Case 19-2011: A 4-Year-Old Haitian Boy with Vomiting and Diarrhea

Jason B. Harris, M.D., M.P.H., Louise C. Ivers, M.D., and Mary Jane Ferraro, Ph.D., M.P.H.

Dr. Ana A. Weil (Medicine): A 4-year-old Haitian boy was admitted to a hospital in Haiti affiliated with this hospital because of vomiting and diarrhea of 10 hours’ duration.

The patient had been well until approximately midnight the night before admission, when vomiting and diarrhea developed. After approximately 6 hours of symptoms, his parents brought him to the hospital by motorcycle taxi, traveling for 4 hours. On arrival, 10 hours after the onset of symptoms, episodes of vomiting and diarrhea were too numerous to count.

The patient’s parents said that he had not urinated for hours. He had reportedly previously been healthy. He lived in a small village in Haiti with his parents and sibling. His 8-year-old brother had had mild diarrhea the previous day.

On examination, the patient seemed irritable and was rapidly drinking offered liquids. The pulse was low volume, at a rate of 150 beats per minute; the respirations were shallow, without retractions, at a rate of 45 breaths per minute; and the skin was not hot to the touch. The blood pressure and temperature were not obtained because of lack of equipment. The weight was estimated at 15 kg. The eyes were sunken, skin recoil was less than 1 second but not instantaneous, capillary refill was 2 seconds, and the skin and mucous membranes were dry. The lungs were clear, and there was mild abdominal tenderness. During the examination, the patient passed a clear, watery stool. He was admitted to the hospital, where he shared a cot with a pediatric patient who had similar symptoms, including diarrhea.

A reduced-osmolarity oral rehydration solution (ORS) consisting of glucose, sodium chloride, potassium chloride, and trisodium citrate dihydrate (with 75 mmol of glucose per liter, 75 mmol of sodium per liter, 20 mmol of potassium per liter, 65 mmol of chloride per liter, and 10 mmol of citrate per liter), with a total osmolality of 245 mmol per liter, was administered. During the next hour, two episodes of vomiting and numerous episodes of diarrhea occurred.

On reexamination 1 hour after the initiation of treatment, the patient had ingested less than 200 ml. He was combative and pushed away the ORS. The pulse was weak, and the hands and feet were cool and clammy. Simultaneous attempts at insertion of intravenous catheters in the antecubital region and the hand were unsuccessful; the patient became increasingly obtunded.

On the third attempt at intravenous access, a catheter was inserted into the saphe-
The patient presented with a common problem. A bolus (500 ml) of isotonic crystalloid solution containing sodium chloride, sodium lactate, potassium chloride, and calcium chloride was administered, with manual pressure applied to the bag. The patient remained lethargic. Dextrose (30 ml of a 20% solution) was administered rapidly into the intravenous catheter, without improvement in mental status. A second intravenous catheter was placed in the right antecubital region. Another bolus (500 ml) of crystalloid solution was infused during a 30-minute period, with improvement in the level of consciousness, followed by a second liter of the solution during the next 2 hours.

Approximately 4 hours after presentation, episodes of diarrhea were occurring too often to count, the frequency of vomiting had decreased, and no urine output had occurred. On examination, the patient was eagerly drinking ORS, and his mental status was markedly improved. The eyes remained sunken, and skin turgor was slightly decreased from normal. Azithromycin (300 mg) was administered orally. His family was encouraged to have the patient consume 200 ml of ORS per stool produced. During the next 4 hours, he had at least six episodes of diarrhea and drank approximately 400 ml of ORS; 1 liter of the crystalloid solution was administered intravenously. Eight hours after presentation, the total intravenous intake was 3 liters, or approximately 200 ml per estimated kilogram of body weight. He had urinated twice. On examination, there were no signs of dehydration, the pulse was 100 beats per minute, and the respiratory rate was 30 breaths per minute, without rales or cough. During the remainder of the first day, an additional liter of intravenous solution was administered (a total of 4 liters during 24 hours, or approximately 267 ml per kilogram). Overnight, the frequency of diarrhea decreased, with an estimated 10 stools and no vomiting. Oral intake included less than 200 ml of ORS and some broth.

On the morning of the second day, the patient’s parents reported that he had cramping in his legs. On examination, signs of dehydration were present, including sunken eyes and slightly decreased skin turgor, with mild abdominal distention and tenderness. A bolus (500 ml) of crystalloid was administered intravenously over a period of 4 hours, and an educator was assigned to assist his parents in understanding the importance of ORS intake. During the next 4 hours, he consumed approximately 800 ml of ORS without vomiting. Signs of dehydration resolved, and abdominal distention decreased. Infusions of intravenous fluid were decreased to minimal flow. His parents were instructed again to match stool output by administering approximately 200 ml of ORS per stool, and his diet was increased to include meals of chicken broth and mashed bananas.

During the second night, three episodes of diarrhea occurred, and another episode between 8 a.m. and 2 p.m. On the third morning, the patient successfully consumed meals of solid food and ORS. He was discharged after 2.5 days, with instructions to the parents about oral hydration, point-of-use water sterilization, and hand sanitation with soap. One week after discharge, a diagnostic test result was received.

**Differential Diagnosis**

**Dr. Jason B. Harris:** I participated in the care of this child who presented with acute watery diarrhea during the second week of a cholera epidemic in Haiti, which began in October 2010 and is ongoing. The patient was admitted to a cholera treatment center that was established the previous week and was providing care for more than 100 patients daily who had diarrhea and, in many cases, other concomitant illnesses. No laboratory facilities were available. Like the vast majority of patients with diarrhea in developing countries, no specific laboratory diagnosis was made in this case.

The patient presented with a common problem. Children in developing countries have a median of three episodes of diarrhea annually, and diarrhea illness is the second leading cause of death among children, resulting in 1.6 million to 2.1 million deaths annually. Before the recent cholera epidemic, an average of 1 of every 93 children born in Haiti died from diarrheal illness before reaching their fifth birthday. For this child, the focus is on empirical management of the acute watery diarrhea, not on extensive clinical or laboratory investigations. Algorithms, such as those developed by the World Health Organization (WHO), are helpful in managing diarrheal illness in children in resource-limited communities.

**Differential Diagnosis of Diarrheal Illness**

The first step in the care of this patient is to classify the type of diarrheal illness (Table 1). Diarrhea lasting for more than 14 days is classified as persistent diarrhea. Persistent diarrhea is caused by a distinct set of organisms and is associated with...
<table>
<thead>
<tr>
<th>Classification</th>
<th>Definition</th>
<th>Associated Findings</th>
<th>Selected Causes</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Persistent diarrhea</td>
<td>≥14 Days of loose or watery stools, with a weight &gt;10 g/kg/day</td>
<td>Malnutrition, malabsorption, immunodeficiency, HIV, defining illness of AIDS</td>
<td>Enteroaggregative <em>Escherichia coli</em> (EAEC)</td>
<td>EAEC is a predominant cause of persistent diarrhea among children in whom a microbiologic cause is identifiable; 10% of cases of diarrhea due to EAEC persist for &gt;14 days.</td>
</tr>
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<td></td>
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<td></td>
<td>Cryptosporidiosis species</td>
<td>Cryptosporidiosis hominis and <em>C. parvum</em> typically cause symptoms lasting 10 to 14 days. Persistent diarrhea occurs more often in immunocompromised hosts.</td>
</tr>
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<td></td>
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<td></td>
<td>Common causes of acute diarrhea</td>
<td>Persistent diarrhea is often triggered by common causes of acute diarrhea (e.g., an episode of rotavirus).</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Shigella species</td>
<td>Shigella species are the most common cause of invasive childhood diarrhea globally. Complications include toxic megacolon, perforation, rectal prolapse, encephalopathy, seizures, hemolytic-uremic syndrome, and sepsis. Empirical therapy for invasive diarrhea is aimed at shigellosis.</td>
</tr>
<tr>
<td>Invasive diarrhea (acute bloody diarrhea)</td>
<td>Stools containing visible blood or melena</td>
<td>Fever, abdominal pain, tenesmus, mucus in stool</td>
<td>Nontyphoidal <em>Salmonella enterica</em></td>
<td>Infants, the elderly, and immunocompromised patients are at higher risk for disseminated infection and complications.</td>
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<td></td>
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<td>Campylobacter species</td>
<td>Predominant species are <em>Campylobacter jejuni</em> and <em>C. coli</em>; complications include Guillain–Barré syndrome.</td>
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<td></td>
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<td></td>
<td>Enteroinvasive <em>E. coli</em> (EIEC)</td>
<td>EIEC is closely related to shigella and causes a syndrome that is essentially identical to shigellosis.</td>
</tr>
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<td></td>
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<td></td>
<td>Enterohemorrhagic <em>E. coli</em> (EHEC)</td>
<td>Fever is usually absent. EHEC produces Shiga toxin associated with hemolytic–uremic syndrome; antibiotics may increase the risk. This is a less common cause of invasive diarrhea in developing countries.</td>
</tr>
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<td></td>
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<td></td>
<td><em>Entamoeba histolytica</em></td>
<td>This protozoal parasite causes diarrheal illness that is clinically indistinguishable from shigellosis but does not respond to empirical therapy for shigella species.</td>
</tr>
<tr>
<td>Noninvasive diarrhea (acute watery diarrhea)</td>
<td>Liquid or watery stools, typically ≥3 times a day without blood</td>
<td>Possible fever, possible mucus in stool</td>
<td>Rotavirus</td>
<td>Rotavirus is the predominant cause of gastroenteritis in infants and is often associated with vomiting and low-grade fever.</td>
</tr>
<tr>
<td>Noncholera</td>
<td></td>
<td></td>
<td>Enterotoxigenic <em>E. coli</em> (ETEC)</td>
<td>ETEC is the predominant cause of gastroenteritis in toddlers and older children in developing countries.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Calicivirus</td>
<td>Calicivirus is associated with abrupt onset of vomiting and diarrhea, along with low-grade fevers.</td>
</tr>
<tr>
<td>Cholera</td>
<td>Fever infrequent, mild illness indistinguishable from other causes of noninvasive diarrhea</td>
<td></td>
<td><em>Vibrio cholera</em> O1</td>
<td><em>V. cholera</em> O1 is associated with large epidemics and with disease that is endemic in a particular area. Vomiting and voluminous “rice water” diarrhea occur in patients with severe cases. <em>V. cholera</em> O1, biotype El Tor, is the cause of the current global pandemic of cholera.</td>
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<td></td>
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<td></td>
<td><em>V. cholera</em> O139</td>
<td>The O139 serogroup was briefly the predominant cause of epidemic cholera in South Asia in the 1990s; it is clinically indistinguishable from <em>V. cholera</em> O1.</td>
</tr>
</tbody>
</table>
malnutrition and chronic enteropathy. Persistent diarrhea should raise suspicion for underlying infection with the human immunodeficiency virus (HIV); in HIV-infected persons, unexplained persistent diarrhea is a defining illness of the acquired immunodeficiency syndrome.5

Since our patient had acute diarrhea, the next step is to classify the diarrhea as invasive (bloody) or noninvasive (watery). Invasive diarrhea is defined by grossly bloody or melanoitic stools. Most patients with invasive diarrhea have fever and mucus in the stool. Shigella species are the predominant cause of invasive diarrheal illness in children in developing countries,6,7 and empirical management of the illness should include antibiotics aimed at treating and preventing complications of shigellosis.

This child passed watery stools without blood and did not have a tactile fever. The most common causes of acute watery diarrhea are rotavirus in infants and enterotoxigenic Escherichia coli in children.6 There is increasing recognition of the role of caliciviruses in causing gastroenteritis in children and also in adults. Many acute systemic illnesses (e.g., measles, dengue fever, and malaria) may also present with diarrhea.

CHOLERA

In this case, cholera was suspected because the patient presented during a known epidemic. It is important to distinguish cholera from the other causes of noninvasive diarrhea. A rapid and simple laboratory test for Vibrio cholerae is dark-field microscopy, which, when positive, reveals characteristic darting bacteria. However, in resource-limited communities, the diagnosis of cholera is most often based on clinical suspicion that takes into account the local epidemiology of diarrheal illness. Although mild illness caused by V. cholerae is clinically indistinguishable from other causes of diarrhea, severe cholera is associated with greater losses of fluid and electrolytes than is seen with other causes of noninvasive diarrhea. Furthermore, patients with cholera benefit from the early administration of appropriate antibiotics. Finally, cholera can cause large epidemics.

A classic finding in cholera is “rice water” stool (Fig. 1D), which may contain more than 1 billion (109) organisms per milliliter. Patients with severe cholera may shed more than 10 trillion (1013) organisms per day.8 Although many V. cholerae serogroups have been identified in the environment, only serogroups O1 and O139 have caused epidemic cholera. V. cholerae O1, biotype El Tor, is the cause of the current global pandemic of cholera, which began in 1961. Emerging strains, termed “hybrid” or “variant” V. cholerae O1 El Tor, are the causes of more recent epidemics, including the current epidemic in Haiti.9 These variant strains appear to combine the enhanced ability of the El Tor biotype to persist in the environment with the greater virulence associated with the previously circulating classical biotype.

This case illustrates the manifestations of severe cholera, or cholera gravis. Epidemic strains of V. cholerae produce cholera toxin — a toxin resulting from the ribosylation of adenosine diphosphate — which causes chloride secretion and the loss of sodium and water into the lumen of the small intestine (Fig. 1C). Stool losses in cholera are typically isotonic, and the mean sodium concentration in the stool of children with cholera is double that seen in the diarrhea of children without cholera (Table 2).

DEHYDRATION AND REHYDRATION

In cases of rapid fluid losses, the large intestine’s capacity for reabsorption is overwhelmed and death may occur within hours. The WHO has provided guidelines for using the physical examination to estimate dehydration in children; laboratory tests provide little additional useful information.4 This patient had deeply sunken eyes, markedly decreased skin turgor, a weak pulse, and mental-status changes, suggesting a 10% loss of fluid per kilogram of body weight within 12 hours after the onset of symptoms. This is typical of severe cholera. During the first weeks of the cholera epidemic in Haiti, deaths occurred in the community a median of 12 hours after the onset of symptoms.11 Children with severe cholera typically present with 5 to 10% dehydration but have additional stool losses that may exceed 20% of their body weight during the first 48 hours after admission.12 Rehydration is the cornerstone of care for patients with cholera, but nutritional interventions, the appropriate use of antibiotics, and recognition of common complications and coexisting conditions are also important.

Rehydration requires the rapid replacement of the initial deficit and ongoing losses with isotonic fluids. Therapeutic fluids for patients with cholera are shown in Table 2. In the United States and other developed countries, a typical approach
For patients with severe dehydration, intravenous fluids are required immediately. Lactated Ringer's solution is the best and most widely available commercial intravenous fluid for cholera. Ideally, the entire fluid deficit should be replaced within 3 to 4 hours after the initiation of therapy in both children and adults. Provided that more than 300 ml per kilogram of isotonic intravenous and oral fluids to restore euvolemia during the first 24 hours of therapy, which is indicative of a rate of purging that is consistent with severe cholera.

Providing adequate volumes of isotonic fluids to patients with such massive ongoing losses is also a challenge, especially for health care workers who are unfamiliar with the fluid requirements of patients with severe cholera. Cholera cots were assembled at this cholera treatment center and generally are useful for recording stool output (Fig. 2). Because of space constraints, this patient shared a cholera cot and bucket with other patients; therefore, a reliable record of the patient's stool output was not made. However, ongoing losses can be estimated at 10 to 20 ml per kilogram per stool, and the volume of these losses can be added to the amount of fluids needed during the initial rehydration period. In this case, euvolemia was initially restored after 8 hours and approximately 200 ml per estimated weight in kilograms; however, had such rapid ongoing losses been factored in, the fluid could have been restored more rapidly, ideally within a 3-to-4-hour window.

Recurrence dehydration, leg cramps, and abdominal distention developed in this patient approximately 12 hours after the initial correction of his fluid deficit. Hypokalemia was the most likely cause of the leg cramps and abdominal distention. Hypokalemia is an important cause of death in patients with diarrheal illness who die after initial rehydration therapy. In this case, the hypokalemia and the recurrent dehydration might have been prevented if oral rehydration therapy had been used to replace ongoing diarrheal losses. Oral rehydration therapy provides more potassium than intravenous lactated Ringer's solution and is preferred over intravenous therapy whenever possible. Similarly, the resumption of normal feeding should also begin as soon as possible, to prevent the sequelae of malnutrition and such complications as hypokalemia and hypoglycemia.

to a child with dehydration is to use hypotonic solutions to replace estimated fluid and electrolyte deficits slowly over a 24-hour period. In resource-limited locations such as Haiti, children with diarrheal illness often present later, with more severe dehydration, and require more rapid rehydration with isotonic solutions, particularly patients with cholera. With optimal fluid management, the mortality associated with severe cholera is less than 0.2%. However, case fatality rates are usually higher in epidemic cholera, especially during the early stages, when there are obstacles to providing appropriate clinical care. This case illustrates some of the barriers to providing optimal rehydration therapy. Ideally, oral rehydration therapy is initiated at the onset of illness, in the home or in the community. This requires the local availability, knowledge, and acceptance of oral rehydration therapy. In this case, had ORS been used at the onset of illness, instead of 10 hours after the onset of symptoms, it is unlikely that life-threatening shock would have occurred during the patient’s hospitalization.
Antibiotic therapy

This patient received azithromycin early during his hospitalization, which is an appropriate treatment for the \textit{V. cholerae} strain that is circulating in Haiti. Antibiotics can lead to reductions of more than 50\% in stool volume and in the duration of diarrhea, from more than 4 days to 2 days. Antibiotics also reduce the shedding of viable \textit{V. cholerae} from more than 6 days to slightly more than 1 day. This treatment can be useful on a patient-by-patient basis and also facilitates more rapid discharge from cholera treatment centers, thus conserving resources for other patients.

After rehydration and antibiotic therapy, the next tier of care is to provide nutritional support and to recognize common complications and coexisting conditions seen in patients with cholera. Zinc supplementation (10 mg per day for infants less than 6 months of age and 20 mg per day for 10 days for children 6 months to 5 years of age) should be provided to reduce the severity and duration of childhood diarrheal illness in countries, such as Haiti, where zinc deficiency is common.\cite{4,18} Zinc has the added benefit of reducing the incidence of subsequent episodes of diarrhea for several months. In developing countries, children with diarrhea, such as this patient, are also at high risk for vitamin A deficiency and should receive supplementation with vitamin A. Patients with clinical signs of vitamin A deficiency should receive a three-dose series of treatment (50,000 IU for infants <6 months of age, 100,000 IU for infants 6 to 12 months of age, and 200,000 IU for children >12 months of age). In this case, neither zinc nor vitamin A was available. In endemic areas, coexisting conditions, such as pneumonia and sepsis, are a leading cause of death in patients with cholera.\cite{19} Therefore, reevaluation for clinical signs of pneumonia and sepsis after rehydration is important. In this case no such conditions were identified on sequential examinations after rehydration.

**Table 2. Chemical Composition of Diarrheal Stool and Therapeutic Solutions.*\**

<table>
<thead>
<tr>
<th>Route and Solution</th>
<th>Sodium (millimoles per liter)</th>
<th>Potassium (millimoles per liter)</th>
<th>Chloride (millimoles per liter)</th>
<th>Bicarbonate (millimoles per liter)</th>
<th>Citrate (millimoles per liter)</th>
<th>Glucose (millimoles per liter)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Losses</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cholera stool, adult</td>
<td>135</td>
<td>15</td>
<td>100</td>
<td>45</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cholera stool, child</td>
<td>105</td>
<td>25</td>
<td>30</td>
<td>30</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Noncholeraic stool, child</td>
<td>52</td>
<td>25</td>
<td>14</td>
<td>14</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Intravenous fluid</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal saline</td>
<td>154</td>
<td></td>
<td>154</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ringer’s lactate</td>
<td>130</td>
<td>4</td>
<td>111</td>
<td>28</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cholera saline†</td>
<td>133</td>
<td>13</td>
<td>98</td>
<td>48</td>
<td></td>
<td>140</td>
</tr>
<tr>
<td><strong>Oral rehydration solution</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Standard</td>
<td>90</td>
<td>20</td>
<td>80</td>
<td></td>
<td>10</td>
<td>111</td>
</tr>
<tr>
<td>Hypo-osmolar</td>
<td>75</td>
<td>20</td>
<td>65</td>
<td></td>
<td>10</td>
<td>75</td>
</tr>
<tr>
<td>ReSoMal‡</td>
<td>45</td>
<td>40</td>
<td>76</td>
<td></td>
<td>7</td>
<td>125</td>
</tr>
</tbody>
</table>

* Data are from the World Health Organization\cite{4} and Sack et al.\cite{10} The dashes indicate not present.
† Cholera saline may be prepared locally.
‡ ReSoMal is a modified oral rehydration solution for patients with severe malnutrition.

**ANTIBIOTIC THERAPY**

**Dr. Jason B. Harris’s Diagnosis**

Life-threatening diarrheal illness due to \textit{Vibrio cholerae}.

**PATHOLOGICAL DISCUSSION**

\textit{Dr. Mary Jane Ferraro:} The specimen that we received in the laboratory was from a different child in Haiti who had a similar illness at the same time as this child’s illness. The isolate that we obtained was identified in our laboratory as \textit{V. cholerae}. Susceptibility testing was performed and showed that the isolate was susceptible to tetracycline and azithromycin and was resistant to sulfa drugs.
and nalidixic acid. This isolate is undoubtedly the same one that had infected our patient, and this result confirms the diagnosis of cholera.

Dr. Harris, can you tell us about the additional molecular characterization that was performed on this isolate?

Dr. Harris: At the onset of the epidemic, the initial isolates were rapidly identified as *V. cholerae* O1, serotype Ogawa, by the National Public Health Laboratory in Haiti. The Centers for Disease Control and Prevention (CDC) subsequently reported that these were hybrid strains of *V. cholerae* O1, biotype El Tor. These are strains that produce the more virulent toxin that is associated with the previously circulating classical biotype of cholera. To determine the phylogeny of the organism, the isolate that Dr. Ferraro described underwent complete genome sequencing. Analysis of variable regions in the organism’s genome placed the isolate in the context of other known isolates in *V. cholerae* and showed that this was a seventh pandemic strain of *V. cholerae* O1 El Tor and that it was most closely related to isolates obtained in Bangladesh in the past decade. These phylogenetic comparisons were based on a single nucleotide variation in selected genes and in the content of selected hypervariable regions in both *V. cholerae* chromosomes. The isolate was distantly related to strains that had circulated in South America in the early 1990s and strains that are known to cause sporadic *V. cholerae* in the U.S. Gulf Coast.

**THE HAITIAN CHOLERA OUTBREAK**

Dr. Eric S. Rosenberg (Pathology): Dr. Louise Ivers is with us by telephone from Port-au-Prince, Haiti. Dr. Ivers, would you give us an update on the status of the epidemic there and the responses to the crisis?

Dr. Louise C. Ivers: This patient presented during the second week of the cholera epidemic in Haiti, which began in October 2010. We know now that the first cases came from the center of the country, but the cases that alerted authorities to...
the epidemic occurred in the large coastal town of Saint-Marc, 2 hours north of the capital city of Port-au-Prince. At the time of this conference, 4 months later, the epidemic continued to evolve. As of April 8, 2011, more than 248,657 cases of cholera had been treated and 4524 patients had died. There continue to be mini-peaks of cases reported, particularly in areas with poor road access and in rural isolated communities that traditionally have limited access to any health services. The number of new cases has decreased substantially since the early phase of the epidemic and now remains relatively stable. Thus, the epidemic has not ended yet, and as the rainy season approaches, the number of cases may increase.

**SANITATION AND CLEAN WATER**

In Haiti, access to clean water is lacking for the majority of the population, as it most likely was for this patient's family. Almost a decade ago, Haiti ranked the worst of 147 countries in terms of water resources, and little has happened since then to substantially improve services and infrastructure. Few households have access to a formal latrine. Housing and shelter, which were inadequate in rural Haiti before the earthquake of 2010, became even less sufficient when persons who were displaced by the earthquake moved to stay with family and friends in already overcrowded and often poorly constructed housing in the countryside. Many rural homes in Haiti are subject to flooding during the rainy season, even during moderate rainfall, and most have dirt floors, which increase the challenges of sanitation and hygiene.

**RESPONSE TO THE OUTBREAK**

The outbreak of cholera, the likes of which had never been seen by the current population of Haiti, caused huge pressure on what was already a weak public health infrastructure. Initially, the causes and methods of transmission of cholera were poorly understood by those at risk, and clean water and soap were not widely available in the areas affected; these two issues in the context of poor access to services led to high initial mortality rates. This patient exemplifies this problem: he did not have access to oral rehydration early enough to prevent severe complications. The learning curve for institutions and providers with no cholera experience was steep. In view of these challenges, the response to the outbreak was relatively fast. Cholera treatment centers were erected, and efforts were made to establish temporary solutions to the problem of the lack of potable water and to introduce hygiene measures. There has been a strong response from the government of Haiti and national and international partners, and there are now more than 400 cholera treatment facilities, such as the one that this patient entered and that saved his life.

As the months have passed, the training of service providers, access to services for patients, water-treatment education and supplies, and education in the community have all increased, contributing to increased survival and a reduction in the number of new cases of cholera. Institutional mortality nationwide, originally as high as 7 or 8%, has fallen to less than 2%. This patient's family was given instructions in water purification and hand hygiene at the time of the patient's discharge. They were also given supplies so they could put their education into practice. However, challenges remain. Rural isolated communities have poor access to health services in general; poor living conditions, lack of sanitation, and lack of access to clean water persist as a result of dire poverty.

**Dr. Rosenberg:** What is the likelihood that cholera will be eradicated from Haiti?

**Dr. Edward T. Ryan** (Infectious Diseases): We have learned from previous cholera outbreaks that once this organism gets a foothold in the water supply in impoverished areas, it is almost impossible to eradicate.

**ANATOMICAL DIAGNOSIS**

*Vibrio cholerae* O1 (toxigenic), serotype Ogawa (testing performed at the CDC).

This case was presented at the Medicine Grand Rounds, February 17, 2011.

Disclosure forms provided by the authors are available with the full text of this article at NEJM.org.

We thank Dr. Lawrence Ronan for helpful input.

**REFERENCES**

5. WHO case definitions of HIV for surveillance and revised clinical staging and immunologic classification of HIV-relat-


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