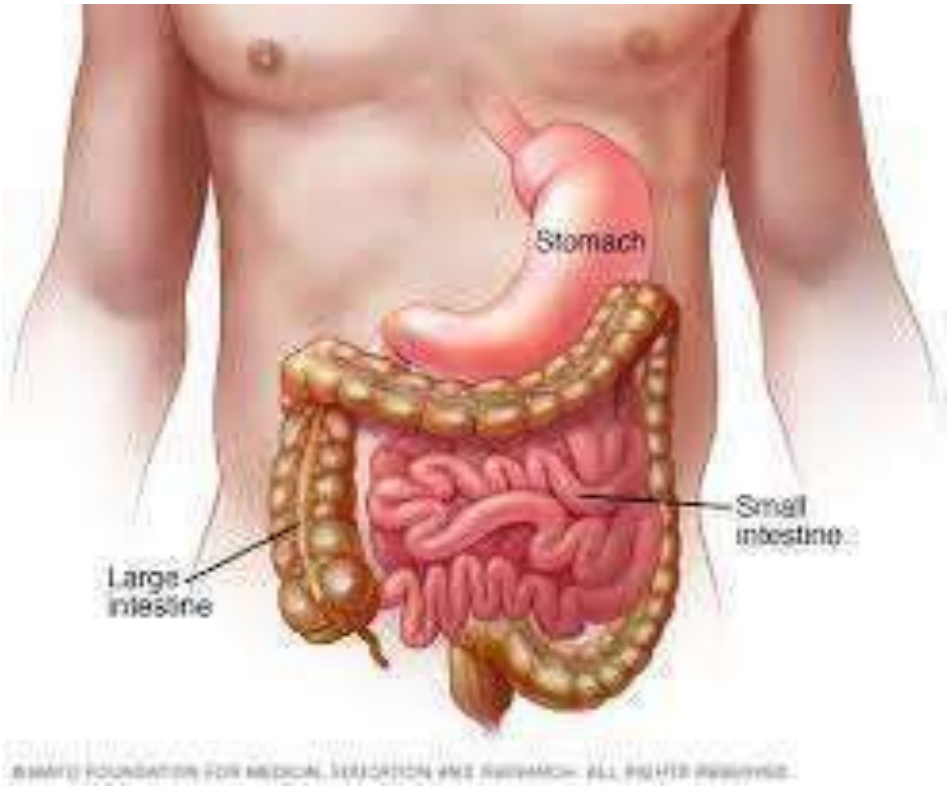
Gastroenteritis



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DEFINITION-GASTROENTERITIS

- Gastroenteritis is inflammation of the gastrointestinal tract, involving the stomach, intestines, or both; usually resulting in diarrhoea, abdominal cramps, nausea and possibly vomiting.
- Gastroenteritis is frequently termed as"stomach flu" or "gastric flu“.

ETIOLOGY

BACTERIA

- *Vibrio cholerae,*
- *Escherichia coli,*
- *Salmonella,*
- *Shigella,*
- *Campylobacter jejuni,*
- *Yersinia enterocolitica*
- *Staphylococcus aureus*
- *Bacillus cereus*
- *Clostridium perfringes*

VIRUS

- *rotaviruses,*
- *noroviruses,*
- *astrovirus,*
- *enteric adenovirus*

ETIOLOGY

PARASITES

- *Giardia,*
- *Entamoeba,*
- *Strongyloides,* and
- *Cryptosporidium*

SIGNS AND SYMPTOMS

The condition is usually of acute onset, normally lasting 1–6 days, and is self- limiting.

- Nausea and vomiting
- Diarrhoea
- Anorexia
- Fever
- Headaches
- Abnormal flatulence
- Abdominal pain
- Abdominal cramps
- Melena
- Fainting and Weakness
- Heartburn

DIAGNOSIS

- Clinical evaluation (MHx, Physical examination)

Findings suggestive of gastroenteritis include

- copious, watery diarrhoea
- ingestion of potentially contaminated food (particularly during a known outbreak)
- known GI irritant;
- recent travel;
- contact with similarly ill people.

DIAGNOSIS(CONT)

Stool testing in select cases

- If a rectal examination shows occult blood or if watery diarrhoea persists > 48 h, stool examination (fecal WBCs, ova, parasites) and culture are indicated.
- However, for the diagnosis of giardiasis or cryptosporidiosis, stool antigen detection using an enzyme immunoassay has a higher sensitivity.
- Rotavirus and enteric adenovirus infections can be diagnosed using commercially available rapid assays that detect viral antigen in the stool, but these are usually done only to document an outbreak.
- All patients with grossly bloody diarrhoea should be tested for E. coli O157:H7, as should patients with non bloody diarrhoea during a known outbreak.

DIAGNOSIS(CONT)

GENERAL TESTS

- Serum electrolytes
- Blood Urea Nitrogen (BUN)
- Creatinine should be obtained to evaluate hydration and acid-base status in patients who appear seriously ill.
- Complete Blood Count (CBC) is nonspecific, although eosinophilia may indicate parasitic infection.

PATHOPHYSIOLOGY

Bacteria can cause diarrhoea in three different ways:

1. Mucosal adherence
2. Mucosal invasion
3. Toxin production

Diarrheal illnesses may be classified as follows:

1. Osmotic, due to an increase in the osmotic load presented to the intestinal lumen, either through excessive intake or diminished absorption
2. Inflammatory (or mucosal), when the mucosal lining of the intestine is inflamed
3. Secretory, when increased secretory activity occurs
4. Motile, caused by intestinal motility disorders

PATHOPHYSIOLOGY

1. Mucosal adherence

- Most bacteria causing diarrhoea must first adhere to specific receptors on the gut mucosa.
- A number of different molecular adhesion mechanisms have been elaborated; for example, adhesions at the tip of the pili or fimbriae which protrude from the bacterial surface aid adhesion.
- For some pathogens this is merely the prelude to invasion or toxin production but others such as enteropathogenic Escherichia coli (EPEC) cause attachment-effacement mucosal lesions on electron microscopy (EM) and produce a secretory diarrhoea directly as a result of adherence.
- Adhere in an aggregative pattern with the bacteria clumping on the cell surface and its toxin causes persistent diarrhoea.
- Diffusely adhering E. coli (DAEC) adheres in a uniform manner and may also cause diarrhoea seen in children.

PATHOPHYSIOLOGY

Mucosal invasion:

- Invasive pathogens such as Shigella sp., enteroinvasive E. coli (EIEC) and Campylobacter sp. penetrate into the intestinal mucosa.
- Initial entry into the mucosal cells is facilitated by the production of ‘invasins’, which disrupt the host cell cytoskeleton.
- Subsequent destruction of the epithelial cells allows further bacterial entry, which also causes the typical symptoms of dysentery: low-volume bloody diarrhoea, with abdominal pain.

PATHOPHYSIOLOGY

Toxin production :

Gastroenteritis can be caused by different types of bacterial toxins:

- Enterotoxins, produced by the bacteria adhering to the intestinal epithelium, induce excessive fluid secretion into the bowel lumen, leading to watery diarrhoea, without physically damaging the mucosa, e.g. cholera, enterotoxigenic E. coli (ETEC).
- Some enterotoxins preformed in the food primarily cause vomiting, e.g. Staph. aureus and Bacillus cereus.
- A typical example of this is 'fried rice poisoning', in which B. cereus toxin is present in cooked rice left standing overnight at room temperature.
- Cytotoxins damage the intestinal mucosa and, in some cases, vascular endothelium as well (e.g. E. coli)

PATHOPHYSIOLOGY

Viral pathophysiology

- Viral spread from person to person occurs by fecal-oral transmission of contaminated food and water.
- Some viruses, like noroviruses, may be transmitted by an airborne route.
- Clinical manifestations are related to intestinal infection, Rotaviruses attach and enter mature enterocytes at the tips of small intestinal villi.
- They cause structural changes to the small bowel mucosa, including villus shortening and mononuclear inflammatory infiltrate in the lamina propria.

PATHOPHYSIOLOGY

- Rotavirus infections induce maldigestion of carbohydrates, and their accumulation in the intestinal lumen, as well as a malabsorption of nutrients and a concomitant inhibition of water reabsorption, can lead to a malabsorption component of diarrhoea.

- Rotavirus secretes an enterotoxin, NSP4, which leads to a Ca2+ -dependent Cl- secretory mechanism. Morphologic abnormalities can be minimal, and studies demonstrate that rotavirus can be released from infected epithelial cells without destroying them.

PATHOPHYSIOLOGY

Pathophysiology of Parasite

- Certain intestinal parasites, notably Giardia intestinalis adhere to or invade the intestinal mucosa, causing nausea, vomiting, diarrhoea, and general malaise.
- The infection can become chronic and cause a malabsorption syndrome. It is usually acquired via person-to-person transmission (often in day care centers) or from contaminated water.
- Cryptosporidium parvum causes watery diarrhoea sometimes accompanied by abdominal cramps, nausea, and vomiting. In healthy people, the illness is self-limited, lasting about 2 wk.
- In immunocompromised patients, illness may be severe, causing substantial electrolyte and fluid loss. Cryptosporidium is usually acquired through contaminated water.
- Entamoeba histolytica (amebiasis) is a common cause of subacute bloody

Gastroenteritis: *Pathogenesis and clinical findings*

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Virus (70%) → *Rotavirus, norovirus, adenovirus, astroviruses, enteroviruses*

Bacteria (10-20%) → *Campylobacter jejuni, Salmonella spp, Escherichia coli, Shigella, Yersinia, Vibrio cholerae*

Other (<10%) → *Cryptosporidium, Giardia lamblia, Entamoeba histolytica, Strongyloides*

Pathogenic Infiltration of Gastrointestinal Tract

Notes:
- Peak age for gastroenteritis is from 6 months to 2 years.
- Spread of infection occurs by respiratory and fecal-oral routes.

Noxious stimuli and GI tract inflammation → Stimulation of visceral nerve afferents → **Abdominal Pain**

Pathogen toxins stimulate enteric chloride secretion

Pathogenic interaction with the enteric nervous system

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Pathogen toxins stimulate enteric chloride secretion + Pathogenic interaction with the enteric nervous system → ↑ GI fluid secretion → **Diarrhea**

Alteration of brush border activity and/or structure → Impaired absorption of substances in small intestine → Osmotically-active substances enter large bowel → Capacity for water reabsorption in the large Intestine is overwhelmed → **Diarrhea**

Alteration of brush border activity and/or structure * → Release of 5HT from enterochromaffin cells → Activation of chemoreceptor trigger zone (area postrema) → Stimulation of vomiting centre (lateral reticular formation) → **Nausea/Vomiting**

Notes:
- Diarrhea = increased frequency and decreased consistency of stool, typically the passage of ≥3 loose/watery stools per day.
- Exact mechanisms for each clinical finding are likely multifactorial and may vary depending on the pathogen involved.

Treatment -Rehydration.

- The treatment of cholera and other dehydrating diarrheal diseases was revolutionized by the promotion of oral **rehydration solutions.**
- The efficacy of which depends on the fact *that* **glucose-facilitated absorption of sodium and water** in the small intestine remains intact in the presence of cholera toxin.
- **The World Health Organization recommends** a solution containing 3.5 g sodium chloride, 2.5 g sodium bicarbonate, 1.5 g potassium chloride, and 20 g glucose (or 40 g sucrose) per liter of water.
- Oral rehydration solutions containing **rice or cereal as the carbohydrate source** may be even more effective than glucose-based solutions, and the addition of L-histidine may reduce the frequency and volume of stool output.
- Patients who are severely dehydrated or in whom vomiting precludes the use of oral therapy should receive IV **solutions such as Ringer's lactate.**
- Although most secretory forms **of traveler's diarrhea—usually due to enterotoxigenic and enteroaggregative *E. coli*—**can be treated effectively with rehydration, bismuth subsalicylate, or antiperistaltic agents, antimicrobial agents can shorten the duration of illness from 3–4 days to 24–36 h.

Pathogen	First-Line Agents	Alternative Agents
Enterotoxigenic (cholera-like) diarrhea		
Vibrio cholerae O1 or O139	Doxycycline 300 mg oral single dose; tetracycline 500 mg orally four times daily × 3 days; or trimethoprim-sulfamethoxazole DS tablet twice daily × 3 days; norfloxacin 400 mg orally twice daily × 3 days; or ciprofloxacin 500 mg orally twice daily × 3 days or 1 g orally single dose	Chloramphenicol 50 mg/kg IV every 6 hours, erythromycin 250–500 mg orally every 6–8 hours, and furazolidone
Enterotoxigenic *Escherichia coli*	Norfloxacin 400 mg or ciprofloxacin 500 mg orally twice daily × 3 days	Trimethoprim-sulfamethoxazole DS tablet every 12 hours
Clostridium difficile	Metronidazole 250 mg four times daily to 500 mg three times daily × 10 days	Vancomycin 125 mg orally four times daily × 10 days; bacitracin 20,000–25,000 units four times daily × 7–10 days
Invasive (dysentery-like) diarrhea		
Shigella species[a]	Trimethoprim-sulfamethoxazole DS twice daily × 3–5 days	Ofloxacin 300 mg, norfloxacin 400 mg, or ciprofloxacin 500 mg twice daily × 3 days, or nalidixic acid 1 g/day × 5 days; azithromycin 500 mg orally × 1, then 250 mg orally daily × 4 days
Salmonella		
Nontyphoidal[a]	Trimethoprim-sulfamethoxazole DS twice daily; ofloxacin 300 mg, norfloxacin 400 mg, or ciprofloxacin 500 mg twice daily × 5 days; or ceftriaxone 2 g IV daily or cefotaxime 2 g IV three times daily × 5 days	Azithromycin 1,000 mg orally × 1 day, followed by 500 mg orally once daily × 6 days
Enteric fever	Ciprofloxacin 500 mg orally twice daily × 3–14 days (ofloxacin and pefloxacin equally efficacious)	Azithromycin 1,000 mg orally × 1 day, followed by 500 mg daily × 5 days; or cefixime, cefotaxime, and cefuroxime; or chloramphenicol 500 mg four times daily orally or IV × 14 days
Campylobacter[a]	Erythromycin 500 mg orally twice daily × 5 days; azithromycin 1,000 mg orally × 1 day, followed by 500 mg daily or clarithromycin 500 mg orally twice daily	Ciprofloxacin 500 mg or norfloxacin 400 mg orally twice daily × 5 days
Yersinia species[a]	A combination therapy with doxycycline, aminoglycosides, trimethoprim-sulfamethoxazole, or fluoroquinolones	
Traveler's diarrhea		
Prophylaxis[a]	Norfloxacin 400 mg or ciprofloxacin 500 mg orally daily (in Asia, Africa, and South America); trimethoprim-sulfamethoxazole DS tablet orally daily (in Mexico)	Rifaximin 200 mg one to three times daily × 2 weeks
Treatment	Norfloxacin 400 mg or ciprofloxacin 500 mg orally twice daily × 3 days, or trimethoprim-sulfamethoxazole DS tablet orally twice daily × 3 days (in Mexico), or azithromycin 500 mg orally once daily × 3 days (only in areas of high prevalence of quinolone-resistant *Campylobacter* species,	Rifaximin 200 mg three times a day or 400 mg twice a day × 3 days

Patient education

- Patients should be **educated on** the importance and proper methods **of oral rehydration** and early appropriate feeding.
- All patients, especially the parents of infants and young children, must be extensively **educated about the signs and symptoms of dehydration.**
- Patients with food-borne exposures should be educated on deterrence.
- **Immunocompromised patients** and individuals with liver disease should be educated not to consume raw shellfish, especially oysters.
- Travelers to underdeveloped areas should be made aware of proper avoidance measures, **appropriate treatment, and current endemic illnesses.**
- Take enteric precautions to avoid spread to family members, especially by **washing hands before eating** and after each stool or diaper change.
- Avoid cross-contamination of foods during preparation (eg, cutting boards).
- Avoid raw or undercooked eggs or poultry.
- Consume acidic foods, such as citrus.
- Consume dry foods, such as bread and nuts.
- Drink carbonated beverages.


Thank
You