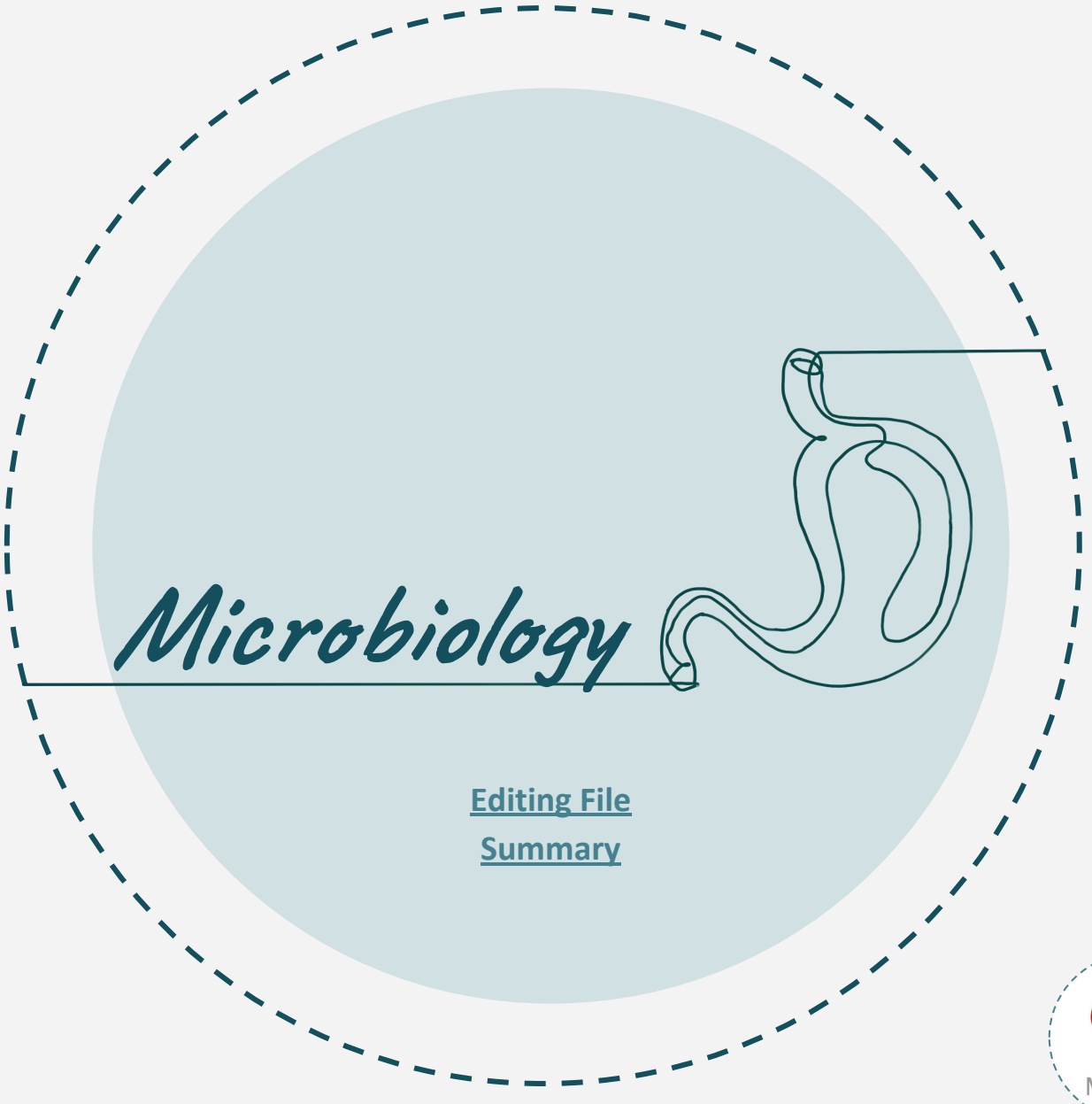
Normal Flora of the GIT and introduction to infectious diarrhea



Editing File

Summary

MED439
KING SAUD UNIVERSITY

MICROBIOLOGY
Team 439

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- Girls' slides
- Main content
- Important
- Boys' slides
- Extra
- Drs' notes

Objectives:

- Recall the common normal flora of the GIT
- Understand the role of the normal flora of the GIT in diseases.
- Describe the epidemiology, risk factors & host defenses in preventing GI infections.
- Describe various types of acute diarrheal illnesses, the pathogens that cause them, their clinical presentation, pathogenic mechanism and prevention.
- Explain the pathogenesis of E.coli, Campylobacter, Yersinea & Clostridium difficile and their management.
- Discuss microbiological methods used for the diagnosis of common bacterial agents causing diarrheal infection.

Normal Flora

Normal Flora

- Microorganisms that are frequently found in various body sites in normal, healthy individuals
- Have symbiotic relationship that benefit the host.
- Inhibit competing intruder.
- Types and number vary according to the age and physiological status
- Can cause disease in immunocompromised patients
- Able to colonize and multiply under the exciting condition of different body sites.

Normal Flora of GIT

 1:	**Oral Cavity**	• Contains high number of flora which vary from site to site of the mouth
 2:	**Saliva**	• Contains mixed flora: 10^8 organisms/ml
 3:	**Stomach**	• Empty stomach has no normal flora in health due to HCL and peptic
 4:	**Small Intestine**	• Very scanty except near colon.
 5:	**Colon** Most colonized area	• 10^{10} organisms/gm stool: >90% are Bacteroides (anaerobic) 10% other bacteria. • Direct effect of diet composition

	Normal flora (low virulence): Only cause disease if there was anatomical disruption/immunosuppression	Potential pathogen (carrier): Can cause variety of infections even without anatomical disruption
Mouth	• **Viridans streptococci** [1] • Neisseria spp. • Moraxella, • Peptostreptococcus	• Candida albicans
Nasopharynx	• Neisseria spp., • Viridans sterpt. • Moraxella, • Peptostreptococcus.	• S.pneumoniae • N.meningitidis • H.influenzae • S.pyogenes • S.aureus
Stomach	• Streptococci, • Peptostreptococcus, • others from mouth.	-
Small intestine	• scanty, variable	-
Colon	• Bacteriodes, • Fusobacterium, • Eubacterium, • Lactobacillus, • Enterobacteriaceae, • Clostridium, • Enterococcus	• B.fragilis, • E.coli, • Pseudomonas, • Candida, • Clostridium (C. perfringens, C. difficile)

Both pure normal flora and potential pathogens can be opportunistic.

1. Especially after dental procedures (remember CVS)

Role of Normal Flora in Diarrhea

Role of Normal Flora in Diseases

Many are opportunistic pathogens: e.g. perforation of the colon from ruptured diverticulum ➔ feces enter into peritoneal cavity and cause peritonitis and maybe septicemia
- Viridans streptococci of oral cavity enters the blood and colonize damaged heart valves. (Esp with prosthetic valve)
- Mouth flora play a role in dental caries

Compromised defense systems increase the opportunity for invasion.

Death after lethal dose of radiation due to massive invasion of normal flora.

E.coli : the most common Enterobacteriaceae, a facultative flora of colon followed by Klebsiella, Proteus and Enterobacter.

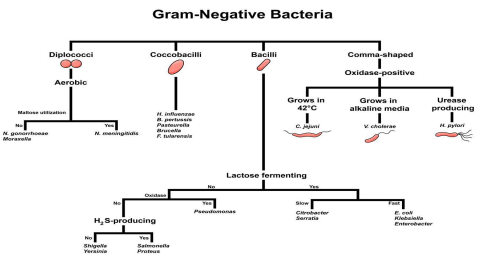
Salmonella, Shigella and Yersinia are NOT normal flora of the intestinal tract.
They are potentially pathogenic

Some strains of E.coli ,Salmonella , ,Shigella and Yersinia enterocolitica are able to cause diseases in the intestinal tract.

Generally speaking, normal flora becomes pathogenic when there is anatomical disruption or damage e.g. perforation.

Enterobacteriaceae

- Gram negative rods
- Facultative anaerobes
- Glucose fermenters
- Reduce nitrates into nitrates
- Oxidase -ve
- Catalase +ve
- Motile except Shigella and Klabsiella
- Non-fastidious
- Grow on MacConkey agar



Intestinal pathogens

- Invasive and Cytotoxic strains[1] produce inflammatory diarrhea (Dysentry) with WBCs and/ or blood in the stool.
- Enterotoxin-producing strains[2] cause watery diarrhea with loss of fluid.[3]
- Some produce systemic illness due to spread to multiple organs such as enteric (typhoid) fever.

1. Enteroinvasive E.coli & Yersinia enterocolitica.

2. Enterotoxigenic E.coli, Enterohemorrhagic E.coli, Clostridium difficile & Staph. aureus

3. Because it causes absorption issues

Diarrhea

Introduction

- Acute diarrheal illness is one of the most common problems evaluated by clinicians.
- A major cause of morbidity and mortality world-wide.
- Most of healthy people have mild illness but other might develop serious squeals so it is important to identify those individuals who require early treatment.

Definition of diarrhea

- Stool weight in excess of 200 gm/day, or three or more loose or watery stools/day.
- Alteration in normal bowel movement characterized by decreased consistency and increased frequency.
- Less than 14 days in duration.

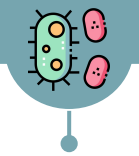
Epidemiology

- 1.2 - 1.9 episodes per person annually in the general population
- 2.4 episodes per child <3 years old annually
- 5 episodes per year for children <3 years old and in daycare
- It is more worrying in children because it is difficult to rehydrate/keep them hydrated.
- Seasonal peak in the winter.

Etiology

Viral

70-80% of infectious diarrhea in developed countries
Majority are self limiting



Bacterial

10-20% of infectious diarrhea but responsible for most cases of severe diarrhea
Majority are self limiting but some of them need treatment

Protozoan

less than 10%.
Majority are self limiting but some of them need treatment

Classification

Infectious diarrhea[1]

Viral or Bacterial infections, e.g.:

- Campylobacter
- Shigella
- Salmonella
- Yersinia
- Vibrio cholerae
- E.coli

Food poisoning[2]

- Staph.aureus[3]
- Clostridium perfringens
- Bacillus spp.

Traveler diarrhea

- Enterotoxigenic E.coli.

Antibiotics associated diarrhea

- Clostridium difficile.

1. The toxin is released within the body

2. The toxin is pre-formed in the food

3. They have a heat stable toxin, short IP (1-6 hours), with severe symptoms in short duration

Risk factors and clinical presentation of diarrhea

Risk factors

Dr: Low stomach acidity, restaurants food and immunity related are the main risk factors

- Antibiotics decrease the normal flora to less than 10^{12} (C. difficile)
- Patient underlying illness & medication, **low stomach acidity**, cyst, spores
  Low stomach acidity makes it easier for bacteria survive.
- Abnormal peristalsis.
- **Food from restaurants**
- Recent travel to developing countries.
- Low Immunoglobulin A (IgA).
  Can affect severity and incidence of the infection.
- Family member with gastrointestinal symptoms.
- Median infective dose (ID50)
- Lack of hygiene and sanitation

Clinical Presentation & Pathogenic Mechanisms:

	Enterotoxin mediated (I)	Invasive (II)
Stool analysis	• Lack of pus in the stool (no gut invasion/cell destruction)	• Pus and blood in the stool
Symptoms	• No fever • Vomiting • Non-bloody diarrhea (Watery) • abdominal cramps.	• Fever due to inflammation • Dysentery syndrome: gross blood and mucous
Location	• Small intestine affected[1]	• Colonic mucosa
Etiology	• Vibrio Cholerae [4] • **Staphylococcus aureus** [3] • Clostridium perfringens [4] • Bacillus cereus • Some viral and parasitic infections.	• Shigella[2] • Salmonella spp[2] • Campylobacter • some E.coli • Entamoeba Histolytica
Characteristics [5]	• Some have rapid onset[3] (<12 hour if due to preformed toxin ingestion)	• Extension to lymph nodes • Incubation period 1-3 days • EHEC bloody diarrhea • Entamoeba histolytica 1-3 wk
Notes	Patient either ingests the toxin itself and It comes very quick, or ingests the organism that will multiply and release toxins (longer incubation period)	

1. causing watery diarrhea
2. they are both cyto-toxic & invasive.
3. Staph. Aureus: food contamination with staph. Aureus (while preparation/exposed in room temperature/open buffet meals) → heat stable toxin is released → patient eats it → diarrhea & severe vomiting 1 hr later and it relieves after 12 hrs it is common in food poisoning and causes severe acute diarrhea. it, comes on quickly and gets relieved quickly.
4. Vibrio cholerae & clostridium perfringens : patient ingest the bacteria itself and it releases the toxin within the body.
5. Onset & incubation period varies according to what has been ingested by the patient (did patient ingest the bacteria? or the toxin itself?)

Infectious Agents

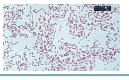
1- Yersinia enterocolitica

mainly causes disease by invasion (a cause of diarrhea both watery & bloody)

Morphology	• Gram negative bacilli • Non lactose fermenter
Disease	• Mesenteric lymphadenitis in children and septicemia in immunocompromised hosts
Prevalence	• Common in Europe, USA, Canada and cat, dog, swine (chitterlings)[1]
Symptoms	• Presented with enteritis, arthritis and erythema nodosum[2]
Characteristics	• Survive cold temperatures and associated with transfusion of packed red blood cells.
Diagnosis	• Growth at 25°c-30°c media Cefsulodin-Irgasan-Novobiocin (CIN)
Other Info	• Generalize infection in adult and children 1-5 years usually mild but in old children and adult **mimic appendicitis**

2- Campylobacter

world wide infection (especially among children)

Morphology	★ Gram-negative **curved** bacilli. (spiral or S-shaped) 
Species	• **C.jejuni**, C. coli, C fetus.
Sources	• Dogs, cats, birds, poultry[3], water, milk & meat. Person to person transmission can occur.
Clinical Presentation	• Incubation period: 2-6 days • Lower abdominal pain , watery or dysenteric diarrhea with pus and blood. • fever in some patients . Nausea and vomiting are rare. • Self limiting after 2-6 days. • Chronic carrier & outbreaks uncommon..
Lab[4] Diagnosis	• Use transport media • Culture on CAMPY BAP media containing antibiotics. • Incubate in microaerophilic atmosphere (5%O_2 - 10%CO_2 - 85%N) at 42°C except C.fetus at 37°C • Identification :Gram stain/culture /biochemical/Serology.
Complications	• Autoimmune disease like Guillain-Barre' syndrome [5] • Extra-intestinal infections eg. reactive arthritis ,bacteremia ,lung infection and others frequently preceded by C.jejuni infection. • Complications can either be caused by immune reaction to bacteria such as GBS and reactive arthritis, or it can be caused by the bacteria itself such as lung infection and bacteremia.
Treatment	• Only severe cases • Erythromycin or Ciprofloxacin

1. Chitterlings are a prepared food usually made from small intestine of a **pig**
2. Erythema nodosum is a type of skin inflammation that is located in a part of the fatty layer of skin
3. Chicken, sometimes not directly, by cross-contamination to other food products, e.g. When you use a knife for cutting an uncooked chicken then using the same knife for other products (e.g. vegetables), so appropriate handling of raw chicken is essential.
4. by taking stool sample
5. Ascending paralysis

Infectious Agents

3- E.Coli

- About 10 -15% of strains of E. coli associated with diarrhea. Other strains associated with extra-intestinal diseases (septicemia, meningitis & UTI).
- Based on virulence factors, clinical manifestation, epidemiology and different O and H serotype.

Types of Diarrheagenic E. coli :

- Enterotoxigenic E. coli (E T E C) similar to vibrio
- Enteroinvasive E. coli (E I E C) similar to shigella
- Enteropathogenic E. coli (E P E C) in children
- Enteroaggregative E.coli (E A E C) in children
- Enterohemorrhagic E. coli (E H E C) similar to shigella

All of them in one page? click here

Enterotoxigenic E. coli Similar to mechanism and symptoms of vibero	
Disease	• **Major cause of Traveler's diarrhea** in infant and adult in developing countries due to consumption of contaminated food and water.
Infective Dose	• It has **high** infective dose 10^6-10^{10}
Symptoms	• **watery diarrhea**, abdominal cramps and some time vomiting .
Toxins	• Produce heat-labile toxin (LT) and Heat-stable toxin (ST), Each has two fragment (A and B) • No invasion or inflammation.
Other info	• LT leads to accumulation of cAMP, which leads to hyper-secretion of fluid with no cellular injury. • **Self Limiting No routine diagnostic method needed.** • if the patient suffered from travelers diarrhea he will get immunity that lasts for a few years.

Infectious Agents

Enteroinvasive E. coli	
Disease	• Produce dysentery (Penetration, **invasion** and destruction). • Common in children. • Infective dose: 10^6
Symptoms	• Fever, severe abdominal cramp, malaise and watery diarrhea
Other info	★ **Similar to Shigella spp.** (non lactose fermenter & non motile) E.coli is motile and lactose fermenter, but this strain (EIEC) is similar to Shigella that it is not motile nor does it ferment lactose. • Transmission: Fecal oral route.

Enterohemorrhagic E.coli	
Disease	★ **0157:H7** Hemorrhagic diarrhea, colitis and **hemolytic uremic syndrome (HUS) manifested with low Platelet count, hemolytic anemia** (Thrombocytopenia) **and kidney failure**
Symptoms	• **Bloody diarrhea** • Low grade fever and stool has no leukocytes.
Toxin	• **Cytotoxin : Shiga-toxin I & II (verotoxin I and verotoxin II)**[1] (Similar to toxin produced by Shigella dysenteriae).
Diagnosis	• culture on SMAC(sorbitol MacConkey agar) • Vertoxin detection by immunological test. • nucleic acid testing (NAT).
Prevalence	• Fatal disease in young and elderly persons in nursing homes
Other Info	• Management of HUS required. ★ **Antimicrobial therapy not recommended**[2] . • E.coli other than 0157:H7 can cause Hemolytic uremic syndrome (HUS) ★ **Caused by Undercooked hamburgers (ground beef)**[3] , unpasteurized dairy products, Apple cider, cookie dough, and contaminated lettuce.

Enteropathogenic E.coli	
Disease	• Cause infantile diarrhea (bottle fed infants)
Symptoms	• Low grade fever • Malaise • Vomiting and watery diarrhea
Other	• Outbreak in hospital nurseries and day-care centers. • Stool mucus but no blood. • **Disrupt microvilli and intestinal absorptive function** (due to aggregation).

1. vero cell are a lineage of cells used in cell culture.
2. Some antibiotics induce shiga-toxin production such as TMP-SMX, and the 2nd theory: if the bacteria broken down, it will release toxins
3. Why only hamburgers? Why not steak? Because steak is not minced (only the outer surface is exposed to the bacteria and it will be exposed to heat). However, in the case of hamburger, beef will be minced and also contaminate the inner portion of the meat (it needs to be cooked very well & reach a certain temperature)

Infectious Agents

Enteroaggregative E.coli	
Disease	• Pediatric diarrheal disease
Symptoms	• Produce mucoid, • watery diarrhea, • Vomiting • Dehydration and abdominal pain
Other	• Produce aggregative stacked bricked appearance (like blocks) adherence to surface of intestinal epithelial cells. • May resolve after 2 weeks or more.

4-Clostridium difficile mainly related to antibiotic use and things that disrupt normal flora	
Morphology	• **Gram-positive Bacilli** • **Anaerobic spore forming** (live on surfaces/important in hospitals) • VERY INFECTIOUS
Disease	★ **Antibiotic associated diarrhea**, (ampicillin, cephalosporins & clindamycin) ○ Antibiotic use for the last 8 weeks (community acquired) • hospital stay for at least 3 days (hospital acquired), (Cultured from inanimate hospital surfaces.)
Pathogenesis	• Transmit from person to person via Fecal-Oral route. • **Disruption of the endogenous bacterial flora of the colon**.
Symptoms	• Patient Presents with fever, leukocytosis, abdominal pain and diarrhea
Toxins	• **Produce toxin A (enterotoxic & cytotoxic effects) and B (cytotoxic)** that can bind to surface epithelial cell receptors leading to inflammation, mucosal injury and diarrhea
Histological findings	• Pseudomembrane can result (neutrophils, fibrin, and cellular debris in the colonic mucosa) and toxic megacolon.
Diagnosis	• Direct toxin and antigen detection from stool by enzyme immunoassay (EIA) • Nucleic acid testing NAT. (Detects the gen of the toxin) • Culture can be done but its not routinely used for detection of c. diff
Treatment	• Metronidazole ± **oral Vancomycin**[1] and supportive treatment.

1. Has to be given orally because I.V Vancomycin does not reach rectum very well

Clinical Characteristics & Lab diagnosis

Pathogen	Typical incubation Period	Duration	Typical Clinical Presentation	Assorted foods
Salmonella species	1-3 Days	4-7 Days	Gastroenteritis	Undercooked eggs or Poultry, produce
Campylobacter jejuni	**2-5 Days**	2-10 Days	Gastroenteritis	**Undercooked poultry, Unpasteurized dairy products**
E. coli, Enterotoxigenic	1-3 Days	3-7 Days	Gastroenteritis	Many Foods
Shigella species	1-2 Days	4-7 Days	Gastroenteritis	Produce, Egg salad
Listeria monocytogenes[1]	2-6 Weeks	Variable	Gastroenteritis, Meningitis abortion	Deli meat Hotdogs Unpasteurized dairy products
Bacillus cereus	1-6 Hors	<24 Hours	Vomiting, Gastroenteritis	Fried rice Meat
Clostridium botulinum[2]	12-72 Hours	Days-Months	Blurred Vision, Paralysis	Home-canned foods Fermented fish
Staphylococcus aureus	**1-6 Hours**	1-2 Days	Gastroenteritis, Particularly nausea	Meats, pork, Potato Unpasteurized Dairy products
Yersinia enterocolitica	1-2 Days	1-3 Weeks	Gastroenteritis, Appendicitis-like syndrome	Undercooked pork Unpasteurized dairy products

Lab diagnosis[3]

- **Stool specimen:**
  - **Microscopy**[4] → for presence of polymorphs or blood may help.
  - **Molecular testing**
  - **Toxin assay**: → if C.difficile toxins is suspected.
  - **Culture:**

➔ **Selective media** to inhibit the growth of normal flora in stool for Salmonella, Shigella & Campylobacter

➔ Culture for Vibrio cholerae, EHEC or Yersinia if suspected

1- gram +bacilli ,causes meningitis in neonates and immunocompromised.

2- clostridium botulinum Type A is the most fatal toxin in the world.

3- most of them are self limiting but in cases of immunocompromised patients we do lab diagnosis

4- 5. only if we suspect shigella;there will be increase in WBC

Drs' notes

Dr. Khalifa

- Normal flora of GIT can be affected by many factors such as diet and antibiotic history. Moreover, some studies revealed that delivery mode (normal vaginal delivery vs. c-section) affects the composition of normal flora as most of it happen to be acquired during delivery from birth canal.
- Acute diarrheal illness is one of the most common problems in developing countries with short access to medical care. Children in these countries might die from acute gastroenteritis due to hygiene issues.
- There are two pathogenic mechanisms for diarrhea:
  - (1) Enterotoxin mediated (watery diarrhea):
    - **1st scenario:** patient directly ingests the toxin produced by the bacteria. A good example of this is food poisoning with staphylococcus aureus: food contamination with staph. aureus while preparing or when left exposed in room temperature (open buffet dinners/poor hygiene) → heat stable toxin is released (toxin is not affected by heat/cooking) → patient eats it → severe diarrhea & vomiting that starts 1 hour later & relieves after 12 hours. (comes on quickly and gets relieved quickly). Important to know: staph aureus has a VERY short incubation period.
    - **2nd scenario:** patient ingests the organism itself which will multiply within the body and release toxins (has longer incubation period).
  - (2) Invasive (bloody diarrhea): such as shigella, salmonella, campylobacter, and some E.coli strains (EHEC).
- Yersinia enterocolitica: most special about it is that it mimics appendicitis.
- Campylobacter: Gram negative curved bacilli. Main source is poultry and raw/undercooked chicken (not directly, usually by cross-contamination. Ie. use same knife/cutting board to cut raw chicken and reuse it on vegetables that will not be cooked later). This bacteria has complications (1) related to immune reaction such as reactive arthritis and guillain barre syndrome. (2) related to bacteria itself such as bacteremia and lung infection.
- Enterotoxigenic E. coli: it causes traveler's diarrhea (patient will get immunity that will last for a few years), and it has two toxins LT & ST.
- Enteroinvasive E. coli: similar to Shigella.
- Enterohemorrhagic E.coli: it produces shiga-like toxin and causes hemolytic uremic syndrome (HUS): manifests as low platelet count, hemolytic anemia (Thrombocytopenia) and renal failure. It is important to know that antimicrobial therapy is NOT recommended with it as some antibiotics like TMP-SMX induce shiga-toxin production (2nd theory: if the bacteria broken down, it will release more toxins).
- Note that EHEC is more likely to cause HUS unlike shigella (much less likely to cause HUS)
- Enteropathogenic E.coli: disrupt microvilli and intestinal absorptive function (due to aggregation) and it is common in children.
- Enteroaggregative E.coli: produce aggregative stacked bricked appearance.
- Clostridium difficile: mainly related to antibiotic use and disruption of normal flora.
  - **Pathogenesis:** antibiotic use & disruption of normal flora → exposure to clostridium → colonization → multiplication→ cytotoxin B production (causes cell destruction/perforation/dilation) - diarrhea (watery/bloody). Note that toxin B is the most important toxin in causing the disease.
  - **Spectrum:** asymptomatic carrier → mild diarrhea → severe diarrhea → toxic megacolon → perforation → pseudomembranous colitis.
  - It is treated with vancomycin (has to be given orally because I.V does not reach rectum very well).
  - In the lab: we first look for antigens (using EIA), then we look for the toxin (also using EIA), and finally we use NAT to look for the genes of toxin.
- it is not important to know the details of cultures (just know that each one has a selective special media).
- Most organisms with high infectious dose except enterohemorrhagic E.coli and shigella which have a low infectious dose
- Treatment of diarrhea is usually supportive (rehydration), and antibiotics use is very limited.
  - Staph aureus: no need to treat (just goes on its own)
  - Vibrio: can be treated sometimes (explained in details in other lecture) Clostridium difficile: has to be treated
  - Clostridium perfringens: usually self limited
  - Salmonella: it depends
  - Shigella: always has to be treated

Prof. Hanan

- Patients with campylobacter and brucella too can present with autoimmune diseases one month later.
- Virulence and epidemiology of E.coli strains changes depending on O (somatic polysaccharide) and H (flagella).
- Normally, if we did a stool culture in the lab and found E.coli, it will be considered as normal flora. However, if it was reported that it is related to many patients in ICU or daycare center and etc.., enteropathogenic E.coli outbreak is suspected.
- You must know that 0157:H7 is the most common strain of enterohemorrhagic E.coli

Quiz

MCQ

Q1: Which organism can cause Guillain-Barrié syndrome?

A- Vibrio cholerae
B- Salmonella typhi
C- Campylobacter jejuni
D- Clostridium

Q2: Young boy was brought to the hospital with diarrhea,skin rash and abdominal pain, his doctors firstly thought it was appendicitis. What is the causative agent?

A- Yersinia enterocolitica
B- Staph.aureus
C- Salmonella
D- Campylobacter

Q3: A 30 Yo male patient presented to hospital with vomiting and diarrhea , during taking history he said that he ate from a restaurant 5 hours ago which of these organisms caused his food poisoning?

A- Campylobacter
B- Shigella
C- Enterotoxigenic E.coli
D- Staph.aureus

Q4: which microbe causes Traveler's diarrhea ?

A- Yersinia enterocolitica
B- Enterohemorrhagic E.coli
C- Enterotoxigenic E. coli
D- Clostridium difficile

Q5: Highlight symptom of Enterohemorrhagic E.coli ?

A- Watery Diarrhea
B- Abdominal cramps
C- Bloody Diarrhea
D- Malaise

Q6: Gram negative bacilli, Non-motile & Non-lactose fermenting?

A- Enteroinvasive E.coli..
B- Clostridium .
C- Bacillus cereus .
D- Enteropathogenic E.coli .

Answers: Q1:C | Q2:A | Q3:D | Q4:C | Q5:C| Q6:A

SAQ

Dr.Khalifa: A 34 years old patient came to the ER complaining from pneumonia, the doctor decided to hospitalize the patient and give him I.V antibiotics for 4 days, after that the patient discharged after he became well, 2 days later he came back to the hospital because of bloody diarrhea, fever and abdominal pain, a blood sample was taken and the lab investigations indicated High WBC.

Q1: What is the most likely causative agent?

A:Clostridium difficile

Q2: What is the most likely cause/inducer of the infection?

A: Antibiotics

Q3: What are the diagnostic methods for this case?

A: EIA and NAT

Q4: What is the Pathogenesis?

A:Antibiotic use & disruption of normal flora → exposure to clostridium → colonization → multiplication→ toxin B production (causes cell destruction/perforation/dilation) - diarrhea (watery/bloody).

Q5: What is the treatment in this case?

A:Oral vancomycin

Quiz

SAQ

Dr.Khalifa: 30 years old presented with 2 days history of diarrhea. He reported that he attended barbecue two days before and had chicken that might not be cooked very well.

Q1: What might be the cause of his diarrhea?

A:Campylobacter or salmonella

Q2: A culture was done and it showed curved Gram -ve rods. What is the most likely organism?

A: Campylobacter

Q3: What are some complications and extra manifestations that is caused by this infection?

A: Autoimmune disease like Guillain-Barre' syndrome
Extra-intestinal infections eg. reactive arthritis ,bacteremia ,lung infection and others frequently preceded by C.jejuni infection.

Q4: What is the treatment plan?

A: No treatment unless if it's severe

Dr.Khalifa: Patient came with bloody diarrhea, high creatinine levels, anemia (thrombocytopenia) after eating undercooked burgers.

Q1: What is the most likely organism?

A: Enterohemorrhagic (EHEC)

Q2: What is the disease caused by this organism?

A: Hemolytic uremic syndrome (HUS)

Q3: What is the source of this infection?

A: Uncooked ground beef burger

Q4: What is the treatment required?

A: no antibiotics treatment but supportive care

Dr.Khalifa: Patient developed severe vomiting and diarrhea one hour after open buffet meal with his friends.

Q1: What is the most likely causative organism?

A:Staph.aureus (Enterotoxin production)

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