

Bacterial Food Poisoning



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Despite the advances of modern civilization, food poisoning still remains a common cause of gastrointestinal illness with an estimated 76 million persons annually experiencing foodborne illness in the United States. The two basic mechanisms in which illness can be transmitted through food are through a bacterial toxin or through bacterial invasion. These can result in either symptoms of nausea and vomiting or a diarrhea-predominant illness.

NAUSEA AND VOMITING CASE

A 17 year old high school athlete was vacationing at the beach and ate a hamburger with mayonnaise and lettuce at a local fast food restaurant. The sandwich did not taste exceptionally fresh, but was otherwise not unusual. Two hours later, uncontrollable nausea and vomiting developed and persisted for 6-8 hours. He had one loose bowel movement during that time and no fever. He felt better immediately after each episode of vomiting. No one else with him who

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ate at the same restaurant became ill. The next day he was completely better.

Introduction

It is clinically useful to divide foodborne illnesses based on symptoms. Symptoms of food poisoning are predominantly nausea and vomiting, diarrhea, and abdominal pain. Although there is some overlap between causative organisms, the order in which patients present with these symptoms can be used to differentiate between groups of causal agents (Figure 1). When combined with a brief history of possible food exposures and timing of symptom onset, the initial symptoms can help identify the most likely pathogen. This can guide the appropriate evaluation and treatment for the patient (1).

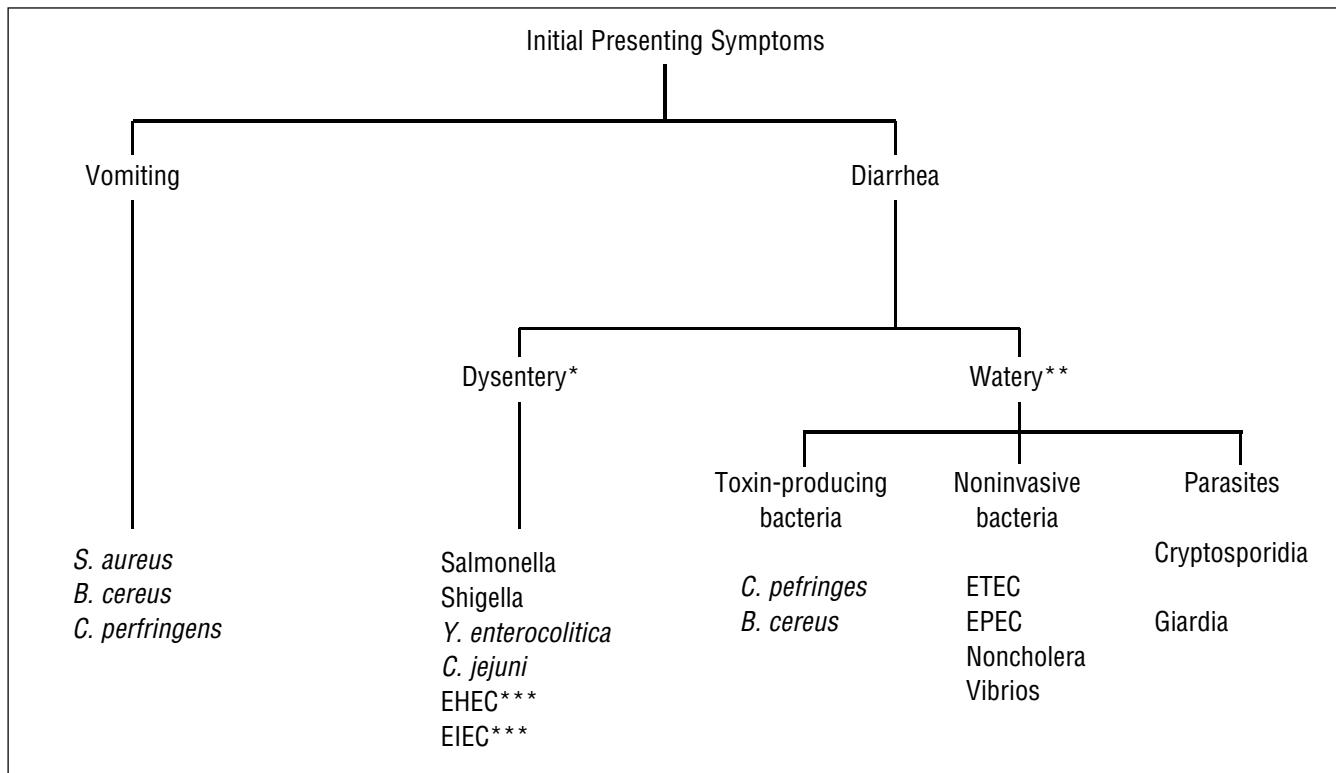


Figure 1. Symptomatic Approach to Causal Agents of Food Poisoning.

Modified from Surawicz (1)

*Dysentery—bloody diarrhea, fecal leukocytes, fever, abdominal pain. **Watery diarrhea, mild abdominal pain, low-grade fever. EHEC (Enterohemorrhagic *E. coli*), EIEC (Enteroinvasive *E. coli*), ETEC (Enterotoxigenic *E. coli*), EPEC (Enteropathogenic *E. coli*)

Nausea and Vomiting

Once history and physical examination suggest that a patient has food poisoning, then the clinician may use the initial symptom of nausea and vomiting to help guide therapy. *S. aureus* and *Bacillus cereus* are the most likely organisms to cause illness with predominantly nausea and vomiting (1). Symptoms usually begin within 6 hours of ingestion of a preformed toxin (2).

S. aureus is one of the most common causes of foodborne illness accounting for 1.5% of all recognized food poisoning outbreaks reported to the Centers for Disease Control (CDC) 1993–1997 (3). Staphylococcal food poisoning is most common during the summer months at large gatherings such as church picnics and family reunions. Foods high in salt, protein, and sugar content, such as dairy products, salads and meats, select for growth of *S. aureus*. If left unrefrigerated for more

than four hours, bacteria can produce enough toxin to cause symptoms of food poisoning. Nausea and vomiting after preformed toxin ingestion usually begins within six hours. Typically several people become ill within the same period if the same foods are eaten. Commercially prepared foods are rarely a source of *S. aureus* infection. Symptoms are typically brief and self-limited, and diarrhea is usually mild when present (1).

Enterotoxins A through E are the causative agents for *S. aureus* food poisoning, with enterotoxin A being most common. Enterotoxin F is associated with Staphylococcal toxic shock syndrome, not foodborne illness. The enterotoxins are heat-stable and are not absorbed. Since the enterotoxins do not elicit an immune response, there is no protective immunity to repeat exposure (4).

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B. cereus is a ubiquitous spore forming, gram-positive rod organism that is found in soil and water throughout the world and in most raw foods. This organism can produce either an emetic or diarrheal toxin depending upon the food in which it grows. The incubation period is usually 1–6 hours for the emetic syndrome, which presents with vomiting and abdominal cramps. The duration of illness is 2–10 hours (1). *B. cereus* should be strongly considered when the history reveals the ingestion of fried rice from a Chinese restaurant. The practice of parboiling rice, which involves boiling a large quantity of rice, draining it at room temperature, and flash frying it later, especially lends itself to spore production (4).

Food poisoning associated with preformed toxins is self-limiting, usually lasting only one day. However, microbiological evaluation is only helpful in this circumstance if it is part of a suspected epidemic. Severe pain, fever, or toxic appearance suggest an invasive pathogen and requires further evaluation.

DIARRHEA AND DYSENTERY CASE

A 30 year old is visiting his family on the Gulf Coast, and over the course of the weekend consumes various forms of seafood, shellfish, salads, and steak. On Sunday morning he develops a severe headache while out in the sun and comes in to lie down. He vomits one time then develops watery diarrhea. A few of the bowel movements contain blood and mucus. He denies significant fever, but feels weak and tired from the diarrhea. Two other people became ill with diarrhea, but none of the others see blood in their stool. He refuses diagnostic workup despite the admonition of his friend, a gastroenterologist. The diarrhea lasts for five days and is very slow to improve, but by day seven he is able to go back to work.

Diarrhea

Diarrhea resulting from food poisoning may be divided into bloody (inflammatory) and non-bloody (non-inflammatory). Bloody diarrhea is caused by tissue invasion and is frequently associated with fever, abdominal cramps, and bloody diarrhea. Non-bloody diarrhea may be produced by toxin producing bacteria, non-invasive bacteria, and parasites.

Bloody Diarrhea

The most common bacterial pathogens implicated with bloody diarrhea are *Salmonella*, *Shigella*, *Yersinia enterocolitica*, *Campylobacter jejuni*, *Vibrio parahemolyticus*, and both enteroinvasive *E. coli* (ETEC) and enterohemorrhagic *E. coli* (EHEC). The dysentery syndrome of fever, abdominal pain, and stool containing blood, mucus, and sheets of leukocytes is only present in about 40% of patients with invasive pathogens. Patients with EHEC may have minimal fever and fecal leukocytes and predominantly bloody diarrhea. The severity of disease may vary, and the decision to treat with antibiotics is frequently made before the results of stool cultures are available and must be based upon initial clinical presentation. Inflammatory bowel disease should be considered in young patients presenting with bloody diarrhea, and ischemic colitis considered in elderly patients (2,4).

Nontyphoidal salmonellosis is one of the most frequent causes of food poisoning in the industrialized world. *Salmonella* caused approximately 35% of outbreaks reported to the CDC via FoodNet in 2000 (5). The major reservoir for *Salmonella* organisms that cause human illness is poultry and livestock, even though outbreaks have been reported from such unusual sources as marijuana and pet turtles. There is a nationwide risk of *Salmonella* contaminated eggs, which has changed U.S. eating habits over the past two decades. One study found 50% of poultry in retail stores to be contaminated with *Salmonella* species, which has led to addition of antibiotics in animal feeds and increased incidence of multiple antibiotic resistance (2,4).

Patients with *Salmonella* gastroenteritis usually develop symptoms within 48 hours of ingestion. The spectrum of disease can vary from a few loose stools to a cholera-like illness with profuse diarrhea and dehydration requiring hospitalization. Fecal leukocytes are usually present and dysentery-like symptoms may occur. The diarrhea usually lasts 3–7 days with fever and will usually resolve in two to three days. Bacteremia and serious complications are uncommon, but are seen more frequently in children and immunosuppressed patients (2).

After resolution of gastroenteritis, the mean duration of carriage of nontyphoidal *Salmonella* in stool is 4–5 weeks and varies by *Salmonella* serotype. Antimicrobial therapy, including fluoroquinolones may pro-

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long intestinal carriage and may increase the incidence of bacteriologic relapse. Therefore, antimicrobials should not be routinely used in patients with mild to moderate gastroenteritis. Antimicrobials should be reserved for infants, elderly or immunosuppressed, signs or symptoms of sepsis, likely bacteremia, focal infection, and chronic typhoidal carrier states (2,4).

Worldwide, *Campylobacter* species is the most common cause of bloody diarrhea, and in 2000 it was the most common cause of foodborne illness in the U.S. accounting for just over one-third of reported outbreaks to the CDC via FoodNet (5). *Campylobacter* species are comma shaped, motile, gram-negative rods found in wild and domestic animals. Common sources include undercooked poultry and raw milk. The spectrum of disease is broad. There is usually a 12- to 24-hour prodrome of fever, headache, and malaise followed by crampy abdominal pain and diarrhea that resolve within 5–7 days. The fever produced by the illness can be low grade or have high spikes. Selective cultures are required for diagnosis, and antimicrobial therapy is reserved for patients with severe diarrhea or immunocompromise (4).

Shigella was the third most common cause of foodborne illness in the U.S. from 1996–1999. *Shigella sonnei* is the most common isolate in the United States whereas *Shigella flexneri* is seen more often overseas. Fecal oral transmission from person to person is the most common source of illness. Disease may occur with as few as ten organisms. Foodborne spread via eggs, cheese, and vegetables occurs most commonly during the summer and fall. Shigellosis causes disease through its enteroinvasive properties as well as in the production of Shiga toxin. Shigella bacilli invade colonic epithelial cells, multiply intracellularly, and invade adjacent cells. Shiga toxin is thought to damage endothelial cells of the lamina propria leading to microangiopathic hemolytic anemia and secondary renal failure seen in hemolytic uremic syndrome (HUS). Diagnosis is made by stool or blood cultures, and fluoroquinolones are the treatment of choice (4).

Recent outbreaks of enterohemorrhagic *E. coli* (EHEC) in the form of *E. coli* O157:H7 have been traced back to hamburger meat from a fast food restaurant in Washington state in 1993 (6), sprouts in California 1996–1998 (7), fresh cheese curds in Wisconsin in 1998 (8), and 35 million pounds of contaminated

ground beef was recalled in Colorado in 1997 from one meat processing plant, only to have another 18 million pounds of ground beef recalled in July 2002, from another processing plant (9). All of this publicity has made *E. coli* a household name. Infection with EHEC is characterized by hemorrhagic colitis and bloody diarrhea. The reasons that serotype O157:H7 is such an important pathogen is that it produces at least two different Shiga-like toxins, one of which is genetically indistinguishable from Shiga toxin. HUS and thrombotic thrombocytopenic purpura can therefore complicate *E. coli* O157:H7 infection (2,4).

Enteroinvasive *E. coli* is a rare colonic pathogen that has the ability to invade the colonic mucosa. It may be difficult to identify because the illness produced is indistinguishable from the initial symptoms of Shigellosis. The last large outbreak reported was from ingestion of soft cheeses on a cruise ship in 1971 (4).

Yersinia enterocolitica remains an underrecognized cause of diarrheal illness in the United States. The clinical features of *Y. enterocolitica* infection often include diarrhea with blood streaked stool and abdominal pain due to ileitis that can mimic acute appendicitis or Crohn's ileitis (1). The diarrhea and constitutional symptoms tend to improve by the second or third day of illness, but in some cases the diarrhea may last up to 45 days. Special media may be required to culture yersenia from stool. However, there is no evidence that antimicrobial therapy alters the course of this usually self-limited illness, and treatment is not recommended (2).

Vibrio parahemolyticus is a salt-water organism that is a major etiologic agent of bacterial diarrhea in Japan and has been implicated frequently in the U.S. as well. Nearly all confirmed cases have been associated with either consumption of raw or improperly stored seafood, or with contamination of food with seawater. The incubation period is 2–48 hours usually presenting in less than 24 hours with watery diarrhea, abdominal cramps, nausea and vomiting. Frank blood is present in the diarrhea less than 15% of the time. The infection is usually self-limited lasting 2–5 days. The diagnosis is confirmed by stool culture, but antibiotic therapy has not been shown to shorten the duration of illness (2).

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Non-bloody Diarrhea

Non-bloody diarrheal syndromes are characterized by profuse watery diarrhea. Although occasional fecal leukocytes may be present in the stool, sheets of fecal leukocytes, blood, and mucus are absent. Low-grade fever and abdominal pain may occur, but vomiting is uncommon. Once a threshold number of organisms are ingested, the bacteria cause diarrhea by producing enterotoxins in the small intestine. These different diseases can only be distinguished by diagnostic tests, but the imperative of treatment is to prevent dehydration whether or not a final diagnosis is made (4).

Clostridium perfringens is the most common cause of toxin-induced diarrhea and may produce one of two distinct food poisoning syndromes. In Western nations, type A organisms produce a 24-hour illness. However, a life-threatening syndrome known as enteritis necroticans is caused by type C organisms and currently only occurs in underdeveloped tropical areas. The type A illness in the U.S. usually occurs after consumption of high protein foods, such as meat or poultry products when there is a delay between cooking and serving the food. *C. perfringens* outbreaks are often connected with banquets and settings where large quantities of food are prepared in advance (2,4). One such incidence involved the consumption of minestrone soup (10).

B. cereus may produce a diarrheal syndrome that is indistinguishable from that of *C. perfringens*. However this syndrome is distinctly different from the emetic syndrome produced by *B. cereus* (1,4).

Enterotoxigenic *E. coli* (ETEC) produces disease by elaborating enterotoxins without invading or damaging intestinal epithelial cells. It may form one or more of at least three enterotoxins with different susceptibilities to heat. The heat labile toxin is similar to cholera toxin. ETEC is a major cause of illness in the traveler in developing countries, and usually manifests as watery diarrhea without vomiting. The illness is usually self-limited, lasting 3–5 days if left untreated. Diagnosis is clinical and treatment with antimicrobials shortens the duration of disease (2).

Fortunately, the prototype bacterial cause of watery diarrheas, *V. cholera*, is rare in the U.S. Once it colonizes the small intestine, it produces numerous enterotoxins that may cause profuse watery diarrhea

and vomiting, which may lead to severe dehydration and death within hours. The diagnosis of *V. cholera* is presumptive based on history, clinical manifestations and laboratory identification of toxigenic *V. cholera*-O1 and *V. cholerae* O139 Bengal. Rehydration is the only treatment necessary, but antibiotics are recommended because they shorten the period of cholerae excretion and reduce the volume and duration of diarrhea (2).

CONCLUSIONS

Food poisoning is still prevalent in the United States, commonly causing nausea, vomiting, and diarrhea. Most cases of foodborne illness are never reported to the CDC and are usually treated by primary care physicians. By dividing the syndromes into categories based on symptoms, one can feel comfortable with the most likely offending pathogen. Most cases are self-limited and do not require antimicrobial therapy. If there is suspicion of an outbreak, this should be reported as soon as possible so that an investigation may be undertaken. ■

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