



FOOD POISONING:

Food poisoning syndrome results from ingestion of water and wide variety of food contaminated with pathogenic microorganisms (bacteria, viruses, protozoa, fungi), their toxins and chemicals. Food poisoning must be suspected when an acute illness with gastrointestinal or neurological manifestation affect two or more persons, who have shared a meal during the previous 72 hours. The term as generally used encompasses both food-related infection and food-related intoxication.

Some microbiologists consider microbial food poisoning to be different from food-borne infections. In microbial food poisoning, the microbes multiply readily in the food prior to consumption, whereas in food-borne infection, food is merely the vector for microbes that do not grow on their transient substrate. Others consider food poisoning as intoxication of food by chemicals or toxins from bacteria or fungi.

Consumption of poisonous mushroom leads to mycetism, while consumption of food contaminated with toxin producing fungi leads to mycotoxicosis.

Some microorganisms can use our food as a source of nutrients for their own growth. By growing in the food, metabolizing them and producing by-products, they not only render the food inedible but also pose health problems upon consumption. Many of our foods will support the growth of pathogenic microorganisms or at least serve as a vector for their transmission. Food can get contaminated from plant surfaces, animals, water, sewage, air, soil, or from food handlers during handling and processing.

Classification Of Food Poisoning:

- I. **Based on symptoms and duration of onset**
 - a. Nausea and vomiting within six hours (*Staphylococcus aureus*, *Bacillus cereus*)
 - b. Abdominal cramps and diarrhoea within 8-16 hours (*Clostridium perfringens*, *Bacillus cereus*)
 - c. Fever, abdominal cramps and diarrhoea within 16-48 hours (Salmonella, Shigella, *Vibrio parahemolyticus*, Enteroinvasive *E.coli*, *Campylobacter jejuni*)
 - d. Abdominal cramps and watery diarrhoea within 16-72 hours (Enterotoxigenic *E.coli*, *Vibrio cholerae* O1, O139, *Vibrio parahemolyticus*, NAG vibrios, Norwalk virus)
 - e. Fever and abdominal cramps within 16-48 hours (*Yersinia enterocolitica*)
 - f. Bloody diarrhoea without fever within 72-120 hours (Enterohemorrhagic *E.coli* O157:H7)
 - g. Nausea, vomiting, diarrhoea and paralysis within 18-36 hours (*Clostridium botulinum*)
- II. **Based on pathogenesis**
 - a. Food intoxications resulting from the ingestion of preformed bacterial toxins. (*Staphylococcus aureus*, *Bacillus cereus*, *Clostridium botulinum*, *Clostridium perfringens*)
 - b. Food intoxications caused by noninvasive bacteria that secrete toxins while adhering to the intestinal wall (Enterotoxigenic *E.coli*, *Vibrio cholerae*, *Campylobacter jejuni*)
 - c. Food intoxications that follow an intracellular invasion of the intestinal epithelial cells. (Shigella, Salmonella)
 - d. Diseases caused by bacteria that enter the blood stream via the intestinal tract. (*Salmonella typhi*, *Listeria monocytogenes*)

Bacterial Etiology Of Food Poisoning:

Food infections by bacteria can be divided into two types:

1. those in which the food does not ordinarily support the growth of pathogens but merely carries them. E.g. Salmonella, Shigella, Vibrio etc.

2. those in which the food can serve as a culture medium for growth of pathogens to numbers that can infect the person.

Food borne infections by bacteria can also be classified as toxicosis and food-infections. In toxicosis, the toxins are released by bacteria such as Clostridia, Bacillus and Staphylococcus. In food-infections, the bacteria are ingested, which later initiate the infection.

Staphylococcus aureus:

S.aureus is gram positive cocci that occurs in singles, pairs, short chains, tetrads and irregular grape like clusters. It is present ubiquitously in the environment. Only those strains that produce enterotoxin can cause food poisoning. Food is usually contaminated from infected food handler. The food handler with an active lesion or carriage can contaminate food.

Incriminated food: Custard and cream filled bakery food, ham, chicken, meat, milk, fish, salads, puddings, pie etc.

Pathogenesis: If the food is stored for some time at room temperature the organism may multiply in the food and produce toxin. The bacteria produce enterotoxin while multiplying in food. *S.aureus* is known to produce six serologically different types of enterotoxins (A, B, C, C2, D and E) that differ in toxicity. Most food poisoning is caused by enterotoxin A. Isolates commonly belong to phage type III. These enterotoxins tend to be heat stable, with type B being most heat resistant. Low temperature heat inactivated enterotoxin can undergo reactivation in some food. Ingestion of as little as 23 µg of enterotoxin can induce vomiting and diarrhoea. Staphylococcal enterotoxins act as superantigens, binding to MHC II molecules and stimulating T cells to divide and produce lymphokines such as IL-2 and TNF-alpha, which induces diarrhea. The toxin acts on the receptors in the gut and sensory stimulus is carried to the vomiting center in the brain by vagus and sympathetic nerves.

Incubation period: Since the ingested food contains preformed toxin, the incubation period is usually 1-6 hours.

Clinical features: The onset is sudden and is characterized by vomiting and diarrhea but no fever. The illness lasts less than 12 hours. There are no complications and treatment is usually not necessary.

Laboratory diagnosis: The presence of a large number of *S.aureus* organisms in a food may indicate poor handling or sanitation; however, it is not sufficient evidence to incriminate a food as the cause of food poisoning. Staphylococcal food poisoning can be diagnosed if they are isolated in large numbers from the food and their toxins demonstrated in the food or the isolated *S.aureus* must be shown to produce enterotoxins. Dilutions of food may be plated on Baird-Parker agar or Mannitol Salt agar. Enterotoxin may be detected and identified by gel diffusion.

Bacillus cereus:

B.cereus is a gram positive aerobic spore bearing bacilli. It is found abundantly in environment and vegetation.

Incriminated food: Commonly associated with rice and vegetables.

Pathogenesis: During the slow cooling, spores germinate and vegetative bacteria multiply, then they sporulate again. Sporulation is also associated with toxin production. The toxin is heat-stable, and can easily withstand the brief high temperatures used to cook fried rice. The short-incubation form is most often associated with fried rice that has been cooked and then held at warm temperatures for several hours. Long-incubation food poisoning is frequently associated with meat or vegetable-containing foods after cooking. The short-incubation form is caused by a preformed heat-stable enterotoxin of molecular weight less than 5,000 daltons. The long-incubation form of illness is mediated by a heat-labile enterotoxin (molecular weight of approximately 50,000 daltons), which activates intestinal adenylate cyclase and causes intestinal fluid secretion.

Incubation period: 1-6 hours in short-incubation form and 8-16 hours in long incubation form.

Clinical features: *B. cereus* causes two types of food-borne intoxications. The 'emetic-type' or the short incubation type has an incubation period of 1 to 6 hours. It is characterized by nausea, vomiting and abdominal cramps and resembles *S. aureus* food poisoning in its symptoms and incubation period. Within 16 hours of eating contaminated fried rice, patients suffer a bout of vomiting that generally lasts for less than a day. The second type is manifested primarily by abdominal cramps and diarrhea with an incubation period of 8 to 16 hours. Diarrhea may be a small volume or profuse and watery. This type is referred to as the "long-incubation" or diarrheal form of the disease, and it resembles food poisoning caused by *Clostridium perfringens*. In either type, the illness usually lasts less than 24 hours after onset.

Laboratory diagnosis: The short-incubation or emetic form of the disease is diagnosed by the isolation of *B. cereus* from the incriminated food. The long-incubation or diarrheal form is diagnosed by isolation of the organism from stool and food. Isolation from stools alone is not sufficient because 14% of healthy adults have been reported to have transient gastrointestinal colonization with *B. cereus*.

Clostridium perfringens:

It is a gram positive anaerobic spore bearing bacilli that is present abundantly in the environment, vegetation, sewage and animal feces.

Incriminated food: food-borne outbreaks of *C.perfringens* involve meat products that are eaten 1- 2 days after preparation. Meats that have been cooked, allowed to cool slowly, and then held for some time before eating are commonly incriminated. Fish pastes and cold chicken too have been incriminated.

Pathogenesis: Spores in food may survive cooking and then germinate when they are improperly stored. When these vegetative cells form endospores in the intestine, they release enterotoxins. The bacterium is known to produce at least 12 different toxins. Food poisoning is mainly caused by Type A strains, which produces alpha and theta toxins. The toxins result in excessive fluid accumulation in the intestinal lumen.

Incubation period: 8-24 hours

Clinical features: Illness is characterized by acute abdominal pain, diarrhea, and vomiting. Illness is self-limiting and patient recovers in 18-24 hours.

Laboratory diagnosis: Since the bacterium is present normally in the intestine, their isolation from feces may not be sufficient to implicate it. Similarly, isolation from food except in large numbers ($>10^9$ /gram) may not be significant. The homogenized food is diluted and plated on selective medium as well as Robertson cooked meat medium and incubated anaerobically. The isolated bacteria must be shown to produce enterotoxin.

Clostridium botulinum:

It is a gram positive anaerobic spore bearing bacilli that is widely distributed in soil, sediments of lakes and ponds, and decaying vegetation.

Incriminated food: Most cases of botulism are associated with home canned or bottled meat, vegetables and fish. In general, the low and medium acid canned foods are often incriminated. The anaerobic environment produced by the canning process may further encourage the outgrowth of spores.

Pathogenesis: Not all strains of *C.botulinum* produce the botulinum toxin. Seven toxigenic types of the organism exist, each producing an immunologically distinct form of botulinum toxin. The toxins are designated A, B, C1, D, E, F, and G. Lysogenic phages encode toxin C and D serotypes. Food-borne botulism is not an infection but an intoxication since it results from the ingestion of foods that contain the preformed clostridial toxin. If contaminated food has been insufficiently sterilized or canned improperly, the spores may germinate and produce botulinum toxin. The toxin is released only after the death and lysis of cells. The toxin resists digestion and is absorbed by the upper part of the GI tract and then into the blood. It then reaches the peripheral neuromuscular synapses where the toxin binds to the presynaptic stimulatory terminals and blocks the release of the neurotransmitter acetylcholine. This results in flaccid paralysis. Even 1-2 μ g of toxin can be lethal to humans.

Incubation period: 12-36 hours

Clinical features: Common features include vomiting, thirst, dryness of mouth, constipation, ocular paresis (blurred-vision), difficulty in speaking, breathing and swallowing. Coma or delirium may occur in some cases. Death may occur due to respiratory paralysis within 7 days.

Laboratory diagnosis: Spoilage of food or swelling of cans or presence of bubbles inside the can indicate clostridial growth. Food is homogenized in broth and inoculated in Robertson cooked meat medium and blood agar or egg-yolk agar, which is incubated anaerobically for 3-5 days at 37°C. The toxin can be demonstrated by injecting intraperitoneally the extract of food or culture into mice or guinea pig.

Enterotoxigenic *E.coli* (ETEC):

E.coli are gram negative enteric bacilli that are carried normally in the intestine of humans and animals. Some specific serotypes harbor plasmids that code for toxin production. The enterotoxin production is limited to following O serotypes: O6, O8, O15, O25, O63, O78, O148 and O159.

Incriminated food: Infection is acquired by ingestion of food or water contaminated with ETEC. Contamination of water with human sewage may lead to contamination of foods. Infected food handlers may also contaminate foods. The infective dose is 10^6 - 10^{10} bacilli.

Pathogenesis: The bacteria colonize the GI tract by means of fimbriae to specific receptors on enterocytes of the proximal small intestine. Enterotoxins produced by ETEC include the LT (heat-labile) toxin and or the ST (heat-stable) toxin. LTs are similar to cholera toxin in structure and mode of action. LTs are holotoxin consisting of A subunit and B subunit. The B subunit of LTs binds to specific ganglioside receptors (GM1) on the epithelial cells of small intestine and facilitates the entry of A subunit where it activates adenylate cyclase. Stimulation of adenylate cyclase causes an increased production of cAMP, which leads to hypersecretion of water and electrolytes into the lumen and inhibition of sodium reabsorption.

Incubation period: 16-72 hours

Clinical features: Sudden onset of watery diarrhea associated with nausea, vomiting, abdominal cramping and bloating is commonly observed. This bacterium is responsible for majority of traveler's diarrhea. The disease is self-limiting and resolves in few days.

Laboratory diagnosis: The sample of feces is cultured on McConkey's agar. The ETEC stains are indistinguishable from the resident *E.coli* by biochemical tests. These strains are differentiated from nontoxigenic *E.coli* present in the bowel by a variety of in vitro immunochemical, tissue culture, or DNA hybridization tests designed to detect either the toxins or genes that encode for these toxins. With the availability of a gene probe method, foods can be analyzed directly for the presence of enterotoxigenic *E.coli* in about 3 days. LTs can be detected by Ligated rabbit ileal loop test, morphological changes in Chinese hamster ovary cells and Y1 adrenal cells, ELISA, immunodiffusion, coagulation etc.

Enterohemorrhagic *E.coli* (EHEC):

E.coli are gram negative enteric bacilli that are carried normally in the intestine of humans and animals. EHEC strains have been associated with many serogroups including O4, O26, O45, O91, O111, O145 and O157. The most serotype is O157:H7.

Incriminated food: Cattle appear to be the main source of infection; most cases being associated with the consumption of undercooked beefburgers and similar foods. This disease is often associated with ingestion of inadequately cooked hamburger meat, raw milk, cream and cheeses made from raw milk.

Pathogenesis: EHEC strains may produce one or more types of cytotoxins, which are collectively referred as Shiga-like toxins (SLT) since they are antigenically and functionally similar to Shiga toxin produced by *Shigella dysenteriae*. SLTs were previously known as verotoxin. The toxins provoke cell secretion and kill colonic epithelial cells.

Incubation period: 72-120 hours

Clinical features: Initial symptoms may be diarrhea with abdominal cramps, which may turn into grossly bloody diarrhoea in a few days. There is however, no fever.

Laboratory diagnosis: Laboratory diagnoses involve culturing the faeces on McConkey's agar or on sorbitol McConkey's agar, where they don't ferment sorbitol. Strains can then be identified by serotyping using specific antisera. SLTs can be detected by ELISA and genes coding for them can be detected by DNA hybridization techniques.

***Vibrio parahemolyticus*:**

They are straight or curved gram negative halophilic bacilli. In morphology and staining it resembles *V.cholerae* and is actively motile in liquid cultures. It is commonly found in coastal seas, where it has been isolated from marine fauna such as crabs, shrimps, fishes and molluscs.

Incriminated food: Infections are associated with consumption of uncooked or undercooked crabs, prawns, shrimps and other seafoods.

Pathogenesis: No enterotoxin has been demonstrated in the bacterium. The infection is thought to result from invasion of intestinal epithelium.

Incubation period: 7-48 hours

Clinical features: The clinical infection is characterized by a sudden onset of acute gastroenteritis. Infection may also result in diarrhoea, abdominal pain, vomiting and fever.

Laboratory diagnosis: Homogenized food may be inoculated into TCBS agar or into double strength alkaline peptone water and incubated overnight at 37°C. This bacterium is positive for Kanagawa phenomenon where isolates from human feces show hemolysis on blood agar.

***Salmonella enteritidis*:**

These are gram negative rod shaped bacteria that are classified under family enterobacteriaceae. This species does not occur normally in humans but several animals act as reservoirs.

Incriminated food: Most important sources are chicken and poultry. Chicken, duck, turkey and goose may be infected with Salmonella, which then find its way into its feces, eggs or flesh of dressed fowl. Milk and milk products including ice creams have been incriminated.

Pathogenesis: Organism penetrates and passes through the epithelial cells lining the terminal portion of the small intestine. Multiplication of bacteria in the lamina propria produces inflammatory mediators, recruits neutrophils and triggers inflammation. Release of LPS causes fever. Inflammation causes release of prostaglandins from epithelial cells. Prostaglandins cause electrolytes to flow into lumen of the intestine. Water flows into lumen in response to osmotic imbalance resulting in diarrhea.

Incubation period: 12-36 hours

Clinical features: Sudden onset of abdominal pain, nausea, vomiting and diarrhea, which may be watery, greenish and foul smelling. This may be preceded by headache and chills. Other findings include prostration,

muscular weakness and moderate fever. In most cases the symptoms resolve in 2-3 days without any complications.

Laboratory diagnosis: Homogenized food is cultured in selenite F broth and then sub-cultured on deoxycholate citrate agar. Plates are incubated at 37°C overnight and growth identified by biochemical tests and slide agglutination test.

Yersinia enterocolitica:

It is a gram negative psychrophilic rod shaped bacterium that is motile only at temperature below 30°C. *Yersinia enterocolitica* is widely distributed in environment and have been isolated frequently from soil, water and animals. The major animal reservoir for *Y. enterocolitica* strains that cause human illness is pigs, but may also found in many other animals including rodents, rabbits, sheep, cattle, horses, dogs, and cats. Serogroups that predominate in human illness are O:3, O:8, O:9, and O:5.

Incriminated food: Infection is most often acquired by eating contaminated food, especially raw or undercooked pork products. Drinking contaminated unpasteurized milk or untreated water can also transmit the infection.

Pathogenesis: This organism may survive and grow during refrigerated storage. Strains that cause human yersiniosis carry a plasmid that is associated with a number of virulence traits. Ingested bacteria adhere and invade M cells or epithelial cells. They exhibit resistance to complement and phagocytosis. They produce ST only at temperatures below 30°C. The role of ST in the disease process remains uncertain.

Incubation period: 4-7 days

Clinical features: Disease produced by *Y. enterocolitica* is a typical gastroenteritis characterized by fever, abdominal pain, and diarrhea, which is often bloody. Illness generally lasts from 1 to 2 weeks but chronic cases may persist for up to a year. Apart from gastroenteritis it may also cause pseudoappendicitis, mesenteric lymphadenitis, and terminal ileitis.

Laboratory diagnosis: Suspected food is homogenized in phosphate-buffered saline and inoculated into selenite F broth and held at 4°C for six weeks. The broth is sub-cultured at weekly intervals on DCA or *Yersinia* selective agar plates. This is termed as cold enrichment technique.

Campylobacter jejuni:

These are small, curved-spiral gram negative bacilli with polar flagella. *Campylobacter jejuni* appear in comma, S-shaped or “gull-wings/sea-gull” form. *Campylobacter* are harbored in reproductive and alimentary tracts of some animals.

Incriminated food: Transmission to humans occurs via a fecal-oral route, originating from farm animals, birds, dogs, and processed poultry, with chicken preparation comprising 50-70% of all campylobacter infections. The organism is transmitted to man in milk, meat products and contaminated water. Undercooked poultry and unpasteurized dairy are most often implicated as a source of *C. jejuni*.

Pathogenesis: As few as 500 organisms can cause enteritis. The organism is invasive but generally less so than *Shigella*. *Campylobacter* produces adenylate cyclase-activating toxins same as of *E. coli* LT and cholera.

Incubation period: Ranges from 2 to 11 days.

Clinical features: Patients present with abdominal pain and cramps, diarrhea, malaise, headache, and usually fever. Typically the diarrhoea is watery, but in severe cases bloody diarrhea may occur. Diarrhea may last 2-7 days and the organism may be shed in the patients stool for up to 2 months. Bacteremia is observed in a small minority of cases. The disease is usually self-limiting.

Laboratory diagnosis: The feces may be inoculated in enrichment medium or on selective media such as Campy BAP or Skirrow's medium. The plates are incubated in microaerophilic conditions at 42°C for 2-5 days.

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