is to be suspected when the symptoms occur within a few hours of ingesting possibly contaminated food. Food poisoning caused by preformed toxin (e.g. Staphylococcus aureus or Bacillus cereus) leads to symptoms within six hours of eating the contaminated food, which consist of epigastric pain, nausea, vomiting and diarrhea. Food poisoning sometimes results from bacteria that multiply within the bowel (e.g. Salmonella or Campylobacter), and diarrhea associated with crampy abdominal pain and fever may occur 12-48 hours after ingestion of contaminated food. Certain organisms such as Yersinia enterocolitica may produce illness due to either preformed toxin or due to proliferation within the bowel, depending on whether or not the organism has had the opportunity to produce toxin in the contaminated food. Some species of fish and shellfish can contain a toxin called ciguatoxin

Textbook of Environmental Emergencies


Diarrheal Diseases

Classifying diarrheas*

As per incidence course: Acute diarrheas (<4 weeks) usually are due to infections, most of which are self-limited or are easily treated. Prolonged diarrheas in immunocompetent individuals can also be caused by infections such as Giardia lamblia or Yersinia spp. Chronic diarrheas may be due to non-infectious illnesses and may require detailed investigations.

As per volume of stools: If the disease causing the diarrheas is in the small bowel, the bowel movements are fewer, stools are watery, painless and large volume. Frequent, small, painful stools are usually due to recto-sigmoid disease. Diarrheas that produce dehydration (in the absence of vomiting or limited oral intake) typically have stool weights greater than 1000 g and therefore point to small bowel disease.

Watery versus Fatty versus Inflammatory: Watery diarrheas imply a defect primarily in water absorption due to increased electrolyte secretion or reduced electrolyte absorption (secretory diarrheas, such as cholera) or in absorption of a poorly absorbed substance (osmotic diarrheas, caused by lactulose). Fatty diarrheas imply defective absorption of fat in the small intestine. Inflammatory diarrheas imply the presence of inflammatory bowel disease or infection.

Epidemiologic Classification:

Travellers diarrheas is typically seen in tourists eating at unhygienic places, of which there is no shortage in our country. Thus, a backpacker in hills, a tourist eating at a roadside dhaba or a student having samosa in front of his school, may have similar etiology for diarrheas.


even when well cooked. The risk of poisoning by such toxins exists in tropical and subtropical islands in the Indian Ocean among other areas of the world. Symptoms of ciguatera poisoning include diarrhea, vomiting, neurologic symptoms such as dysestheasias, fever, hypothermia, and weakness. Certain fish contain high levels of histidine which, under conditions of improper refrigeration or preservation, is converted to histamine. This can cause diarrhea, nausea, vomiting, urticaria and flushing.

Profuse watery diarrheas associated with dehydration usually suggests an infection such as cholera or toxin-producing Escherichia coli, which elaborate enterotoxins that induce intestinal fluid secretion [2]. In these patients, diarrheas is typically painless, although mild, diffuse abdominal pain sometimes occurs secondary to the high volumes of secreted fluid. The stool in cholera is typically described as ‘rice water’ stools, but similar stool characteristics may be found in other toxin-induced diarrheas. Fever and abdominal pain are generally absent in cholera, but may be present in some patients with Vibrio cholera O139 infection, which is more invasive in the mucosa than traditional Vibrio cholera O1. Vomiting when it occurs in these patients is usually early in the illness and subsides quickly.

The majority of diarrheal illnesses are characterised by watery to loose stool without severe dehydration [3,4]. These illnesses may be caused by a variety of organisms including bacteria, viruses and parasites. In epidemics or outbreaks of diarrheas caused by these organisms, mortality is usually quite low in adults, although there may be significant morbidity. This is in contrast to cholera where mortality can be high in adults. In persons with this milder watery diarrheal syndrome, the presence of abdominal pain usually signifies inflammation of the bowel, especially if accompanied by fever. Mild abdominal pain may also result from carbohydrate maldigestion and gasousness. Non-typhoidal salmonellae may cause diarrheas that may be accompanied by low grade fever and crampy abdominal pain located around the umbilicus or in the lower abdomen. Pain is usually present before or after bowel movement. The stool is semi-formed or watery and may be greenish in colour. Similar syndromes are associated with diarrheas caused by toxigenic E. coli, Aeromonas, Plesiomonas and a variety of other bacterial enteropathogens. Yersinia enterocolitica infects the terminal ileum and cecum, and is associated with right lower quadrant abdominal pain and diarrheas. Acute yersiniosis is sometimes mistaken for acute appendicitis. Some patients will describe initial watery diarrheas, that later turns bloody in nature. This syndrome may be observed with pathogens such as Shigella, Campylobacter and Aeromonas which produce enterotoxins (causing initial small bowel fluid secretion) and also invade the intestinal mucosa, causing bloody diarrheas. The majority of viral diarrheas also presents as this form of undifferentiated watery diarrheas syndrome without severe dehydration. These viruses typically invade the epithelium of the small intestine, with minimal inflammation and no ulceration, and cause watery diarrheas. Rotavirus diarrheas is possibly the most common cause of diarrheas in children, but is not common in adults. In children, rotavirus infection can lead to severe dehydration and death if not promptly attended to. Parasitic organisms that infect the small intestine typically cause diarrheas that is semi-formed or watery, associated with borborygm, bloating and mild crampy abdominal pain. Parasites that do not invade the intestinal mucosa (Giardia intestinalis, Cryptosporidium) usually cause only mild abdominal discomfort. Helminthic parasites (Strongyloides stercoralis, Capillaria philippinensis) are more uncommon causes of diarrheas. These usually cause chronic diarrheas with features of malabsorption including steatorrhea, weight loss, hypoproteinaemia and other nutritional deficiencies.

The presence of blood and mucus in the stool suggests the presence of a colonic infection. The common causes of this syndrome of dysentery are Shigella, and Entamoeba histolytica. Less commonly, dysentery may be due to infection with enteroinvasive Escherichia coli, Campylobacter, Salmonella or Balantidium coli. Affected patients typically have frequent small volume bowel movements, associated with urgency and tenesmus, reflecting inflammation of
Diarrhea in Hospitalized Patients

Diarrhea in a hospitalized and severely ill patient is likely to be drug induced, especially antibiotic-associated diarrhea, diarrhea associated with tube feeding, intestinal ischemia, and fecal impaction or post operative diarrhea. Antibiotic associated diarrhea is due to toxins produced by C. difficile superinfection. Other drugs that commonly contribute to diarrhea are theophyllin, magnesium antacids, H2 receptor antagonists, Sorbitol containing effuex, and Cancer chemotherapy. Diarrhea also may be a complication of enteral nutrition but is often due to coexisting problems. It may be related to hypertonic feeding formulas or faster rate of infusion. Some patients develop intestinal ischemia during hospitalization, especially if they have hypotension or shock. They are at risk for developing bloody diarrhea due to ischemic colitis or more profound diarrhea if small bowel ischemia develops. Fecal impaction is a common phenomenon in the elderly, patients at prolonged bowel rest, and those receiving constipating drugs. Some of these may present with either spurious or "overflow" diarrhea. Rarely, osmotic diarrhea due to impaired colonic fermentation of carbohydrates may be seen in very sick patients.

Diarrhea in post-operative period

Postcholecystectomy diarrhea occurs in up to 20% of patients after cholecystectomy, and may begin long time after surgery. It is bile acid binders given at bedtime and perhaps at other times during day as well. Opiate antidiarrheals can also be helpful for refractory cases. Diarrhea after Gastric Surgery may be due to dumping syndrome, a condition characterized by flushing, hypotension, diarrhea, and hypoglycemia postprandially. Gastric surgery may also predispose patients to bacterial overgrowth in the small intestine, abnormally rapid intestinal transit, bile acid malabsorption, and pancreatic exocrine insufficiency due to poor stimulation or inadequate mixing. Diarrhea after Bowel Resection is related to loss of absorptive surface and will abate as process of intestinal adaptation improves intestinal electrolyte absorption with time. Resection of the terminal ileum results in a permanent reduction in conjugated bile acid absorption that may produce bile acid-mediated fluid and electrolyte secretion by the colon. Causes of ileostomy diarrhea include stomal stenosis, partial bowel obstruction, bacterial overgrowth, recurrent disease proximal to the stoma, medication-associated diarrhea, and intrauterine infection. Diarrhea in patients with ileostomy anastomosis after colectomy for ulcerative colitis may be due to inflammation of the pouch (pouchitis) and often responds to antibiotics, metronidazole, and/or with anti-inflammatory drugs, 5ASA compounds.


the colon and rectum. Fever and abdominal pain are common. Abdominal pain is characteristically located in one or both iliac fossa and may precede or follow bowel movement.

A history of medication prior to the diarrheal episode is helpful in sporadic diarrhea. Commonly used medications, such as antibiotics, may lead to diarrhea.

Antibiotics may also lead to Clostridium difficile overgrowth and antibiotic-associated (pseudo-membranous) colitis. Details of the social history are important including a history of recent travel, occupation, and living situation. Diarrhea related to recent travel is common, and usually is caused by toxigenic Escherichia coli. Protozoan parasites such as Giardia intestinalis, Cryptosporidium parvum and Cyclospora cayatensensi may also cause diarrhea in travellers, especially in persons who have previously not been exposed to these organisms. Eating contaminated ground beef is responsible for haemorrhagic Escherichia coli colitis, while Campylobacter infection may result from eating undercooked chicken or turkey. Both these organisms may cause point source outbreaks of diarrhea secondary to food contamination. Yersinia infection with diarrhea may result from ingesting contaminated milk or milk products.

Knowledge of the patient's sexual practices is important in certain circumstances such as in proctitis caused by E. histolytica, Neisseria gonorrhoea, or Herpes simplex. Opportunistic infections in the acquired immunodeficiency syndrome may also present as acute diarrhea. Infection with the human immunodeficiency virus is often a predisposing factor in patients with chronic diarrhea, especially if the patient has other symptoms of advanced disease, e.g. opportunistic infections such as oesophageal candidiasis or pulmonary tuberculosis.

One major objective of the history and physical examination is to exclude conditions that may actually result from a serious intra-abdominal crisis but presenting as acute diarrhea [1]. These conditions include appendicitis and diverticulitis, which may occasionally present with diarrhea accompanied by abdominal pain and fever. Other abdominal conditions that may present with acute bloody diarrhea and abdominal pain include ischemic colitis, inflammatory bowel disease, and superior mesenteric arterial or venous thrombosis. The history is usually informative and findings of abdominal guarding and rebound tenderness on physical examination must alert the clinician to the correct diagnosis.

The physical examination is important to determine the severity of dehydration and toxicity, features that indicate a possible need for hospitalization. Frank hypotension, signifying loss of greater than 20% of circulating blood volume, is common in cholera. Evaluation for concomitant medical illness should be made particularly in elderly patients who may develop a frank myocardial infarct or stroke secondary to hypoperfusion, and pulmonary aspiration secondary to vomiting. Abdominal distension with infrequent or absent bowel sounds indicates ileus secondary to hypokalemia, ischaemia or peritonitis. Fulminant infection or inflammation of the colon resulting in toxic megacolon also results in abdominal distension and a silent abdomen. Localized tenderness and/or rebound tenderness in the right lower quadrant may signify ileal or caecal inflammation as in Yersinia infection, tuberculosi or Crohn's disease. Localized tenderness in the left lower quadrant of the abdomen with or without signs of peritonitis could signify inflammation of the sigmoid colon (E. histolytica, Shigella, or inflammatory bowel disease) or
Diarrheal Diseases

**Simplified approach for an HIV positive patient with diarrhoea**

<table>
<thead>
<tr>
<th>Chronic diarrhoea &gt;4 weeks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical evaluation</td>
</tr>
<tr>
<td>Lab evaluation if possible</td>
</tr>
<tr>
<td>Trimeprprim-sulfamethoxazole (480mg) TMP-SMX, 2 tablets twice a day for 5 days. If there is no response</td>
</tr>
<tr>
<td>Improvement</td>
</tr>
<tr>
<td>Ask patient to return in case of relapse</td>
</tr>
<tr>
<td>Relapse within 4 weeks treat with same drug for 3 weeks</td>
</tr>
<tr>
<td>No improvement treat with norfloxacin &amp; metronidazole. Try constipating agent if no response, needs further evaluation stool microscopy for 3 days</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>No response use metronidazole</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maintain hydration</td>
</tr>
<tr>
<td>Supplemental Feeds</td>
</tr>
<tr>
<td>K Supplementation</td>
</tr>
<tr>
<td>Treat with TMP-SMX</td>
</tr>
</tbody>
</table>

Diverticulitis.

Rectal examination is mandatory in the initial evaluation of patients who are elderly or bedridden. This allows the physician to ascertain the character of stool and accurately assess the type of diarrhea whether watery or bloody. This is particularly helpful in situations in which patients, especially the elderly or those with poor eyesight, might not be able to give an accurate history. It also allows one to exclude the possibility of faecal impaction and spurious diarrhea.

Acute watery diarrhea leads to dehydration secondary to the loss of fluid and electrolytes. Even mild diarrhea may lead to some degree of dehydration, which is difficult to assess quantitatively. Adults normally have better compensatory mechanisms than children through the larger body fluid reserve, the better kidney compensatory mechanisms, and better response to correct thirst. Together with poorer tissue elasticity and slower shift of extra cellular fluid, the clinical signs of dehydration in adults are less obvious than in children. Severity of dehydration may be assessed [5] using a combination of symptoms and signs as shown in Table 9.2.

**COMPLICATIONS OF DIARRHEA**

Dehydration and electrolyte abnormalities (hypokalaemia due to loss of potassium and metabolic acidosis due to loss of bicarbonate) may occur in severe diarrhea. The electrolyte abnormalities and dehydration are accentuated if the patient also has significant vomiting, and severe hypokalaemia and acidosis are often the cause of mortality in acute watery diarrhoeal illnesses. Severe dehydration may result in acute tubular necrosis and renal failure. Haemolytic uremic syndrome with renal failure can be a complication of specific infections including shigellosis and enteroinvasive E. coli infection [6]. Arthropathy, Reiter’s syndrome, thyroiditis, or pericarditis may occasionally complicate *Yersinia, Shigella*, or *Campylobacter* infections. Acute diarrhea continues to be a major cause of mortality in children [7], but may also cause adult deaths. Such mortality may occur particularly in the elderly or immunocompromised, due to dehydration and electrolyte imbalance, cerebral or cardiac ischaemia, renal failure, and sepsis.

**DIAGNOSIS**

Most episodes of acute diarrhea are self-limited, and do not call for investigation. Medical evaluation is advisable for patients with moderate or severe illness, i.e. those with more than 6 loose stools per day, with dehydration, fever greater than 38.5°C, those with dysentery, or diarrhea persisting longer than 48 hours. Patients over the age of fifty presenting with diarrhea should be specifically questioned about severe abdominal pain and evaluated to exclude ischemic bowel disease. All patients should be examined to exclude signs of peritonitis, which would necessitate more aggressive diagnosis and management. Patients without these risk factors may be managed without further investigation, with advice on symptomatic therapy and oral hydration. Patients

---

**Table 9.2: A simple guide to assessing severity of dehydration**

<table>
<thead>
<tr>
<th>General state</th>
<th>Alert, active, up and about</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ability to perform daily activities</td>
<td>Able to perform daily activities without difficulty</td>
</tr>
<tr>
<td>Thirst</td>
<td>Not increased</td>
</tr>
<tr>
<td>Pulse</td>
<td>Normal</td>
</tr>
<tr>
<td>Blood pressure</td>
<td>Normal</td>
</tr>
<tr>
<td>Hypotension</td>
<td>None</td>
</tr>
<tr>
<td>Dry mucosa (mouth, tongue)</td>
<td>No</td>
</tr>
<tr>
<td>Skin turgor</td>
<td>Good</td>
</tr>
<tr>
<td>Sunken eye balls</td>
<td>No</td>
</tr>
<tr>
<td>Weak, lethargic. Able to sit and walk</td>
<td>Able to perform daily activities with some difficulty, e.g. Stays away from work, needs support</td>
</tr>
<tr>
<td>Increased thirst</td>
<td>Tachycardia</td>
</tr>
<tr>
<td>Normal or decrease 10-20 mmHg systolic</td>
<td>Postural</td>
</tr>
<tr>
<td>Slight</td>
<td>Fair</td>
</tr>
<tr>
<td>Minimal</td>
<td>Dull, inactive. Unable to sit or walk.</td>
</tr>
<tr>
<td>Unable to perform daily activities. Stays in bed or needs hospitalization.</td>
<td></td>
</tr>
<tr>
<td>Feels very thirsty.</td>
<td></td>
</tr>
<tr>
<td>Tachycardia</td>
<td></td>
</tr>
<tr>
<td>Decrease &gt; 20 mmHg systolic</td>
<td></td>
</tr>
<tr>
<td>Supine</td>
<td></td>
</tr>
<tr>
<td>Severe</td>
<td></td>
</tr>
<tr>
<td>Poor</td>
<td></td>
</tr>
<tr>
<td>Sunken</td>
<td></td>
</tr>
</tbody>
</table>
Parasites as a cause of diarrhea

A study from Vellore has examined 4103 samples from patients presenting with diarrhea. Enteric parasites were identified in 5.8% of samples. Giardia lamblia was the commonest identified pathogen, seen in 2.61%, with Isospora in 0.51%, Cryptosporidium in 0.37% and Cyclospora in 0.15%. The helminths identified were Strongyloides in 0.95%, hookworm in 0.80% and Ascaris 0.39%. Multiple parasites were not noted. They also examined 258 samples from HIV positive patients presenting with a history of diarrhea. It was noted that parasites were involved in 57.3% of 258 samples from HIV infected individuals, with multiple pathogenids identified in 6.6%. Protozoan parasites were common, with Isospora belli (19.7%), Cryptosporidium (15.5%), Giardia lamblia (5.0%) and Cyclospora cayetanensis (3.8%). Microsporidia were seen in 4.6%. The helminths identified were Strongyloides stercoralis larvae (8.3%), Ascaris lumbricoides ova (2.3%) and hookworm ova (4.3%).

This five year study showed that parasitic diarrhea is 10 times more common in HIV positive patients than in HIV negative patients (57.3% versus 5.8%, p<0.00). Protozoal pathogens that cause opportunistic infections of the gut are Cryptosporidium parvum, Isospora belli and microsporidia and these were seen in 40% of all HIV positive patients with diarrhea. In recent years, it has been shown that HIV infection and parasitic infections interact and have a mutual deleterious effect. Parasitic infection may facilitate the progression from asymptomatic HIV infection to AIDS by a chronic immune activation, particularly with T-helper 2 type responses. Studies using surrogate markers of AIDS, like CD4 counts and HIV viral load in addition to these tests will elucidate the dynamics of infective diarrhea in HIV patients.


with high fever, bloody diarrhea, systemic toxicity, dehydration, or rebound tenderness should be further investigated. Freshly collected stool should be examined for white and red blood cells, parasites (ova, cysts and trophozoites), and occult blood. The presence of large numbers of white blood cells suggests infection with one of the invasive organisms, or acute inflammatory bowel disease. When parasites are suspected, examination of three fresh stool samples provides the optimal yield. When choleria is suspected clinically, the stool should be examined for motile organisms by dark field microscopy. Stool culture is not routinely indicated, but should be reserved for patients with high fever or bloody diarrhea, those with faecal leukocytes, prolonged diarrheal illness, or those with special epidemiologic circumstances [8]. Diagnosis of less common pathogens may require special techniques including biopsy of the small or large intestinal mucosa and electron microscopy. Sigmoidoscopy and colonoscopy do not help in most instances, but may be carried out if bloody diarrhea does not improve within ten days. Sigmoidoscopy may help to identify antibiotic-associated colitis where characteristic pseudomembranes are noted, and amoebic colitis where characteristic discrete irregular ulcers with normal intervening mucosa are seen. Identification of amoebic trophozoites on biopsy is diagnostic in the latter condition.

TREATMENT

Most patients with acute diarrhea can be managed symptomatically with rest and fluid replacement [9-12]. Since death usually results from dehydration and electrolyte imbalance, serum electrolytes and blood urea nitrogen should be closely monitored in those with severe diarrhea, particularly when associated with vomiting. Intravenous fluid therapy is definitely indicated in all individuals with severe dehydration, and probably indicated in infants and elderly patients with milder degrees of dehydration. The intravenous fluid of choice is full strength Ringer’s lactate solution, which replaces both potassium and bicarbonate losses and can therefore be given in large quantities. 50 ml/kg of the IV fluid should be given in the first hour, and another 50 ml/kg in the next three hours. At the end of four hours, most patients should be fully rehydrated in the absence of continuing severe diarrhea.

Oral rehydration therapy with sugar and electrolyte solutions is sufficient for the rehydration of patients with mild to moderate dehydration. Oral rehydration solution (ORS) is also used to prevent dehydration in patients who have clinically milder forms of diarrhea. Oral rehydration utilizes the fact that glucose and amino acids can efficiently stimulate sodium absorption from the intestine through absorptive pathways that are not inhibited by enterotoxins. ORS formulations contain a carbohydrate, sodium and potassium and either bicarbonate or citrate as a base. The goal of oral hydration therapy is to correct dehydration, and to maintain hydration once dehydration is corrected. In dehydrated patients, the first four hours of therapy is generally sufficient to restore them to a state of normal hydration, and this called the rehydration phase. One to four litres of ORS is given in the first four hours for rehydration. At the end of this period, if the patient is fully rehydrated, the phase of maintenance of hydration begins and the ongoing stool losses should be replaced with additional 1-4 litres per day. The World Health Organization currently recommends an ORS of the following composition for the rehydration of all patients with diarrhea: Na 75 mmol/l, K 20 mmol/l, Cl 65 mmol/l, citrate 10 mmol/l (or bicarbonate 30 mmol/l), and glucose 75 mmol/l [13]. While this solution is adequate in most patients with diarrhea, there is a potential for the development of symptomatic hyponatraemia in patients with very severe watery diarrhea as in severe choleria due to a relatively low sodium content of the ORS. A variety of ORSs are available commercially, which are mostly broadly satisfactory for the purpose of hydration. Oral hydration therapy does not reduce or shorten diarrhea, except when modified solutions incorporating rice or other cereals are used. The addition of starch to ORS, currently under investigation [14], promises to reduce and shorten diarrhea.
Diarrheal Diseases

Table 9.3: Recommended antibiotics against specific enteric pathogens

<table>
<thead>
<tr>
<th>Organism</th>
<th>Single-agent antibiotic</th>
<th>Alternative antibiotic</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Vibrio cholerae</em></td>
<td>Doxycycline, 300 mg single dose</td>
<td>Fluoroquinolone* X 3 d</td>
</tr>
<tr>
<td><em>Vibrio parahaemolyticus</em></td>
<td>Antibiotics are usually not required, except in certain situations.a</td>
<td>Fluoroquinolone* X 3 d</td>
</tr>
<tr>
<td>Non-O1 Vibrios</td>
<td>Doxycycline, 300 mg single dose TMP-SMZ,</td>
<td>Gentamicin*, 80 mg X 5-7 d</td>
</tr>
<tr>
<td>Shigella species</td>
<td>160-800 mg b.i.d. X 3 d (if susceptible)</td>
<td>Cefotaxime*, 1 g q.i.d. X 5-7 d</td>
</tr>
<tr>
<td>Non-typhoidal species of <em>Salmonella</em></td>
<td>Antibiotics are usually not required, except in certain situations.b</td>
<td>Fluoroquinolone* X 3 d Naïdacid</td>
</tr>
<tr>
<td>Aeronomas species</td>
<td>TMP-SMZ, 160-800 mg b.i.d. X 5-7 d (if susceptible)</td>
<td>Ceftriaxone*, 1 g b.i.d. X 5-7 d Azithromycin 250 mg single dose</td>
</tr>
<tr>
<td>Plesiomonas species</td>
<td>Antibiotics are usually not required, except in certain situations.c</td>
<td>Fluoroquinolone* X 5-7 d</td>
</tr>
<tr>
<td>Enterotoxigenic <em>Escherichia coli</em></td>
<td>Antibiotics are usually not required, but may be required in certain situations. a</td>
<td>Ceftriaxone*, 1 gm b.i.d. X 7-14 d</td>
</tr>
<tr>
<td>Enteroopathogenic <em>Escherichia coli</em></td>
<td>Antibiotics have no established therapeutic value and are usually not required, except in certain situations. a</td>
<td>Fluoroquinolone* X 3 d</td>
</tr>
<tr>
<td>Enteroinvasive <em>Escherichia coli</em></td>
<td>Antibiotics are usually not required, except in certain situations.a</td>
<td>Fluoroquinolone* X 3 d</td>
</tr>
<tr>
<td>Enteroaggregative <em>Escherichia coli</em></td>
<td>Unknown therapeutic value</td>
<td>Fluoroquinolone* X 3 d</td>
</tr>
<tr>
<td>Enterohaemorrhagic <em>Escherichia coli</em> (STEC)</td>
<td>Role of antibiotics is unclear and administration should be avoided as they may be harmful.</td>
<td>Fluoroquinolone* X 5 d</td>
</tr>
<tr>
<td>Campylobacter species</td>
<td>Antibiotics are usually not required, except in certain situations.c</td>
<td>Combination of doxycycline and aminoglycoside, or TMP-SMZ, or fluoroquinolone</td>
</tr>
<tr>
<td><em>Yersinia</em> species</td>
<td>Antibiotics are usually not required, except in certain situations.c</td>
<td>Vancomycin 125-250 mg q.i.d. X10 14 d</td>
</tr>
<tr>
<td><em>Toxigenic Clostridium difficile</em></td>
<td>Offending antibiotics should be withdrawn if possible. Metronidazole, 250-500 mg q.i.d. X 10-14 d</td>
<td></td>
</tr>
</tbody>
</table>

a: antibiotics may be required in severely ill patients or in septicemic prone conditions e.g. cirrhosis, uncontrolled diabetes mellitus, or immunocompromised host. b: antibiotics may be required in the following conditions: severely ill patients; age < 6 months or > 60 years old; immunocompromised hosts; patients with internal prostheses; valvular heart disease; severe atherosclerosis; malignancy; uremia; or uncontrolled diabetes mellitus. c: Antibiotics may be required in severely ill patients, immunocompromised, those with associated bacteremia or traveller's diarrhea.

Fluoroquinolone e.g. 300 mg ofloxacin, 400 mg norfloxacin, or 500 mg ciprofloxacin b.i.d. *antibiotics for suspected septicemic cases

Rehydration, either through oral or intravenous route, is expected to be complete in four to six hours depending on the pace of administration of fluids. Typically at the end of this period, the patient needs to be reassessed with regard to state of hydration as well as to urine output. If the patient is adequately hydrated at the end of this time, and has passed urine, the treatment enters the maintenance phase in which the patient is administered maintenance fluids to prevent further dehydration. The patient is urged to take 100-200 ml of fluid after each loose stool. It is important to commence normal feeding at this time. In children and adults, the normal diet can be introduced at the end of four hours of rehydration therapy. In the case of infants, breast feeds can be resumed at this time.

Evaluation of fluid and electrolyte deficit is crucial in calculation of the amount of fluids to be replaced. Hence stool volume loss should be closely monitored and, if possible, weighed or accurately measured. In those patients who are not sick enough and still can go to toilets on their own, it may be difficult to estimate the amount of deficit and ongoing loss correctly. The usage of “cholera cots” can be very helpful to monitor the amount of ongoing loss. If cholera cots are not available, it may be safer to replace twice the amount of estimated loss and closely monitor the status of hydration of the patient. Recently the use of resistant starch has been shown to be of benefit
in reducing stool volume loss and shortening the clinical course in adult patients with cholera. The use of zinc has also been advocated in ORS in order to hasten mucosal repair and regeneration and hasten recovery. In children, the administration of zinc is advocated for a period of two weeks but not beyond this.

Anti-diarrheal drugs have a very limited role in the management of acute diarrhea. Attapulgite, which binds water, is a safe therapy, exerting its effect only in the lumen. Bismuth subsalicylate (which has an antisecretory salicylate moiety in addition to antimicrobial activity) ameliorates both diarrhea and nausea and vomiting, and is safe in the treatment of traveller's diarrhea. Medications like codeine or tincture of paregoric are generally reserved for diarrhea that does not respond to other medications, and are used more commonly in severe chronic diarrhea. The most widely used medications to control the symptoms of diarrhea are antimitoty agents, such as diphenoxylate HCl with atropine sulphate (Lomotil) or loperamide. Most patients with mild non-dehydrating or non-bloody diarrhea can safely take these to relieve symptoms. Antimitoty agents should be avoided when an invasive organism is suspected, since colonization may be prolonged, with worsening of diarrhea, increased bowel wall penetration and toxic megacolon. Loperamide may be useful as the sole therapy for mild travellers' diarrhea. It is generally withheld in severe watery diarrhea and in children because of its lack of efficacy and its propensity to cause abdominal distension. Raccadotril (acetrophan) is an enkephalase inhibitor that acts through enkephalergic receptors in the intestine and colon to reduce intestinal fluid secretion and promote fluid absorption. Since it does not have any effect on intestinal motility it has been held up as having an advantage over loperamide, which can cause constipation and abdominal distension. In clinical trials, it has been found to reduce duration of diarrhea in children with acute watery diarrhea, and in adults with non-dehydrating acute diarrhea, but is not useful in adult cholera.

Dietary recommendations are often requested by patients with acute diarrhea. In general, the patient should resume a normal diet after the initial four hour period of rehydration. In breast-fed infants, breast feeding should be resumed at this time. Certain dietary modifications may be helpful in patients with mild acute diarrhea. Except in infants, milk and dairy products are preferably restricted or diluted and administered for 24-48 hours. However, feeding with cereals and starches should continue. Yoghurt and buttermilk may be administered without restriction.

Antibiotic therapy is not necessary, and should be avoided, in the majority of patients with diarrhea, especially those with mild or resolving disease. Antibiotics are indicated in patients with cholera, as well as those with amoebic dysentery or shigellosis [15,16,17]. They are also indicated in patients with traveller's diarrhea, pseudomembranous colitis, and parasitic diseases, when diarrhea is disabling or not self-limited. Salmonella enteritis in immunocompetent subjects (most young adults and adults) is not to be treated with antibiotics since the infection is usually self-limited. It has been shown that administration of antibiotics to these individuals results in prolonged carriage and excretion of the Salmonella organism. However, certain categories of patients with Salmonella enteritis require antibiotic therapy. These include those at the extremes of age, those with abnormal heart valves, those with vascular or orthopaedic prostheses, and those with haemolytic anaemia. The latter three classes of patients are prone to develop colonization of the prostheses of abnormal heart valves with the organism and hence require antibiotic treatment. Antibiotics demonstrably reduce stool volume loss and shorten clinical course in patients with cholera and dehydration. If there is recent epidemiological data available, empiric antibiotics should be given according to the sensitivity of Vibrio cholerae in the region. Doxycycline 300 mg as a single oral dose, has largely supplanted the use of tetracycline in cholera. In regions where resistance to tetracycline is greater than 20 percent, ciprofloxacin 500 mg twice daily for 3 days is recommended. For pregnant patients, furazolidone 1000 mg/day for 3 days has been suggested. The various antibiotics that can be used in therapy of acute diarrhea are shown in Table 3. Antibiotic therapy with one of the fluoroquinolones may sometimes be given empirically in the treatment of diarrhea, in those with high fever and faecal leukocytes, in moderate to severe traveller's diarrhea and in dysentery.

Probiotics are nonpathogenic organisms, such as Lactobacillus rhamnosus and Saccharomyces boulardii, which multiply in the patient's intestine and attenuate the effect of enteric pathogens. Such an effect of probiotics occurs through a variety of mechanisms including production of metabolites which increase acidity of stool and prohibit the growth of enteropathogens, preventing the invasiveness of pathogenic organisms through effects on epithelial and subepithelial immune cells, and production of molecules such as short chain fatty acids that are beneficial for intestinal mucosal recovery and increase the rate of fluid and electrolyte absorption. In both children and adults, there is some evidence that the use of probiotics shortens acute diarrhoeal illness, especially when it is due to rotavirus infection or antibiotic-associated colitis.

An algorithm for the management of acute diarrhea is shown in Fig. 9.1. This is applicable to institutions with some facilities available for testing for diarrhea. In the emergency room or in family practice, other modifications of this algorithm may be employed.

Special Situations

Acute diarrhoea in the elderly: Acute diarrhoea that occurs in patients aged > 60 years old is associated with higher mortality, due to coronary and cerebrovascular events. Deaths related to diarrheal illnesses are recognized among older adults living in the community as well as among
Diarrheal Diseases

Fig. 9.1: Algorithm for managing patients with acute diarrhoea.

Those confined to nursing homes. Outbreaks have most often been associated with excess deaths from diarrhoea among nursing-home patients. The approach to an elderly patient with diarrhoea is to ensure proper hydration using available oral rehydration solutions, proceed with diagnostic tests likely to yield a positive result, avoid the use of harmful antiperistaltic drugs, and provide adequate follow-up of the nutritional state. Antibiotics should be administered in acute diarrhoea due to invasive bacteria, especially Salmonella.

**Traveller's Diarrhea**

Diarrhoea is common in travellers, especially if the traveller consumes food from street vendors rather than restaurant or hotel service. Symptoms and severity depend on the prevalence of common pathogens endemic in the areas visited. The common causes of acute traveller's diarrhoea vary from one geographical area to another. The most frequently identified pathogen causing traveller's diarrhoea is toxigenic *Escherichia coli*, although in some parts of the world (notably North Africa and Southeast Asia),
Campylobacter infections appear to predominate. Other common causative organisms include Salmonella, Shigella, rotavirus, and the Norwalk agent. Except for giardiasis, parasitic infections are uncommon causes of traveller's diarrhea. The disease is usually short-lived, self-limited, however many of them are amenable to antibiotics, which are chosen depending on what is most likely to have caused the diarrhea. Antidiarrheal drugs can be given alone in mild cases, or in conjunction with antibiotics in others. A growing problem for travellers is the development of antibiotic resistance in many bacterial pathogens; examples include strains of Campylobacter resistant to quinolones and strains of E. coli, Shigella, and Salmonella resistant to trimethoprim-sulfamethoxazole.

**Antibiotic Associated Diarrhea**

Diarrhea that occurs as a result of administered antibiotics, which alter the normal intestinal flora and increase the proliferation of Clostridium difficile. This organism acts through the production of cytotoxins A and B that cause enterocolitis and pseudomembranes. A history of antibiotic usage prior to the development of diarrhea suggests the possibility of this diagnosis. Onset of symptoms occurs either during antimicrobial administration or within 4 weeks after treatment. The clinical spectrum of antibiotics-associated enterocolitis is variable, ranging from mild loose stools to severe colitis causing bloody diarrhea. Some patients may manifest systemic toxicity characterised by fever and abdominal pain. Examination of stool may reveal large numbers of red blood cells and some leukocytes. Stool culture for *C. difficile* is not practical in most microbiology laboratories, and the diagnosis is often made by performing an assay (commercially available) for toxin in the stool. Sigmoidoscopy or colonoscopy may reveal normal, minimally erythematous colonic mucosa with some oedema, or granular, friable, haemorrhagic mucosa with typical pseudomembrane formation. The course is highly variable. In those with clinically mild disease, withdrawal of offending antibiotics and use of oral or intravenous rehydration as necessary usually leads to prompt resolution of symptoms. In patients who have more severe or protracted illness, metronidazole 400 mg 4 times daily may be administered while waiting for the result of cytotoxin assay, and should be continued for 7-14 days. If cytotoxin study is positive and the patient does not get better after a week of metronidazole, then vancomycin 125-250 mg/day should be substituted. Antidiarrheal drugs have no advantage in treating antibiotic-associated enterocolitis, except cholestyramine and probiotics, which can be helpful in chronic or relapsing disease.

**Haemorrhagic Colitis due to Enterohaemorrhagic Escherichia coli (EHEC, Shiga toxin producing E. coli, STEC)**

Haemorrhagic colitis, caused by EHEC should always be a differential diagnosis in patients who present with acute bloody diarrhea. Patients who present with non-bloody diarrhea that progresses to bloody diarrhea should also raise the possibility of EHEC infection. Other prominent complaints include striking abdominal pain and tenderness often in the absence of fever. In an outbreak situation, some patients with EHEC infection may be asymptomatic and are only recognised during epidemiologic surveillance in association with symptomatic cases. In general, the mortality rate is 1 to 2 percent, although it may be substantially higher in the elderly. The most worrisome complication of EHEC infection is the haemolytic-uremic syndrome (HUS), which most frequently involves children under the age of 5 to 10 years. HUS is characterized by the triad of acute renal failure, microangiopathic haemolytic anaemia, and thrombocytopenia. Patients who also have fever and neurologic symptoms are considered to have the related disorder thrombotic thrombocytopenic purpura (TTP), which has also been associated with E. coli O157:H7 infection. HUS usually begins 5 to 10 days after the onset of diarrhea. The incidence of subclinical renal dysfunction is substantially higher, particularly in patients with prolonged anuria during the initial presentation. Stool culture using sorbitol-MacConkey agar should be done in all suspected EHEC diarrhea. A number of newer diagnostic approaches for EHEC infection focus on direct detection of Shiga toxins in stool, or the use of DNA probes for detecting the toxin genes in fecal isolates. One such assay, the Premier EHEC assay, utilizes an enzyme-linked immunosorbent assay to detect both Shiga toxin 1 and Shiga toxin 2 in stool. The only current treatment of EHEC infection is supportive, with monitoring for the development of microangiopathic complications such as HUS. The impact of antibiotic therapy on the duration of diarrhea or on the subsequent occurrence of systemic complications is controversial. The use of antibiotics may actually increase the risk of HUS, perhaps by increasing production or release of toxin. New approaches to therapy of EHEC infection are currently being evaluated, but are not yet proven effective or available routinely. These include toxin-binding residues given orally and hyperimmune antitoxin antisera.

**Nosocomial Diarrhea**

Acute diarrhea that occurs in hospitalized patients is an important problem in hospitals and in critical care units in particular. Infectious causes of nosocomial diarrhea include contaminated feeds, outbreaks in hospital, and infection with *Clostridium difficile*. All cases of nosocomial diarrhea should be properly investigated with stool examination, culture and *C. difficile* cytotoxin assay. Proper hydration together with dietary adjustment should immediately be employed. If possible, discontinuation of offending drugs or antibiotics should be considered. Empiric metronidazole can be started in patients with possibility of antibiotic associated diarrhea. Systemic antibiotics may be necessary in immunocompromised patients. *C. difficile* is a resident of the human colon and...
Diarrheal Diseases
does not cause disease if its toxins are not elaborated. Chemotherapeutic agents and more commonly, antibiotics, induce the elaboration of toxin A and B from *C. difficile* in the distal gastrointestinal tract. Infection control measures are necessary to prevent the spread of this spore forming organism within the institution since it is capable of surviving in the hospital environment for prolonged periods.

**Outbreak Diarrhea**

Acute diarrhea that occurs in two or more persons from the same exposure, assumed to be caused by the same pathogens, is considered as an outbreak. In an outbreak situation, the disease spectrum is highly variable, ranging from very mild to very severe form of disease. All acute diarrhea in the outbreak area should be reported to the Health Authority and proper epidemiological investigation should be employed. Rapid testing or kits for identifying the “outbreak pathogen” should be used in the field and further confirmation can be done later in a reference centre. Antibiotics that are known to be effective in eradicating the pathogen should be empirically administered to all acute diarrhea cases in the outbreak area, in order to contain spread of the infection. Epidemiological surveillance is necessary until the outbreak completely subsides.

**Acute Diarrhea in Specific Medical Illnesses**

Septicaemia may complicate illness in acute diarrhea in persons suffering from certain medical illnesses, e.g. cirrhosis especially alcohol cirrhosis, uncontrolled diabetes mellitus, patients with heart valves, prosthesis, severe atherosclerosis, malignancy, and uraemia. There are several reports of non-O1 Vibrio septicaemia in patients with cirrhosis. Uncontrolled diabetics with diarrhea are prone to gram negative septicaemia. Patients with heart valve prosthesis, severe atherosclerosis and orthopaedic prostheses are prone to lodging of salmonella infection at the diseased heart valve and prosthesis, and require early treatment with antibiotics in the event that they acquire salmonella gastroenteritis.

**Acute Diarrhea in Immuno compromised Patients**

Acute diarrhea may occur in patients infected by the human immunodeficiency virus. This may occur while they are still immunocompetent (CD4 count > 500 cell/cu mm), or while they are immuno compromised. In the latter case, they will require investigation including routine stool culture and examination, stool staining for AFB, modified AFB, modified trichrome staining and *C. difficile* cytotoxin assay. Empiric treatment can be considered if the nature of enteropathogens infecting these patients locally is known. Specific treatment with antibiotics, if being administered, should be given in a more prolonged course to ensure the complete eradication of the pathogen and prevent early relapse. Nutritional management is also required in this group of patient. Acute diarrhea may also occur in other states that compromise immunity, such as patients treated with immunosuppressive agents or chemotherapy, those with autoimmune diseases, malignancy (especially haematologic). In these patients, acute diarrhea may lead to septicaemia, hence early antibiotic therapy is advisable.

**SUMMARY**

Acute diarrhea is a common condition and may occur more commonly in certain environments than in others. In practice, it is important to first exclude other medical and surgical emergencies that may have a rare presentation as acute diarrhea. In most instances, stool examination and culture results are often not available and proper hydration either through oral or intravenous route, combined where necessary with antibiotics, constitutes the therapy of the vast majority of cases. A practical approach for the management of patients with acute diarrhea is presented in an algorithm. Patients with symptomatically severe vomiting are likely to have food poisoning or viral gastroenteritis, while those with diarrhea are classified as having watery or bloody diarrhea. Patients with bloody diarrhea are managed by hydration using ORS, with antibiotics in the case of severe cholera. Patients with bloody diarrhea are evaluated by stool examination and managed with appropriate antibiotics. Further investigation by repeating stool examination and culture together with sigmoidoscopy or colonoscopy are essential if they do not get better. Certain categories of individuals with acute diarrhea need to be considered separately and managed somewhat differently.

**References**

4. Prevention of Diarrheal Diseases

BS Ramakrishna

Epidemic diarrhea occurs under conditions related to crowding and poor environmental sanitation, as for example during large religious gatherings as may occur during the Kumbh mela, or during conditions of civil war and strife as in the African continent. Epidemic diarrhea is also common when there are floods as happens annually in the Indo-Gangetic delta. There are several measures that can be taken by the civil or other administration to reduce or prevent diarrhea under such conditions. Prevention of sporadic diarrheal disease is more difficult, and requires an understanding in members of the community of the means by which these diseases spread. Most diarrheal illnesses are caused by waterborne infection, through drinking water that has been contaminated at its source (faecal contamination of an incompletely sealed well) or during storage, as also ice made from contaminated water. Seafood taken from contaminated water, and eaten raw or partially cooked, may also be responsible. Fruit and vegetables grown at or near ground level in soil contaminated with night-soil, or washed in contaminated water may be responsible. Flies and other insects may carry infection to open or uncovered food and fruit but this is not a major source of infection in epidemics. Food may also be contaminated after preparation by an infected carrier. Faecal contamination is usually from infected humans although faeces of infected cattle and other animals may also be responsible for sporadic or point source diarrheal illnesses such as cryptosporidiosis.

CLEAN WATER

In urban areas, properly treated drinking water must be made available to the public by the municipal or other authorities through a piped system, at stand posts or through tanker trucks. The most commonly used method for disinfecting water supplies on a public health scale is the use of chlorination [1]. Chlorine may also be used at an individual level for disinfection of drinking water. Over 98% of water supply systems that disinfect drinking water use chlorine because of its germicidal potency, economy and efficiency. In addition, chlorine-based disinfectants are the only major disinfectants with the lasting residual properties that prevent microbial regrowth and provide continual protection throughout distribution from the treatment plant to the tap. Chlorine disinfects due to the formation of hypochlorous acid; the pH of the solution should be at or below 7 to achieve high levels of hypochlorous acid rather than the hypochlorite salt. Fortunately, most water available has pH of 7 or below. When water is turbid and has much organic matter, the amount of chlorine that must to added to provide free residual chlorine is much greater than with clean water. Ideally, water should be tested to determine the basic chlorine demand, which is estimated by adding the chlorine and measuring residual chlorine levels after 60 minutes [2]. A free residual chlorine concentration of 0.5 mg/L or more and exposure time of 1 hour are recommended for disinfection. The estimated basal chlorine demand (see above) needs to be added to the desired free residual chlorine concentration (0.5 mg/L) to determine the total amount of chlorine that will need to be added to disinfect the water. Chlorination may be achieved by using chlorine gas, chloramines, or perchorlor (high test hypochlorite, HTI) to the water. Chlorine gas is favoured by many, but chloramines which are compounds of chlorine with ammonia have some advantages. HTI is a calcium compound that releases chlorine for disinfection. In practice, bleeding powder (chlorinated lime, CaOCl2) is often used to chlorinate water supplies. When fresh, the available chlorine is approximately 35%, but in practice this is taken to be 25%. 4 kg of bleaching powder in 20 litres water provides a 5% chlorine solution (2.5% available chlorine) when fresh. For disinfecting 1000 L water, it is necessary to add 2.5 g of bleaching powder, 1 g of HTI, or 14 mL of liquid bleach (5% sodium hypochlorite). Chlorine tablets developed by the National Institute of Environmental Engineering (NIEE), Nagpur, are useful in chlorination: one 0.5 g tablet may be used per 20 L water. The concentration of free residual chlorine must be measured in any public water supply; recommended levels are 0.5 mg/L at all points in a piped water system, 1 mg/L at stand posts in systems with stand posts, and 2 mg/L in tanker trucks at filling. Free chlorine levels are measured by the orthotolidine test where 0.1 mL OT reagent is added to 1 mg/L water, and the yellow colour that develops is matched against standards. Readings taken after 10 seconds reflect free chlorine levels, while readings taken after 20 minutes reflect both free and bound chlorine. The orthotolidine arsenaile (OTA) test is a refinement of the OT test. Chlorination is effective against most enteric pathogens, however it may sometimes not be effective against protozoa. Membrane filtration of public water supplies is practised in many developed countries, and has reduced the incidence of diarrhea due to protozoa such as Cryptosporidium parvum [3].

In rural areas, drinking water may be obtained from tube wells, from protected dug wells or protected springs. In the case of the two latter sources, it must be ensured that there is no faecal contamination of the soil within a 50 feet radius of the well [4]. Wells must be fenced off to keep animals away; household containers must not be used to take water from the well; a hand winch with bucket must be installed and the bucket should not be allowed to touch the ground. Drainage patterns for surface water and soil permeability must be assessed in order to estimate the extent of protection needed around the well. Potential sources of pollution (latrines, refuse pits) should be located at least 15 metres away and downhill (not uphill) from the well. Good drainage must be ensured around the well by installing a cement apron or digging surface drainage canals. If surface water such as a lake or river is used for drinking water, a different set of strategy is required. People must be prevented from using the bank or shore for defecation. Distinct use zones may be created: upstream for the collection of drinking water; downstream for bathing, washing, laundering; and further downstream for watering animals. Defecation 'fields' must be located at least 50 metres away and downstream from use zones. A securely protected pipe may be installed in the drinking water zone. When safe water sources are not available, people must be
Diarrheal Diseases
taught that water can be made safe by bringing it to a boil, or by
adding chlorine-releasing chemicals, or by filtration, all of which can
reduce transmission of diarrheal diseases [5-9]. Filtration may be
effective in removing parasites and cholera bacteria, but must ideally
be followed by chlorination. Ultraviolet treatment and reverse osmosis
are facilities available for the relatively affluent section of the
population. There are efforts being made to determine low cost
alternative methods of treatment such as keeping water out in the
sunlight or adding easily available biomaterials in order to make
water microbiologically clean and safe, but no widely applicable
system has yet been developed that will reliably provide safe water.

Water that has been adequately chlorinated, by using minimum
recommended water treatment standards, will afford substantial
protection against viral and bacterial waterborne diseases. However,
chlorine treatment alone, as tested by using E. coli as a surrogate for
Escherichia coli O157:H7, might not kill some enteric viruses and parasites including Giardia
intestinalis, Entamoeba histolytica, and Cryptosporidium parvum. In areas
where chlorinated tap water is not available or where hygiene and
sanitation are poor, diarrhea and other waterborne enteric infections
may be avoided by drinking water that has been boiled or beverages
made with boiled water; by drinking canned or bottled beverages.
Boiling is by far the most reliable method to make water of uncertain
purity safe for drinking. Water should be brought to a vigorous rolling
boil for 1 minute and allowed to cool to room temperature; ice should
not be added. This procedure will kill bacterial and parasitic causes of
diarrhea at all altitudes and viruses at low altitudes. To kill viruses at
altitudes >2,000 m, water should be boiled for 3 minutes or chemical
disinfection should be used after the water has boiled for 1 minute.
Adding a pinch of salt or pouring the water several times from one
clean container to another will improve the taste.

Chemical disinfection with iodine is an alternative method of
personal drinking water treatment when it is not feasible to boil water.
However, this method cannot be relied on to kill Cryptosporidium
unless the water is allowed to sit for 15 hours before it is drunk. Two
well-tested methods for disinfection with iodine are the use of tincture
of iodine and triclosan tablets, available commercially. If water is cloudy,
the number of tablets used should be doubled; if water is extremely cloudy,
(>5°C), an attempt should be made
to warm the water, and the recommended contact time should be
increased to achieve reliable disinfection. Cloudy water should be
strained through a clean cloth into a container to remove any sediment
or floating matter, and then the water should be boiled or treated with
iodine.

Ceramic filters will provide varying degrees of protection against
microbes. Reverse-osmosis filters provide protection against viruses,
bacteria and protozoa, but they are expensive, and the small pores on
this type of filter are rapidly plugged by muddy or cloudy water. In
addition, the membranes in some filters can be damaged by chlorine
in water. Microstrainer filters with pore sizes in the 0.1- to 0.3-μm
range can remove bacteria and protozoa from drinking water, but
they do not remove viruses. All filters, viruses, travelers using microstrainer
filters should be advised to disinfect the water with iodine or chlorine
after filtration. Filters with iodine-impregnated resins are most effective
against bacteria, and the iodine will kill some viruses; however, the
contact time with the iodine in the filter is too short to kill the protozoa
Cryptosporidium and in cold water, Giardia. Filters collect organisms
from water. Anyone changing cartridges should wear gloves and
wash hands afterwards. Filters may not remove Cryptosporidium as
well as boiling does because even good brands of filters may sometimes
have manufacturing flaws that allow small numbers of organisms to
pass through the filter. In addition, poor filter maintenance or failure
to replace filter cartridges as recommended by the manufacturer can
cause a filter to fail.

SAFE DISPOSAL OF WASTE
Safe disposal of waste is another measure that is necessary to
prevent diarrhea [4]. Sanitary latrines may not be available in rural
areas or especially in conditions where large groups of people
congregate such as for fairs, funerals, religious festivals etc. In an
emergency, simple pits can be dug for disposal of human excreta.

These should measure 0.3 x 0.3 metre, be located at least 30 metres
from a well or ground source of drinking water, should not be in
marshy soil or upland from the water source, and at least 6 metres from
the nearest house. After use, a layer of soil should be laid down in the
pit, and if there is an epidemic of diarrhea, a layer of slaked lime
should also be laid down in the pit. Trench latrines, to accommodate
20 people, may serve a similar purpose. In hot and dry climates,
defecation fields may be preferable to badly used and poorly maintained
pit latrines. Defecation fields should be at least 50 metres away
from water sources or houses, but near enough to be easily reached.
Showers should be available for burying the faeces. Children’s stools,
in particular, can be a source of diarrhoea and need to be buried
or put into a latrine [10]. Disposal of solid waste and of waste water
also needs consideration under these conditions.

The causes of water borne outbreaks may differ between community
water systems and non-community water systems (Table 9.5), and
appropriate measures have to be taken for prevention. An analysis of
46 studies reporting water, sanitation, and hygiene interventions on

Table 9.4 : Methods to disinfect personal or household
drinking water in an emergency

Personal drinking water : A concentrated (stock) solution of
the chlorine solution is made, which is then used to treat larger quantities
of water. The stock solution must NOT be drunk or used directly and
must be kept safely out of the reach of children. To make up the
concentrated stock solution, add 2 teaspoons (8 grams) of sodium
hypochlorite OR 5 teaspoons of bleaching powder (20 grams) to half
a litre of water. This concentrated stock solution may be added to
drinking water in the following proportion ensuring that there is
proper mixing : 1 litre water 3 drops stock solution; 30 litres water 1
teaspoon stock solution. Treated water should be allowed to stand for
half to one hour before using.

Household well : Find the volume of water in the well from
the formula Volume (litres) = 3.14 x diameter of well x height of water
column x 1000 4 where diameter and height are in metres. Add 2.5 g
of good quality bleaching powder per 1000 litres of water into a
bucket (not more than 100 g per bucket) and make a thick paste. Fill up
to nearly three fours. Stir well, allow to settle and decant the
supernatant chlorine solution to another bucket. The bucket is lowered
into the water at least 1 m below the water surface and agitated violently.
Treated water may be used after an hour of contact.

Double pot method : This method validated by NEFERI, Nagpur,
consists of using two cylindrical pots, one inside another. The outside
pot is 30 cm in height and 25 cm in diameter and is made of
plastic. A hole 1 cm diameter is made in each pot, at the upper portion
in the inner pot, and 4 cm above the bottom in the outer pot. 1 kg of bleaching powder
mixed with 2 kg of coarse sand is moistened and filled into the inner
pot up to 3 cm below the hole in the pot, and this pot lowered into the
outer pot, which is then closed with polyethylene foil. The double pot
is lowered into the well 1 metre below the water surface. The pot
disinfects water satisfactorily for 2-3 weeks in household wells
containing about 4500 litres of water.

Table 9.5 : Common causes of waterborne outbreaks

<table>
<thead>
<tr>
<th>Causes of Outbreak</th>
<th>Percent of Outbreaks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Contamination of the distribution system</td>
<td>29%</td>
</tr>
<tr>
<td>Inadequate disinfection of unfiltered surface water</td>
<td>24%</td>
</tr>
<tr>
<td>Inadequate or no disinfection of groundwater</td>
<td>25%</td>
</tr>
<tr>
<td>Inadequate filtration of surface water</td>
<td>11%</td>
</tr>
<tr>
<td>Miscellaneous or unknown causes</td>
<td>11%</td>
</tr>
</tbody>
</table>

Crumey Community Water Non-Community Water Water Systems

<table>
<thead>
<tr>
<th>Causes of Outbreak</th>
<th>Percent of Outbreaks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Contamination of the distribution system</td>
<td>7%</td>
</tr>
<tr>
<td>Inadequate disinfection of unfiltered surface water</td>
<td>8%</td>
</tr>
<tr>
<td>Inadequate or no disinfection of groundwater</td>
<td>72%</td>
</tr>
<tr>
<td>Inadequate filtration of surface water</td>
<td>1%</td>
</tr>
<tr>
<td>Miscellaneous or unknown causes</td>
<td>12%</td>
</tr>
</tbody>
</table>
diarrhea morbidity revealed that all of these reduced significantly the risks of diarrhea illness to a similar extent, with the relative risk estimates from the overall meta-analyses ranging between 0.63 and 0.75. Water quality interventions (point-of-use water treatment) were found to be more effective than previously thought, and multiple interventions (consisting of combined water, sanitation, and hygiene measures) were not more effective than interventions with a single focus [1].

Food Safety and Personal Hygiene Measures

Food is an important vehicle for the transmission of diarrheal illnesses. There is therefore a need for proper controls for handling and processing of food. Safe practices include avoidance of raw foods (except undamaged fruit and vegetables which can be hygienically peeled by the consumer), cooking food until it is hot throughout, eating food while it is still hot, or reheating food that has been cooked earlier and cooled, and washing and thoroughly drying all cooking and eating utensils before use. Handwashing (with or without soap or ash), after defecation and before preparing and serving food, has repeatedly been shown to be very effective in preventing transmission of diarrheal illness [12]. Where water might be contaminated, ice should also be considered contaminated. If such contaminated ice has been in contact with containers used for drinking, the containers should be cleaned thoroughly, preferably with soap and hot water, after the ice has been discarded. It is safer to drink a beverage directly from the can or bottle than from a questionable container. However, water on the outside of beverage cans or bottles might also be contaminated. Therefore, wet cans or bottles must be dried before they are opened, and surfaces that will have direct contact with the mouth should be wiped clean. Where water might be contaminated, travelers should be advised to avoid brushing their teeth with tap water.

All raw food is subject to contamination. Particularly in areas where hygiene and sanitation are inadequate, the traveller should be advised to avoid salads, uncooked vegetables' unpasteurized milk and milk products such as cheese. To eat only food that has been cooked and is still hot or fruit that has been peeled by the traveller personally. Undercooked and raw meat, fish and shellfish can carry various intestinal pathogens. Cooked food that has been allowed to stand for several hours at ambient temperature can provide a fertile medium for bacterial growth and should be thoroughly reheated before serving. Consumption of food and beverages obtained from street food vendors has been associated with an increased risk of illness.

Breast feeding is an important practice that helps in prevention of diarrhea in babies. Mothers should be encouraged to give only breast milk to their babies for the first 4–6 months and then continue breast feeding up to the age of two years. If the mother works outside the home, then she should breast feed before leaving home, on returning in the evening and at any other time that she is with the baby. If the infant has been weaned from the breast, boiled diluted cow milk or infant formula reconstituted in boiled and cooled water is safest. In weaned children, weaning practices should be improved. Family members should wash hands before preparing weaning food or feeding the baby. Food should be prepared in a clean place using clean pots and utensils. Cooked food should be eaten while still hot [13]. Plenty of water should be used for hygiene and clean water should be used for drinking. Repeated hand washing is useful in preventing diarrheal disease.

Specific precautions are required to prevent disease due to Shiga toxin producing E. coli. This organism, which produces haemorrhagic colitis, is responsible for point source outbreaks where contaminated or undercooked beef is particularly implicated. Similar precautions are necessary for prevention of point source outbreaks due to Campylobacter jejuni. Farm hygiene measures are important both before harvest and after harvest (i.e., milk processing and meat packing) for decreasing the risk of dairy product contamination. Preharvest measures include sanitation during milking and management practices designed to decrease pathogen prevalence in the dairy herd or chicken farm (i.e., animal factors, manure handling, drinking water, and both feeds and feeding). Postharvest measures include practices or treatments that could be implemented during processing of milk, beef, or their products to eliminate or minimize pathogen contamination [14]. Adequate monitoring and reporting is necessary for prevention from the community health point of view.

Some species of fish and shellfish can contain poisonous biotoxins, even when well cooked. The most common type of biotoxin in fish is ciguatoxin. The flesh of the barracuda is the most toxic toads and should always be avoided. Red snapper, grouper, amberjack, sea bass and a wide range of tropical reef fish contain the toxin at unpredictable times. The potential for ciguatera poisoning exists in all subtropical and tropical insular areas of the Caribbean and the Pacific and Indian Oceans where the implicated fish species are eaten. Symptoms of ciguatera poisoning include gastrointestinal follow by neurologic problems such as dysesthesias, temperature reversal, weakness and rarely, hypotension. Scombroid is another common fish poisoning that occurs worldwide in tropical as well as temperate regions. Certain fish such as the bluefin, yellowfin tuna, mackerel, mahimahi, herring, amberjack and bluefish may contain high levels of histidine in their flesh. With improper refrigeration or preservation, histidine is converted to histamine, which can cause flushing, headache, nausea, vomiting, diarrhea, and urticaria.

CHEMOPROPHYLAXIS

Giving an antibiotic to prevent spread of cholera is of use only when the secondary attack rate within a community is high, i.e., at least one household member in five becomes ill after the first affected individual. In such instances, selective chemoprophylaxis (e.g. Doxycycline 300 mg as single dose) may be given to all close family contacts.

Chemoprophylaxis of travellers' diarrhoea has been attempted using rifaximin, a poorly absorbable antibiotic [15]. This was effective in preventing diarrhea in Americans travelling to Mexico, in whom the expected pathogen is toxin producing Escherichia coli. Its use in other settings where other enteropathogens may prevail is not yet tested. However, it is probably not practical to advise the use of this drug in the total population that travels. In most cases, the prompt treatment of a diarrheal illness, should it occur, is likely to have more impact.

VACCINATION

Vaccines for prevention of diarrhea have not measured up to their initial promise [16]. A killed cholera vaccine comprising the Inaba and Ogawa strains of Vibrio cholerae was available for decades. This vaccine did not provide reproducible immunity and any immunity it provided was for only six months (Table 9.6). The World Health Organization recommended against routine of this vaccine for travelers and the vaccine is therefore defunct. Several oral vaccines have more recently been tested for cholera. Cholera vaccines frequently lack the required potency. Most are not very effective, any protection that does occur lasts only for 3-6 months and vaccination does not prevent subclinical infection or spread of infection within the community. The World Health Organization has not recommended the use of killed cholera vaccine in prophylaxis any longer. A number of oral cholera vaccines have been developed. They are generally of two types, attenuated live cholera vibrios or oral B subunit alone as
Diarrheal Diseases

Table 9.6: Summary data of commercially available cholera vaccines

<table>
<thead>
<tr>
<th>Vaccine</th>
<th>Age</th>
<th>Dose schedule</th>
<th>Route</th>
<th>Time to protection</th>
<th>Time of booster</th>
<th>Prepotency (Median)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phenol-inactivated whole cell vaccine</td>
<td>6 months</td>
<td>2 doses 1-4 weeks apart</td>
<td>IM or SC</td>
<td>6 days after 2nd dose</td>
<td>6 months</td>
<td>30-50%</td>
</tr>
<tr>
<td>Wyeth-Ayerst Laboratories, USA</td>
<td></td>
<td></td>
<td>Oral</td>
<td>7 days after final dose</td>
<td>6 months</td>
<td>50-85%</td>
</tr>
<tr>
<td>Oral killed whole cell-recombinant B subunit vaccine</td>
<td>2 years</td>
<td>2-3 doses 1-6 weeks apart</td>
<td>Oral</td>
<td>7 days after final dose</td>
<td>2 years</td>
<td>50-85%</td>
</tr>
<tr>
<td>Dukoral, SBL Vaccine AB, Stockholm, Sweden</td>
<td></td>
<td></td>
<td>Oral</td>
<td>8 days after dose</td>
<td>6 months</td>
<td>60-100%</td>
</tr>
<tr>
<td>Oral live attenuated Vibrio cholerae strain CVB 103-H9R</td>
<td>2 years</td>
<td>Single dose</td>
<td>Oral</td>
<td>8 days after dose</td>
<td>6 months</td>
<td>60-100%</td>
</tr>
<tr>
<td>Maastricht Berna, Switzerland Serum Vaccine Institute, Berna, Switzerland</td>
<td></td>
<td></td>
<td>Oral</td>
<td>8 days after dose</td>
<td>6 months</td>
<td>60-100%</td>
</tr>
</tbody>
</table>

More recently, genetically engineered vaccines have been generated, which express B subunit but with the rest of the toxin operon removed from the bacterium. The cholera vaccines have had variable success in field trials. In a recent field trial in Mozambique, an oral B subunit vaccine was found to afford approximately 75% protection against cholera, but not other diarrhoea when administered in two doses. An indigenous developed Indian vaccine (IMTECH, Chandigarh) consisting of an attenuated cholera vibrio expressing the toxin B subunit alone, is currently in Phase 1a clinical trials in India.

The first live oral tetravalent rotavirus vaccine (rhelus-based RRV-TV), was incorporated into the US immunization schedule in 1998. The vaccine was withdrawn in 1999 when reports of cases of intussusception were linked to recent vaccination. Several new rotavirus vaccines are in late stages of development [19,20]. One vaccine (Rotarix, GlaxoSmithKline) was licensed for sale in Mexico in 2004 after clinical trials. A second live oral pentavalent vaccine (Rotavec, Merck) has completed clinical trials in the United States and Europe and may be licensed shortly within the US and elsewhere. An Indian live oral vaccine is also in development. Meta-analyses of several trials show that rotavirus vaccine protects against both rotavirus and non-rotavirus diarrheas.

ETEC organisms express fimbrial colonization factor antigens that function as adhesions to promote their attachment to the small intestinal epithelium. They secrete either [or both] of two major protein enterotoxins that induce fluid and electrolyte secretion. Vaccine development during the last decade has targeted the components of the three major colonization factor antigens as well as the immunogenic heat-labile enterotoxin [21]. This strategy was expected to cover 90% of infecting strains, leaving unprotected only those strains without major colonization factor antigens and those that express only the poorly immunogenic heat-stable enterotoxin. Recent experiences have questioned the validity of the current vaccine strategy since new reports indicate that the number of recognized colonization factor antigens of ETEC has increased to more than 21. Epidemiologic field studies of children in endemic areas suggest that infection with ETEC of a given colonization factor antigen/toxin phenotype may not confer protection on reinfection with other strains of the same colonization factor antigen/toxin phenotype and a major field trial of a heat-killed ETEC vaccine expressing colonization factor antigens and containing the B subunit of cholera toxin as a surrogate for E. coli heat-labile enterotoxin was ineffective against ETEC infections that should have been "vaccine preventable." Although new vaccines are being developed to improve immunogenicity over that of the heat-killed vaccine, the current strategy for antigen inclusion has been challenged and new, common antigens may have to be defined to achieve the goal of an effective vaccine against ETEC.

Vaccines against Shigella and other enteric pathogens are in variable stages of development, but there is currently no vaccine available for clinical use in prevention of diarrhea [22,23].

PROBIOTICS

Probiotic bacteria may prevent diarrhea [24]. Probiotics expressing recombinant molecules are also being developed. A harmless Escherichia coli strain (CWG308), expressing glycosyltransferase genes from Neisseria meningitidis or Campylobacter jejuni resulting in the production of a chimeric lipopolysaccharide capable of binding heat-labile enterotoxin with high avidity, has been shown to prevent traveler's diarrhea caused by enterotoxigenic Escherichia coli [25].

References


15. DuPont HL, Jiang ZD, Oldburyen PC, Ericsson CD, de la Cabada FF, Ke S, DuPont

---

Textbook of Environmental Emergencies


