GI micro 3 summary

Enterobacteriaceae: G(-) rods; Escherichia, shigella, salmonella, yersinia,...

Characteristics:

1. Motile with peritrichous flagella (except shigella, yersinia and klebsiella)
2. Facultative anaerobes
3. Ferment glucose with gas production
4. Oxidase negative, catalase positive, reduce nitrate to nitrite.
5. Grown on peptone, meat extract media and MacConkey agar.

Antigenic structure:

1. heat-stable O antigen (lipopolysaccharide) / common enterobacterial core antigen → endotoxin, agglutination with IgM.
2. Heat-labile K antigen (capsular) → resistant to serum killing but not virulent, except that of salmonella typhi (vi antigen).
3. H antigen (flagellar) –motile Enterobacteriaceae. Agglutinate with IgG.
4. Bacteriocins: peptide pore forming toxins to inhibit growth of other bacteria.

   Bacteriocin of E-coli is colicin.

**E-coli: most common G(-) rods cultured and one of the commonest bacteria to cause infections (commonest in UTI).

Oxidase negative and lactose fermenters.

Use MacConkey agar or eosin methylene blue agar (EMB) to see lactose fermentation.

On MacConkey: pink

On EMB: black nucleated colonies (green sheen specifically for E-coli).

Cause disease by:

1. Gain virulence factors through plasmids or phages, e.g. toxins
2. Opportunistic infection (immunocompromised)

Access to sterile sites (blood, urine,..)

E-coli diarrheal diseases:

1. Enteropathogenic E-coli (EPEC):

Major cause of infantile diarrhea associated with outbreaks in nurseries. (bottle-fed babies).

Pathogenesis: (attachment and effacement)

1. Bundle forming pilus for attachment to mucosa (adherence factor).
2. Enterocyte effacement pathogenicity island for tight adherence.
3. Effacement (loss of microvilli and degeneration of epithelia).

transmission: patient to patient (feco-oral)

Unusual in adults (high inoculum needed).

Signs &symptoms: severe, watery diarrhea, vomiting and fever

Rx: Self-limited but can be chronic, antibiotics shorten duration of diarrhea

2. Enterotoxigenic E-coli (ETEC): traveler's diarrhea

Important cause of infant diarrhea in developing countries.

Pathogenesis:

1. ETEC colonizing factors (antigens), specific for humans and promote adherence to cells of small intestines.

2. Toxins:

a) ST: heat-stable, activates guanylyl cyclase → increased cGMP

b) LT: heat-labile, exotoxin, activates adenylyl cyclase → increases cAMP

Both toxins result in hypersecretion of fluids and electrolytes, less absorption of Na+ → watery diarrhea (lasts few days).

LT toxin and colonizing factors are immunogenic. (induce antibody production).

People in developing countries are less prone to develop diarrhea because they are likely to have antibodies from previous exposure.

3. Shiga toxin-producing E-coli (STEC): (verocytoxin-producing E-coli (VTEC)/ enterohamorrhagic E-coli (EHEC)

- Most common E-coli strain that causes diarrhea (gastroenteritis)
- Does not ferment sorbitol
- Linked to consumption of fresh products e.g. lettuce and undercooked beef (hamburger), …
- Most common serotype **O157:H7**

Diseases:

- Linked to hemorrhagic colitis
- 10% of patients develop hemolytic uremic syndrome (HUS) –frequently in children

Clinical picture:

- Colonic edema and non-bloody watery diarrhea that may develop to bloody-diarrhea later.
- Significant abdominal pain and fecal leukocytes in 70% of cases, **NO fever**

Pathogenesis:

- shiga-like toxin 1 and 2 → affect 60s ribosomal subunit.
- no invasion, bloody diarrhea is due to hemolysis effect of the toxin

Dx: toxin detection by enzyme immunoassay, PCR to detect toxin genes directly, cell culture using vero cells, direct test for O157:H7 strain, sorbitol MacConkey agar (doesn't ferment sorbitol)

Rx: self-limited in 5-10 days

Antibiotics are contraindicated for they increase the HUS risk

Anti-motility and opioids are contraindicated.

4. Enteroinvasive E-coli (EIEC): foodborne illness

Most commonly in children in developing countries.

The only E-coli that's non or late-lactose fermenter and it's nonmotile.

Pathogenesis: invade intestinal mucosa, requires large inoculum

Disease: very similar to shigellosis, invasion→ bloody diarrhea, fever

5. Enteroaggregative E-coli (EAEC):

Causes acute and chronic diarrhea (>2 weeks)

Severe, persistent diarrhea in HIV patients.

Stacked-brick pattern of adherence to small intestines epithelia

Pathogenesis: shiga-like toxin, enterotoxin, hemolysin

Dx: clinically confirmed by tissue culture

ETEC, EPEC, EAEC → non-inflammatory diarrhea

EIEC, STEC → inflammatory diarrhea

Rx: G(-) bacteremia: fluid and electrolyte replacement, institution of antibiotics, and treatment of disseminated intravascular coagulation.

- No single specific therapy is available. The ampicillin, cephalosporins, … have marked antibacterial effects against the enterics. Trimethoprim-sulfamethoxazole is also used.
- Drug resistance is common.

Prevention: 1. Daily ingestion of bismuth subsalicylate suspension and regular doses prophylactics.

2. Caution to sanitation and hygiene when travelling, and avoid undercooked food.

3. Control of infection for hospitalized patients by sterilization, disinfection,..

****Shigella:** shigellosis (bacillary desentry)

- G(-) rods, facultative anaerobes but grow best aerobically, form transparent convex, circular colonies, coccobacillary shape in young cultures.
- All ferment glucose, non-lactose fermenters except shigella sonnei → colorless on EMB
- Non-motile (no H antigen)
- Do not produce H2S (to differentiate from salmonella).

Epidemiology:

A strictly human pathogen, most commonly affect children under 5 or 10

Most common serotype in developed countries → S.sonnei then S.flexneri

in developing countries → S.flexneri

Transmission: highly communicable. feco-oral, person to person , toilet seats, (food, fingers, flies, feces)

Etiology: Low inoculum needed.

1. Group A: shigella dysenteriae: produces type 1 shiga toxin, most severe and highest mortality but not common.
2. Group B: shigella flexneri: mild disease
3. Group C: shigella boydii
4. Group D: shigella sonnei: intermediately severe

Pathogenesis: limited to GI (no bacteremia)

Invasion of mucosal epithelial cells e.g. M cells

Shiga toxin is synergistic but not main factor for invasion

Micro abscesses → necrosis of mucosa → ulceration and bleeding → pseudomembrane of fibrin, leukocytes, debris, necrotic tissue → granulation and scar tissue formation.

Toxins: 1. endotoxin (Lipopolysaccharide) → irritation to the bowel wall

2. exotoxin (shigella dysenteriae toxin): heat-labile, neurotoxic, cytotoxic and enterotoxic.

Enterotixic: diarrhea (similar to shiga-like toxin)

Neurotoxin: severe, fatal, febrile convulsions and seizures.

Clinical findings:

incubation period of 1-2 days, sudden onset of abdominal pain and watery diarrhea (due to exotoxin). Later, number of stools increases with mucus and blood (due to invasion).

Each bowel movement is accompanied by straining and tenesmus.

Most cases are self-limited in 2-5 days.

In elderly and children → risk of dehydration, acidosis, and death.

A person can be re-infected even though body develops antibodies.

Dx:

1. specimens: stool, mucus, rectal swabs → Large numbers of fecal leukocytes and RBC are seen microscopically
2. culture: MacConkey or EMB → colorless

   selective media; hektoen enteric agar or salmonella-shigella agar
3. serology: not used → large false positive

Rx: ciprofloxacin, ampicillin, …

Convalescent carriers: they shed pathogenic bacteria even though they're healthy.

Opioids should be avoided.

Control: IgA antibodies may be important to prevent reinfection.

Serum antibodies to salmonella are IgM