Lec 5 : Bacterial Infections of the Gastrointestinal Tract

B341 **By :Dr. Ruaa Abdallah**

Bacterial Infections of the Gastrointestinal Tract

Overview (general concepts)

Bacterial GI disease results from infection (organisms colonize the gut and produce toxins in vivo) or intoxication (preformed toxins in food). Toxins and virulence factors damage intestinal epithelial cells (often the colon), producing: watery diarrhea, abdominal cramps, nausea, vomiting, and sometimes dysentery (bloody, mucoid stool). Most illnesses are self-limiting; the principal immediate danger is dehydration — oral rehydration (electrolytes) is essential. Diagnosis usually relies on stool testing (microscopy, culture, antigen/PCR, toxin assays) and targeted lab media or molecular tests for specific pathogens.

1- Staphylococcus aureus (staphylococcal food poisoning)

• Agent & nature: Gram-positive coccus that can produce ≥21 enterotoxins/enterotoxin-like proteins.

• Mechanism: Food intoxication — toxins produced in food are heat-stable and acid-resistant (survive boiling and stomach acid). Some act as superantigens and provoke strong immune responses.

• Common foods: dairy products, cooked meats, salads (hand contamination and poor hygiene common). Risk when food is held at temperatures that allow growth (below ~60 °C / 140 °F).

• Onset & course: Rapid onset (typically 1–6 hours after ingestion); symptoms (nausea, vomiting, cramps, diarrhea) usually resolve within 24–48 hours.

• Diagnosis: Detect toxin in food, vomitus, or stool (ELISA, immunoassays). Bacterial culture may be absent.

• Treatment: Supportive care and rehydration; hospital care if severe dehydration.

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2- Shigella (shigellosis, bacillary dysentery)

• Species: S. dysenteriae (produces Shiga toxin), S. flexneri, S. sonnei, S. boydii.

• Transmission: Fecal–oral; very low infectious dose (person-to-person spread common).

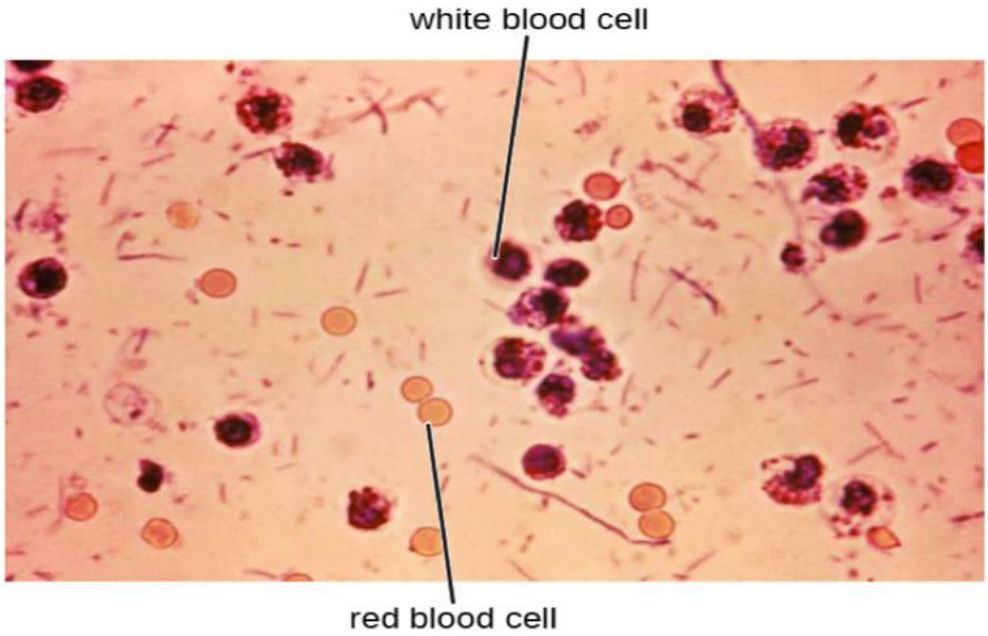
• Pathogenesis: Invades intestinal epithelial cells, escapes phagosomes, replicates intracellularly and spreads cell-to-cell; Shiga toxin (in some strains) inhibits ribosomal function → endothelial damage.

• Clinical: Fever, severe abdominal cramps, watery diarrhea that may progress to bloody/mucoid stools; WBCs and RBCs often seen in stool.

• Complications: Hemolytic-uremic syndrome (HUS) with certain strains, reactive arthritis, post-infectious IBS.

• Diagnosis: Stool culture, immunoassays, PCR; process stool promptly.

• Treatment: Mostly supportive; severe cases may need antibiotics (ciprofloxacin, azithromycin) — antibiotic resistance is an increasing concern.



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3-Salmonella (salmonellosis) & Typhoid fever

Non-typhoidal Salmonella (salmonellosis)

• Agent: Salmonella enterica (many serotypes, e.g., Enteritidis) and S. bongori.

• Reservoirs: Poultry, eggs, some animals (reptiles), contaminated foods.

• Pathogenesis: Invades via M cells in Peyer's patches, triggers inflammation and fluid secretion, can survive in macrophage phagosomes and sometimes disseminate. Some strains produce enterotoxins.

• Clinical: Fever, nausea, vomiting, abdominal cramps, diarrhea; symptoms last days to a week.

• Diagnosis: Culture and serotyping; DNA fingerprinting for outbreak strains; reportable to public health authorities (CDC).

• Treatment: Usually self-limited — rehydration. Antibiotics reserved for severe cases or high-risk patients (fluoroquinolones, third-gen cephalosporins, ampicillin). Resistance is a major issue.

Typhoid fever (S. typhi / Paratyphi)

• Disease: Systemic infection; can be severe (untreated mortality ≈10%).

• Pathogenesis: S. typhi survives in macrophages and disseminates to liver, spleen, gallbladder; Vi capsule antigen and chimeric A2B5 toxin contribute to virulence.

• Carriers: Asymptomatic chronic carriers (gallbladder colonization) can shed bacteria in feces (historical example: "Typhoid Mary").

• Clinical: High fever, headache, malaise, abdominal pain, possibly rash; intestinal ulceration/perforation possible.

• Diagnosis: Culture from blood, stool, urine, bone marrow; serology and PCR adjuncts.

• Treatment & prevention: Fluoroquinolones, ceftriaxone, azithromycin; vaccination recommended for travelers to endemic areas.

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4- Escherichia coli (pathogenic groups)

There are multiple pathogenic E. coli groups; four major food/waterborne groups:

1-ETEC (Enterotoxigenic E. coli)

• Virulence: Heat-stable (ST) and/or heat-labile (LT) enterotoxins (LT similar to cholera toxin); colonization factors (adhesins).

• Disease: Traveler's diarrhea — watery diarrhea, cramps, low fever; usually self-limited.

• Dx/treatment: Culture/PCR; rehydration; antibiotics (e.g., fluoroquinolones, doxycycline, rifaximin, TMP-SMX) may shorten duration but resistance occurs.

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2-EIEC (Enteroinvasive E. coli)

• Virulence: inv plasmid genes enabling epithelial invasion (Shigella-like).

• Disease: Dysentery/inflammatory colitis with watery/bloody diarrhea; usually self-limited.

• Treatment: Supportive; antibiotics not routinely recommended.

3-EPEC (Enteropathogenic E. coli)

• Virulence: LEE pathogenicity island → injection of Tir protein, Intimin receptor → pedestal formation and microvillus effacement.

• Disease: Severe non-bloody diarrhea, vomiting, dehydration — significant in infants.

• Dx/treatment: PCR/culture for LEE; supportive; antibiotics sometimes used.

4-EHEC (Enterohemorrhagic E. coli, e.g., O157:H7)

• Virulence: Shiga-like (verotoxin) acquired by phage; LEE for pedestal formation.

• Disease: Bloody diarrhea, severe abdominal cramps, often no fever; can progress to HUS (esp. children).

• Dx: MacConkey sorbitol agar (O157:H7 typically does not ferment sorbitol), PCR for toxin genes.

• Treatment: Antibiotics are not recommended (may increase HUS risk from toxin release); supportive care and close monitoring.

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Group	Virulence Factors and Genes	Signs and Symptoms	Diagnostic Tests	Treatment
Enterotoxigenic *E. coli* (ETEC)	Heat stable enterotoxin similar to cholera toxin	Relatively mild, watery diarrhea	Culturing, PCR	Self-limiting; if needed, fluoroquinolones, doxycycline, rifaximin, TMP/SMZ; antibiotic resistance is a problem
Enteroinvasive *E. coli* (EIEC)	*Inv* (invasive plasmid) genes	Relatively mild, watery diarrhea; dysentery or inflammatory colitis may occur	Culturing, PCR; testing for *inv* gene; additional assays to distinguish from *Shigella*	Supportive therapy only; antibiotics not recommended
Enteropathogenic *E. coli* (EPEC)	Locus of enterocyte effacement (LEE) pathogenicity island	Severe fever, vomiting, nonbloody diarrhea, dehydration; potentially fatal	Culturing, PCR; detection of LEE lacking Shiga-like toxin genes	Self-limiting; if needed, fluoroquinolones, doxycycline, rifaximin (TMP/SMZ); antibiotic resistance is a problem
Enterohemorrhagic *E. coli* (EHEC)	Verotoxin	May be mild or very severe; bloody diarrhea; may result in HUS	Culturing; plate on MacConkey agar with sorbitol agar as it does not ferment sorbitol; PCR detection of LEE containing Shiga-like toxin genes	Antibiotics are not recommended due to the risk of HUS

5- Vibrio species (cholera and others)

Vibrio cholerae (O1 mainly)

• Mechanism: Motile curved gram-negative rod; cholera toxin (A-B toxin) → persistent activation of adenylate cyclase → ↑cAMP → massive chloride and water secretion → “rice-water” stool and rapid dehydration.

• Transmission: Contaminated water/food; associated with poor sanitation or disasters.

• Diagnosis: Stool culture, oxidase positive organism; TCBS agar (selective).

• Treatment: Aggressive rehydration (oral/IV); antibiotics (tetracyclines, azithromycin, fluoroquinolones) in severe disease. Public health measures and water sanitation are critical.

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Other vibrios

• V. parahemolyticus: raw/undercooked seafood → gastroenteritis (watery diarrhea, cramps); hemolysin production.

• V. vulnificus: warm seawater, raw oysters or wound exposure → can cause septicemia (high fatality in liver disease) or severe wound infections; treat aggressively (doxycycline + cephalosporin/fluoroquinolone).

Campylobacter jejuni

• Agent: Gram-negative curved/spiral rod; microaerophilic; grows best at ~42 °C on selective media.

• Sources: Contaminated poultry, unpasteurized milk, contaminated water.

• Virulence: Hemolysins and cytolethal distending toxin (CDT; DNase activity) that damages host DNA.

• Clinical: Fever, abdominal cramps, diarrhea (sometimes bloody).

• Complications: Guillain-Barré syndrome (autoimmune neuropathy) and rarely HUS.

• Diagnosis: Culture on Campy selective agars under microaerophilic conditions; prolonged incubation.

• Treatment: Usually self-limiting; severe cases treated with erythromycin or ciprofloxacin.

Helicobacter pylori (peptic ulcers)

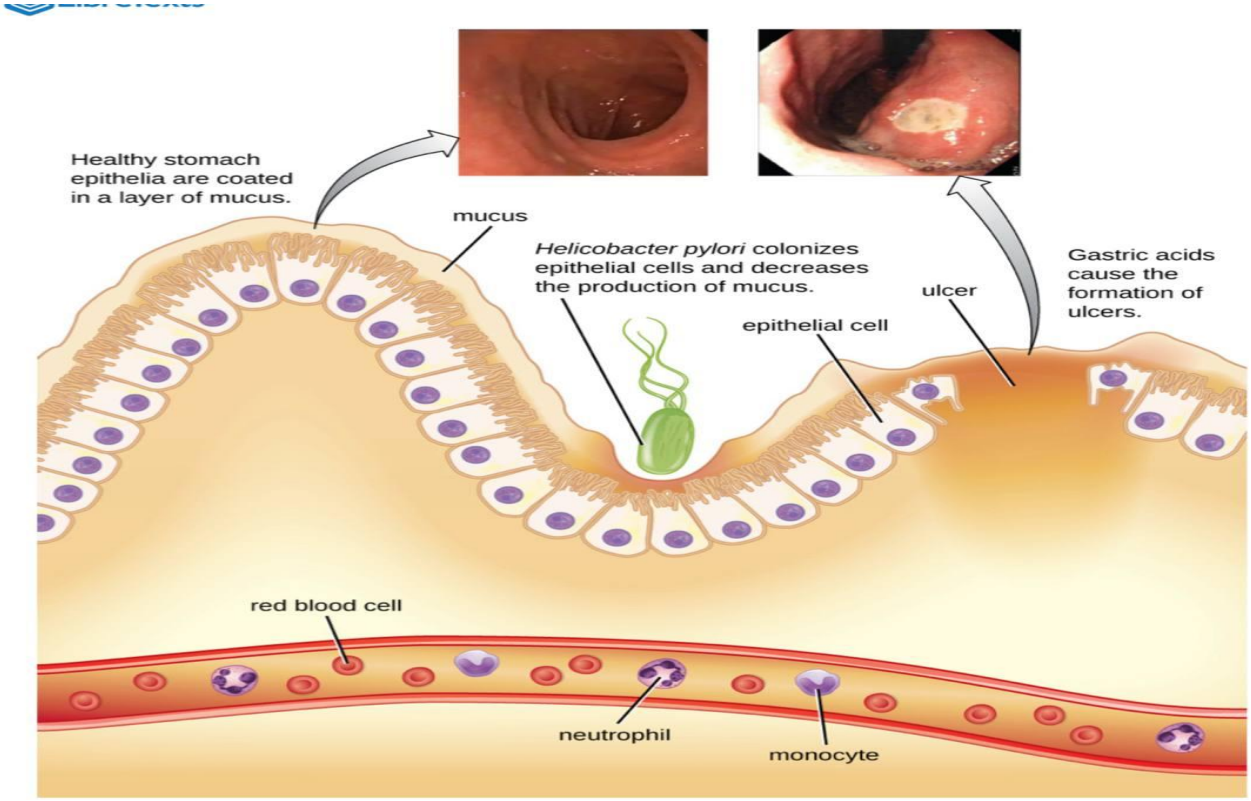
• Agent & adaptation: Gram-negative curved rod able to survive stomach acidity by producing urease (makes ammonia).

• Pathogenesis: Adhesion via pili, urease activity neutralizes acid locally, VacA exotoxin (vacuolating cytotoxin) damages epithelial cells; infection causes gastritis and peptic ulcers and is associated with increased gastric cancer risk.

• Diagnosis: Urea breath test (radiolabeled urea → CO_2 if urease present), stool antigen, serology, endoscopic biopsy with culture/histology.

• Treatment: "Triple therapy" regimens — examples from the chapter: OAC (omeprazole + amoxicillin + clarithromycin for 10 d), BMT (bismuth subsalicylate + metronidazole + tetracycline for 14 d), LAC (lansoprazole + amoxicillin + clarithromycin for 10–14 d). Acid suppression meds are adjuncts.

• Note: Eradication may have trade-offs (some evidence H. pylori may protect against some esophageal diseases).



6- Clostridium perfringens

• Agent: Gram-positive anaerobic, spore-forming rod.

• Disease: Foodborne enteritis (type A strains produce enterotoxin); commonly associated with improperly handled/cooked meats and gravies. Sedentary food left at room temp allows spore germination/toxin production.

• Clinical: Cramps and diarrhea; usually mild and self-limited; rare severe necrotizing forms ("pig-bel") with high mortality.

• Diagnosis: Detect enterotoxin or toxin gene (PCR/ELISA) in stool/food.

• Treatment: Supportive (rehydration); correct food handling prevents disease.

7- Clostridioides (Clostridium) difficile

• Agent: Gram-positive anaerobe, spore-forming; part of normal flora in some.

• Risk factors: Antibiotic exposure (disrupts normal flora), hospitalization, PPI use, immunosuppression.

• Toxins & pathology: Toxin A (TcdA) and Toxin B (TcdB) inactivate small GTPases → actin condensation, cell rounding, death → pseudomembranous colitis with fibrinous pseudomembrane and severe inflammation.

• Clinical: Watery diarrhea, abdominal pain, fever, leukocytosis; severe disease → toxic megacolon, perforation, sepsis.

• Diagnosis: Detect toxin genes by NAAT/PCR (preferred) or toxin assays (ELISA); consider clinical history.

• Treatment: First step — stop offending antibiotic if possible. Chapter notes metronidazole historically preferred; vancomycin for certain patients or refractory cases. Fecal microbiota transplantation (FMT) is a highly effective option for recurrent disease (>90% success reported).

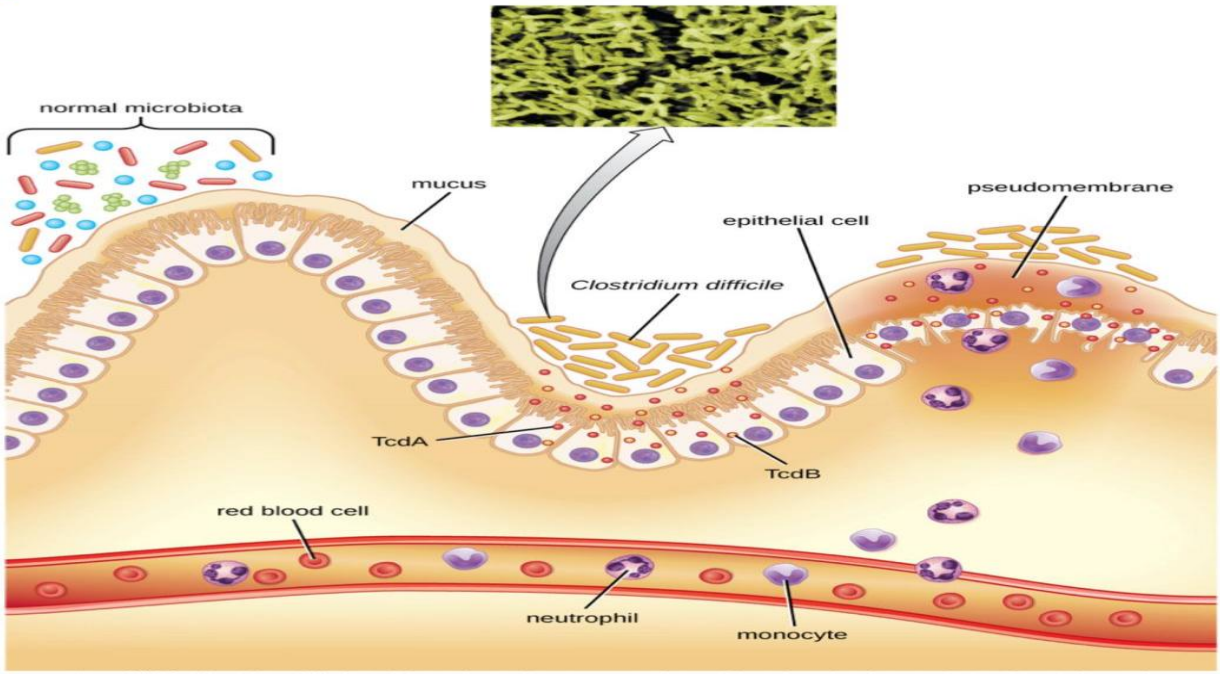


Figure 24.3.6: Clostridium difficile is able to colonize the mucous membrane of the colon when the normal microbiota is disrupted. The toxins TcdA and TcdB trigger an immune response, with neutrophils and monocytes migrating from the bloodstream to the site

8- Bacillus cereus

• Agent: Gram-positive spore-forming rod; spores survive cooking.

• Syndromes: Emetic form (preformed toxin in rice → vomiting within hours) and diarrheal form (enterotoxin produced in gut → diarrhea).

• Diagnosis/treatment: Isolate organism from stool/vomit/food; treatment supportive and rehydration.

9- Yersinia (Y. enterocolitica, Y. pseudotuberculosis)

• Agent: Gram-negative rods (Yersinia genus).

• Transmission: Fecal–oral; contaminated food/water; sometimes zoonotic reservoirs.

• Clinical: Usually mild gastroenteritis; infants may develop severe disease with dysentery. In adults, may cause or be associated with reactive arthritis, thyroid disorders, endocarditis, glomerulonephritis, eye inflammation, erythema nodosum.

• Diagnosis: Stool culture; other tissue/fluid samples if systemic.

• Treatment: Supportive for GI disease; antibiotics (fluoroquinolones, aminoglycosides, doxycycline, TMP-SMX) for bacteremia/systemic infections.

Laboratory & diagnostic

• Use stool culture for many pathogens; special media help identification (MacConkey, sorbitol-MacConkey for EHEC O157:H7, TCBS for Vibrio, Campy selective media at 42 °C).

• Toxin detection (ELISA/PCR) for S. aureus, C. perfringens, B. cereus, C. difficile, EHEC (Shiga toxin).

• Molecular methods (PCR/NAAT) increasingly used for rapid and specific diagnosis.

• Presence of WBCs or blood in stool suggests invasive organisms (Shigella, EIEC, Campylobacter, Salmonella).

Treatment & prevention

• Mainstay: Rehydration (oral or IV) and electrolyte replacement.

• Antibiotics: Indicated selectively — severe systemic disease, immunocompromised patients, or specific pathogens. Avoid or use cautiously where antibiotics may worsen outcomes (e.g., EHEC — HUS risk; antibiotic use predisposes to C. difficile).

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Bacterial Infections of the GI Tract					
Disease	**Pathogen**	**Signs and Symptoms**	**Transmission**	**Diagnostic Tests**	**Antimicrobial Drugs**
Bacillus cereus infection	*Bacillus cereus*	Nausea, pain, abdominal cramps, diarrhea, or vomiting	Ingestion of contaminated rice or meat, even after cooking	Testing stool sample, vomitus, or uneaten food for presence of bacteria	None
Campylobacter jejuni gastroenteritis	*Campylobacter jejuni*	Fever, diarrhea, cramps, vomiting, and sometimes dysentery; sometimes more severe organ or autoimmune effects	Ingestion of unpasteurized milk, under-cooked chicken, or contaminated water	Culture on selective medium with elevated temperature and low oxygen concentration	Generally none; erythromycin or ciprofloxacin if necessary
Cholera	*Vibrio cholerae*	Severe diarrhea and fluid loss, potentially leading to shock, renal failure, and death	Ingestion of contaminated water or food	Culture on selective medium (TCBS agar); distinguished as oxidase positive with fermentative metabolisms	Generally none; tetracyclines, azithromycin, others if necessary
Clostridium difficile infection	*Clostridium difficile*	Pseudomem-branous colitis, watery diarrhea, fever, abdominal pain, loss of appe-tite, dehydration; in severe cases, perforation of the colon, septicemia, shock, and death	Overgrowth of *C. difficile* in the normal microbiota due to antibiotic use; hospital-acquired infections in immunocompro-mised patients	Detection of toxin in stool, nucleic acid amplification tests (e.g., PCR)	Discontinuation of previous antibiotic treatment; metronidazole or vancomycin
Clostridium perfringens gastroenteritis	*Clostridium perfringens* (especially type A)	Mild cramps and diarrhea in most cases; in rare cases, hemor-rhaging, vomiting, intestinal gangrene, and death	Ingestion of undercooked meats containing *C. perfringens* endospores	Detection of toxin or bacteria in stool or uneaten food	None
E. coli infection	ETEC, EPEC, EIEC, EHEC	Watery diarrhea, dysentery, cramps, malaise, fever, chills, dehydration; in EHEC, possible severe compli-cations such as hematolytic uremic syndrome	Ingestion of contaminated food or water	Tissue culture, immunochemi-cal assays, PCR, gene probes	Not recommended for EIEC and EHEC; fluoroquinolones, doxycycline, rifaximin, and TMP/SMZ possible for ETEC and EPEC

Figure 24.3.7: Bacterial infections of the GI tract.

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Bacterial Infections of the GI Tract (continued)					
Disease	**Pathogen**	**Signs and Symptoms**	**Transmission**	**Diagnostic Tests**	**Antimicrobial Drugs**
Peptic ulcers	*Helicobacter pylori*	Nausea, bloating, burping, lack of appetite, weight loss, perforation of stomach, blood in stools	Normal flora, can also be acquired via saliva; fecal-oral route via contaminated food and water	Breath test, detection of antibodies in blood, detection of bacteria in stool sample or stomach biopsy	Amoxicillin, clarithromycin metronidazole, tetracycline, lansoprazole; antacids may also be given in combination with antibiotics
Salmonellosis	*Salmonella enterica*, serotype Enteritides	Fever, nausea, vomiting, abdominal cramps, headache, diarrhea; can be fatal in infants	Ingestion of contaminated food, handling of eggshells or contaminated animals	Culturing, serotyping and DNA fingerprinting	Not generally recommended; fluoroquinolones, ampicillin, others for immunocompromised patients
Shigella dysentery	*Shigella dysenteriae*, *S. flexneri*, *S. boydii*, and *S. sonnei*	Abdominal cramps, fever, diarrhea, dysentery; possible complications: reactive arthritis and hemolytic uremic syndrome	Fecal-oral route via contaminated food and water	Testing of stool samples for presence of blood and leukocytes; culturing, PCR, immunoassay for *S. dysenteriae*	Ciprofloxacin, azithromycin
Staphylococcal food poisoning	*Staphylococcus aureus*	Rapid-onset nausea, diarrhea, vomiting lasting 24–48 hours; possible dehydration and change in blood pressure and heart rate	Ingestion of raw or undercooked meat or dairy products contaminated with staphylococcal enterotoxins	ELISA to detect enterotoxins in uneaten food, stool, or vomitus	None
Typhoid fever	*S. entrica*, subtypes Typhi or Paratyphi	Aches, headaches, nausea, lethargy, diarrhea or constipation, possible rash; lethal perforation of intestine can occur	Fecal-oral route; may be spread by asymptomatic carriers	Culture of blood, stool, or bone marrow, serologic tests; PCR tests when available	Fluoroquinolones, ceftriaxone, azithromycin; preventive vaccine available
Yersinia infection	*Yersinia enterocolitica*, *Y. pseudotuberculosis*	Generally mild diarrhea and abdominal cramps; in some cases, bacteremia can occur, leading to severe complications	Fecal-oral route, typically via contaminated food or water	Testing stool samples, tissues, body fluids	Generally none; fluoroquinolones, aminoglycosides, others for systemic infections

Figure 24.3.8: Bacterial infections of the GI tract (continued).