Taking a Bite out of Food Poisoning

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Food poisoning, more properly referred to as a food-borne illness, is the result of ingestion of food (or water) contaminated with pathogenic microbes, toxic chemicals or toxins produced by microbes. Most cases of food poisoning are actually intoxications, caused by bacterial toxins, either ingested pre-formed or produced by the micro-organisms in the gastrointestinal (GI) tract.

Heavy metal intoxication (copper, zinc, tin, cadmium) may cause similar symptoms to those caused by pathogenic microorganisms. Incubation periods for the heavy metal intoxications are short, often ranging from five to 15 minutes, with resolution within two to three hours once emesis has cleared the offending material from the GI tract.

The symptoms of nausea, vomiting and abdominal cramps are due to irritation of the gastric mucosa. Most of the common food-borne illnesses present with abdominal cramps, nausea, vomiting and diarrhea and have a relatively acute onset. Table 1 demonstrates the most common causes of food poisoning caused by micro-organisms.

How do we get food poisoning?

Exposure to possible causes of food poisoning is virtually unavoidable in our society. Much of our food is handled, transported and prepared by dozens of (potentially unwashed) hands before it reaches our mouths. Any type of food can be a source of GI infection. Despite this, however, food poisoning is an uncommon occurrence.

Factors leading to the development of food poisoning are multi-fold, ranging from pathogen-related factors, such as intrinsic virulence and magnitude of exposure, as well as host factors, such as immune status, extremes of age, GI integrity and behaviour. Most potential causes of food poisoning are mitigated by a GI tract. The natural immunogenic activity of the intestine's mucosal-associated lymphoid tissue (MALT), including production of immunoglobulin A antibodies, prevents the adhesion of pathogens to the intestinal wall; the normal bowel flora prevent colonization by "foreign" microbes through the process of competitive inhibition and normal GI motility expels pathogens quickly before they have a chance to cause problems.

Any factor that alters the GI homeostasis will favour a GI infection, specifically if the patient has an altered GI structure or function or is taking medications that decrease gastric acidity (antacids, proton pump inhibitors, histamine blockers), slow intestinal motility (narcotics) or alter normal colonic flora (antibiotics).

The immune compromise associated with extremes of age, chemotherapy and infection with HIV can also play a role.

What are the mechanisms of food poisoning?

Most cases of food poisoning result in diarrhea that is either secretory or inflammatory, resulting from either damage to the intestinal mucosa causing inflammation (via cytotoxins) or alteration of the balance between absorption and secretion, causing loss of salt and water from the gut (via enterotoxins). Occasionally, pathogens may act by invading the GI mucosa and causing systemic infection or symptoms related to the production of neurotoxins.

What are the clinical presentations?

In most cases of infectious food poisoning, the symptoms are predominantly GI-related, manifesting as abdominal cramps, nausea, vomiting or acute diarrhea or any combination of these. The clinical manifestations relate to the infectious organism or toxin and the predominant site of involvement (small versus large bowel). There are two major types of diarrhea associated with food poisoning:

1. Secretory diarrhea

This type of diarrhea is characterized by the relative absence of damage or inflammation in the intestinal epithelium. As a result, there is not typically any frank blood or significant numbers of leukocytes in the stool sample. This type of diarrhea is classically caused by pathogens, which elaborate enterotoxins, usually involving the small bowel. The toxins can act very quickly and cause a relative imbalance between fluid absorption and secretion in the intestinal lumen. The result is acute onset of watery diarrhea, potentially within an hour of eating.

2. Inflammatory diarrhea

Inflammatory diarrhea is the result of damage to the intestinal wall caused by pathogens or by cytotoxins released by certain organisms. Damage or destruction of cells lining the gut results in bleeding and inflammation. As a consequence, the patient experiences bloody diarrhea that will demonstrate an elevated leukocyte count, if tested. The large bowel is more commonly involved. The onset of symptoms is generally delayed compared to the acute onset of secretory diarrhea. Micro-organisms that invade through the wall of the GI tract and enter the blood stream may produce only mild GI symptoms or none at all, with associated signs of systemic disease, such as fevers, chills and general malaise.

Which agents typically cause secretory diarrhea?

The most frequent causes of secretory diarrhea are shown in Table 1 and include *Staphylococcus aureus*, which produces a neurotoxin resulting in the acute onset of severe nausea, vomiting and diarrhea. It is short-lived, but can result in severe dehydration. *S. aureus* food poisoning is traditionally associated with foods containing creams and outbreaks have been traced back to food handlers with paronychia.

Bacillus cereus, which produces a toxin, results in severe vomiting that resolves within 24 hours. *B. cereus* has been implicated in food poisoning associated with fried rice from Chinese restaurants. *Clostridium perfringens* often results in a watery diarrhea after an eight- to 10-hour incubation period after consuming cooked meat products.

Exposure to possible causes of food poisoning is virtually unavoidable in our society.

Which agents typically cause inflammatory diarrhea?

The most frequently implicated micro-organisms in North America are shown in Table 1 and include *Campylobacter jejuni*, which is frequently associated with consumption of fecally contaminated unpasteurized milk and undercooked poultry products. The non-typhoidal salmonellae, such as *Salmonella enteritidis*, are associated with the consumption of raw meat and poultry, eggs, milk and other dairy products

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that are fecally contaminated.

Shigella spp can result in a fulminant bloody diarrhea associated with pus and mucous. Consumption of fecally contaminated foods, specifically, poultry and dairy products are the most frequently implicated foods. Long-term sequelae of infections with *Campylobacter spp*, *Salmonella spp* and *Shigella spp* include Reiter's syndrome and reactive arthritides.

There are many types of *Escherichia coli*, the enterotoxigenic form results in the most common cause of traveller's diarrhea, while enterohemorrhagic *E. coli* (E coli 0157:H7) produces a toxin that can result in severe inflammatory diarrhea and may result in hemolytic uremic syndrome. The most frequently implicated foods are under-cooked hamburger, unpasteurized dairy products, unpasteurized apple cider and contaminated water.

Clostridium botulinum can lead to a neuromuscular disorder resulting from the botulinum toxin blocking release of acetylcholine at the neuromuscular synapses. The characteristic finding is that of diplopia and flaccid paralysis. Canned foods have typically been implicated, as the canning process allows for an anaerobic environment allowing *C. botulinum* to flourish and produce toxins.

Finding the cause...

The following steps are vital to establishing the etiology of food-borne illness:

1. **History:** A travel and dietary history is critical as it will hold clues to dietary indiscretions and food and drink typically associated with different pathogens.
2. **Stool analysis:** Stool should be submitted for culture; if the patient has reported bloody diarrhea, this fact should also be indicated on the laboratory requisition, as the laboratory staff will initiate laboratory-based protocols for the detection of the enteroinvasive pathogens. Stool specimens may also be submitted for detection of leukocyte to determine whether there is inflammation. Depending upon the index of suspicion, a stool specimen for the evaluation of ova and parasites should also be performed. Ideally, specimens should be obtained on three separate days to increase diagnostic yield.
3. **Blood cultures:** If the patient appears toxic, blood cultures may actually yield the pathogen.
4. **Bowel examination:** Depending on the gastrointestinal symptoms, direct visualization of the gastrointestinal tract may be warranted. It is critical not to overlook non-infectious causes of inflammatory diarrhea, such as ulcerative colitis or Crohn's disease. Diagnostic evaluation, consisting of direct visualization by sigmoidoscopy, colonoscopy, endoscopy and radiographic means, is most appropriately guided in discussion with a specialist in bowel diseases.

What other agents can be implicated in the cause?

Viruses

Although viral gastroenteritis is not typically a food poisoning, any fecally contaminated food or water source can lead to viral gastroenteritis. The most frequently implicated viruses are the noroviruses, which have led to the contamination of food and water sources and outbreaks aboard cruise ships and holiday resorts. Norwalk-like viruses result in a short-lived vomiting illness, which may be associated with a transient diarrhea. The hepatitis A virus, which is associated with fecally contaminated water sources, may cause GI symptoms, ultimately manifesting with hepatitis.

Protozoans

Giardia lamblia is the most common protozoal cause of infectious diarrhea. Although not a characteristic cause of food poisoning, it has been implicated in outbreaks of diarrhea amongst those camping or consuming contaminated lake water where animals, particularly beavers, have defecated, leading to the colloquial name of this condition, "Beaver Fever." *G. lamblia* disease is characterized by abdominal bloating, cramping, nausea and flatulence in addition to malabsorption of nutrients, resulting in a greasy, foul-smelling diarrhea.

Seafood

Scombroid fish poisoning and ciguatoxin result from the consumption of seafood and can lead to a number of clinical manifestations. Scombroid fish poisoning results from histamine produced by certain marine bacteria that accumulate in the fish flesh; when consumed by humans, this results in clinical manifestations that

may be mistaken for allergic reactions. The symptoms include urticarial eruptions, bronchospasm, diarrhea, headache and diffuse erythema, developing almost immediately upon consumption of contaminated fish. The symptoms resolve in several hours. The most frequently implicated fish include tuna and mackerel.

Ciguatera toxin is typically found in tropical fish and results in a syndrome characterized by abdominal cramps, vomiting, diarrhea and, occasionally, neurologic changes, such as numbness and paresthesias of the lips, tongue and throat. Other neurologic symptoms have been observed, such as blurred vision, photophobia and transient blindness, pains in the legs and a sensation of looseness and pain in the teeth and, in severe cases, reversal of hot and cold temperature sensations. When evaluating return travellers with "allergic reactions" or vague neurologic symptoms, it is important to always maintain scombroid fish poisoning and ciguatoxin as potential etiologies.

How is food poisoning managed?

In the vast majority of cases, food poisoning results in transient symptoms that resolve promptly. The following are broad guiding principles for the management of persons with food poisoning:

1. Supportive therapy: Food and electrolyte replacement is important to ensure dehydration does not occur. In most cases, the illness associated with food poisoning is self-limited and no further therapy will be required, beyond replacement of fluids. For those with diarrhea alone, or limited vomiting, oral rehydration is sufficient. For those with more significant vomiting or voluminous fecal losses, parenteral rehydration may be prudent.

2. Anti-diarrheal agents: Anti-peristaltic agents are generally not recommended for the treatment of acute diarrhea. These agents slow GI motility and reduce the rate of clearance of the causative agent. It has been suggested that anti-diarrheal agents can cause an ileus or even toxic meg-colon in extreme cases.

3. Dietary modification: With viral enteritis, lactases on the microvilli may be stripped off. Lactose-containing food may exacerbate the underlying diarrhea, as this sugar is metabolized by bacteria in the bowel, leading to acid production. A bland diet is, therefore, recommended for those able to tolerate oral intake. Normal dietary habits can resume once the diarrhea or vomiting resolves.

4. Antimicrobial therapy: Most cases of food poisoning are actually toxin-mediated and do not require antimicrobial therapy. In the management of most of the enteroinvasive bacteria, such as *E. coli* 0157:H7, antibiotics are actually contraindicated as they may exacerbate the condition by lysing bacteria and releasing preformed toxins. Empiric antibiotic therapy, however, may be appropriate in selected circumstances, specifically with those who are the extremes of age (the elderly or infants) and who may become profoundly dehydrated with a GI infection.

Public health

Laboratory-based surveillance exists in all provinces for pathogens of public health importance. When the laboratory detects a pathogen of importance, the Public Health Department is notified directly. Regardless, it is important that all health-care providers be vigilant in looking for organisms of public health importance, such as those described herein. It is critical to notify the Public Health Department so that they may undertake the appropriate patient interviews and contact tracing to ensure that your case is not part of a larger outbreak. The department can intervene and instruct the patient on appropriate hygienic measures to prevent the spread of communicable agents.

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Turn the page for Table 1 ⟶

Table 1

Food poisoning classification—common causative agents

Organism	Source	Incubation period (Hours)	Pathogenic mechanism	Clinical features	Duration of symptoms (Hours)	Sources of diagnostic material
Bacillus cereus	Fried rice, cream dishes, meatballs, boiled beef, barbecued chicken	1-16	Toxin	Nausea, vomiting, abdominal cramps, diarrhea	< 1	Vomitus, stool, food
Clostridium perfringens	Beef, turkey, chicken	8-22	Toxin	• Diarrhea, abdominal pain • Nausea, vomiting and fever less common	< 1	Stool, food-contact surfaces
Vibrio parahaemolyticus	Seafood, rarely salt water or salted vegetables	12 (2-48)	Toxin	Diarrhea, abdominal pain, nausea, vomiting, headache, fever	2-10	Stool, food/ food-contact surfaces, seawater
Staphylococcus aureus	Ham, pork, canned beef, cream-filled pastry	1-6	Toxin	Nausea, vomiting, abdominal pain, diarrhea	< 1	Stool, vomitus, food/food-contact surfaces, nose, hands, purulent lesion on food preparer
Campylobacter jejuni	Milk, chicken, pet animals, beef	24-48	Direct invasion of bowel mucosa	Nausea, vomiting, bloody diarrhea, headache, myalgia, fever	7	Stool
Escherichia coli	Salads, beef unpasturized apple cider, contaminated water	24-96			1-4	Stool, food, water
Enterotoxigenic			Toxin	Nausea, vomiting, diarrhea		
***E. coli* 0157H7** (enterohemorrhagic)			Verotoxin	Hemolytic uremic syndrome with bloody diarrhea		
Salmonella spp	Eggs, meat, poultry	24	Direct invasion of bowel mucosa	Nausea, vomiting, abdominal cramps, bloody diarrhea	3	Stool from patients and food-preparation workers, raw food
Shigella spp	Milk, salads (potato, tuna, turkey)	24	• Direct invasion of bowel mucosa • Toxin	Nausea, vomiting, abdominal cramps, bloody diarrhea	3 (0.5-14)	Stool from patients, food workers
Norwalk virus	Shellfish, various foods, drinking water	12-48	Mucosal injury by virus	• Nausea, vomiting, watery diarrhea • Fever, headache, myalgia less common		24-48 Stool
Hepatitis A	Shellfish, fecally contaminated food, drinking water	4-6 weeks	Mucosal injury and cellular trauma due to virus replication	Flu-like illness, jaundice	Weeks	Viral assay
Scombroid	Tuna, mackerel, skip jack	Directly after ingestion	Toxin	Abdominal cramps, rashes, bronchospasm	3-6	Fish
Ciguatoxin	Toxin from various fish	< 1 hour to 30 minutes	Toxin	Abdominal cramps, nausea, vomiting, diarrhea, neurologic symptoms	< 10 days	Fish