M17 - Gastrointestinal Tract Infection

3 more properties

Learning Objectives

- Common clinical syndromes caused by infection affecting the gastrointestinal tract and the microbes that cause them:
  - Food poisoning
  - Gastroenteritis
  - Colitis
  - Antibiotic-associated colitis
- Causes and disease process in enteric fever

Introduction to GE

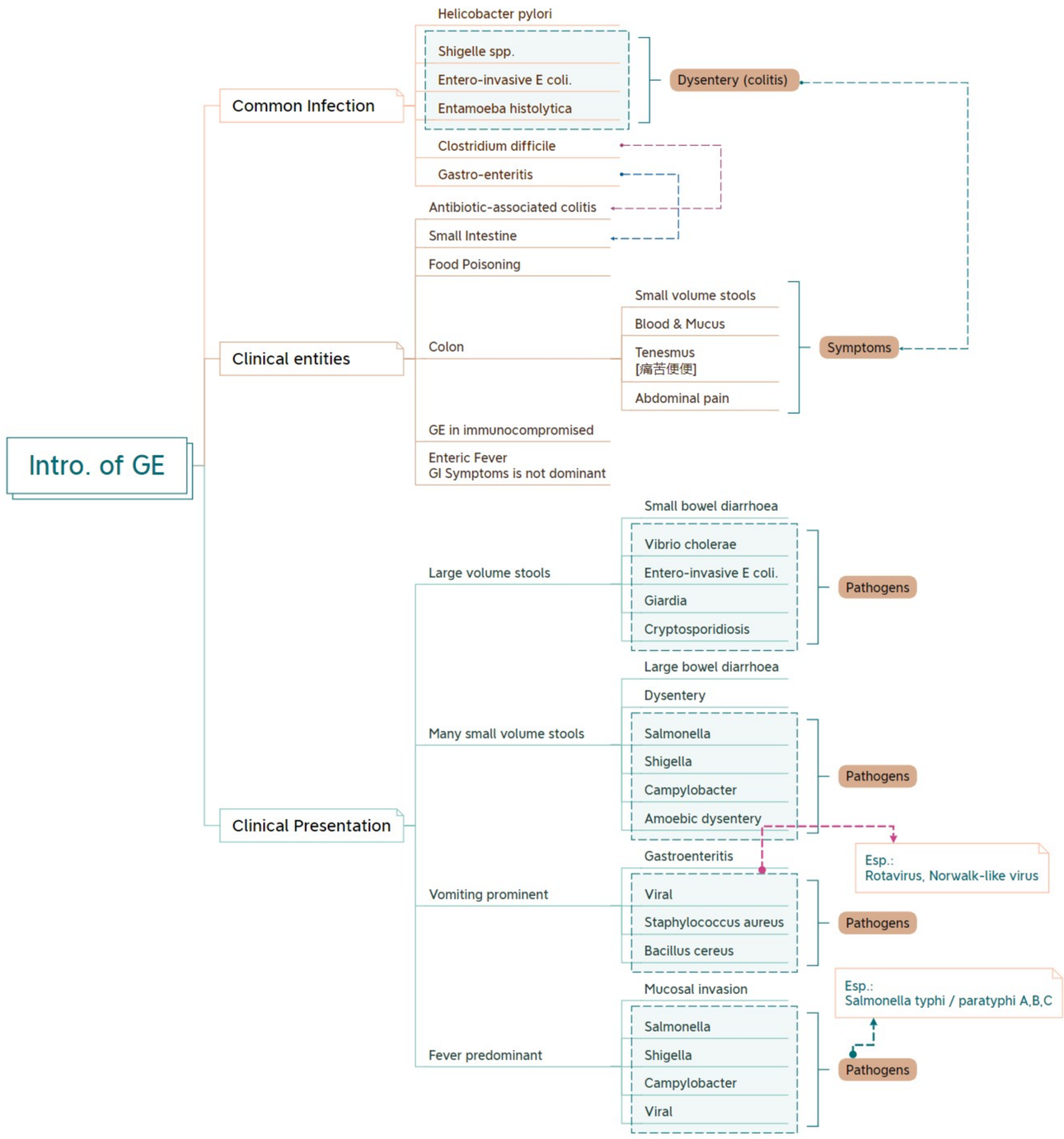
- Clinical entities

	Symptoms-1	Symptoms-2	Pathogens
Colon	Small volume stools	Blood & Mucus	Shiggelle spp.
	Tenesmus	Abdominal pain	Entamoeba histolytica
			Entero-invasive E coli.
Small Intestine			Gastroenteritis
Antibiotic-associated colitis			Clostridium difficile [G+ anaerobic bacilli]
** **Pseudomembranous Colitis**			Clostridium difficile [G+ anaerobic bacilli]
Food Poisoning			
Enteric Fever			
GE in immunocompromised			

More about **Pseudomembranous Colitis:**

- More severe form of antibiotic-associated colitis.

Enterotoxin → Watery diarrhea
Cytotoxin → Cell Die
[Formation of Pseudomembrane on the mucosal surface]



- Clinical Presentation

	Remarks	Pathogens
Large volume stools	Small bowel diarrhoea	Vibrio cholerae
		Entero-invasive E coli.
		Giardia
		Cryptosporidiosis
Many small volume stools	Large bowel diarrhoea	Dysentery
		Salmonella
		Shigella
		Campylobacter
		Amoebic dysentery
Vomiting prominent	Gastroenteritis	Viral
		Staphylococcus aureus
		Bacillus cereus
Fever predominant	Mucosal invasion	Viral **[Rotavirus]** **[Norwalk-like virus]**
		Salmonella **[Salmonella typhi]** **[paratyphi A,B,C]**
		Shigella
		Campylobacter

Clinical Practices to the Gastrotestinal Tract Infections

History-Taking Method

Frequency	Type of stool	Presence of Vomiting	Presence of Fever	Other illness in family or contacts
Severity of illness	Duration of illness	Clinical presentation	Underlying diseases	Travel history

★ Physical Examination For GE

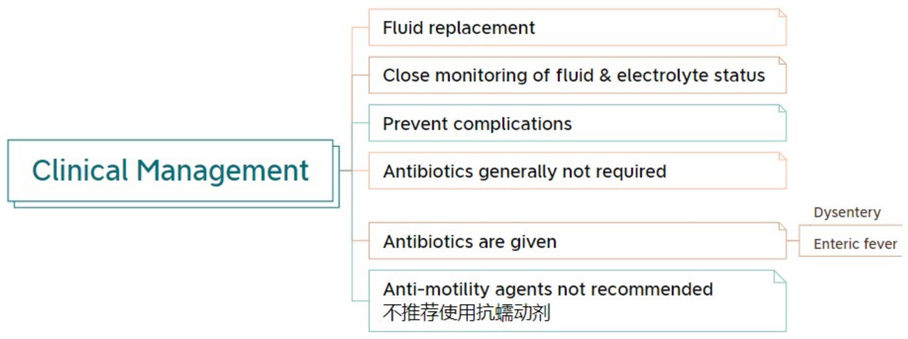
Fever	Toxicity	Degree of Dehydration

★ Investigation of Stool and vomit examination For GE

Clinical Courses

Bacteria	Average infective doses	Incubation period
Staph. aureus	10^8	4- 6 hours
Cl. perfringens	10^7	Nil
V. cholerae & V. parahaemolyticus	10^7	Nil
Salmonella spp. (not S. typhi)	10^7	6-72 hours
Salmonella typhi	10^4	6-72 hours
Shigella spp.	50-500	Nil
E. coli O157:H7	50-500	1-10 days

Clinical Management



▼ Text Version

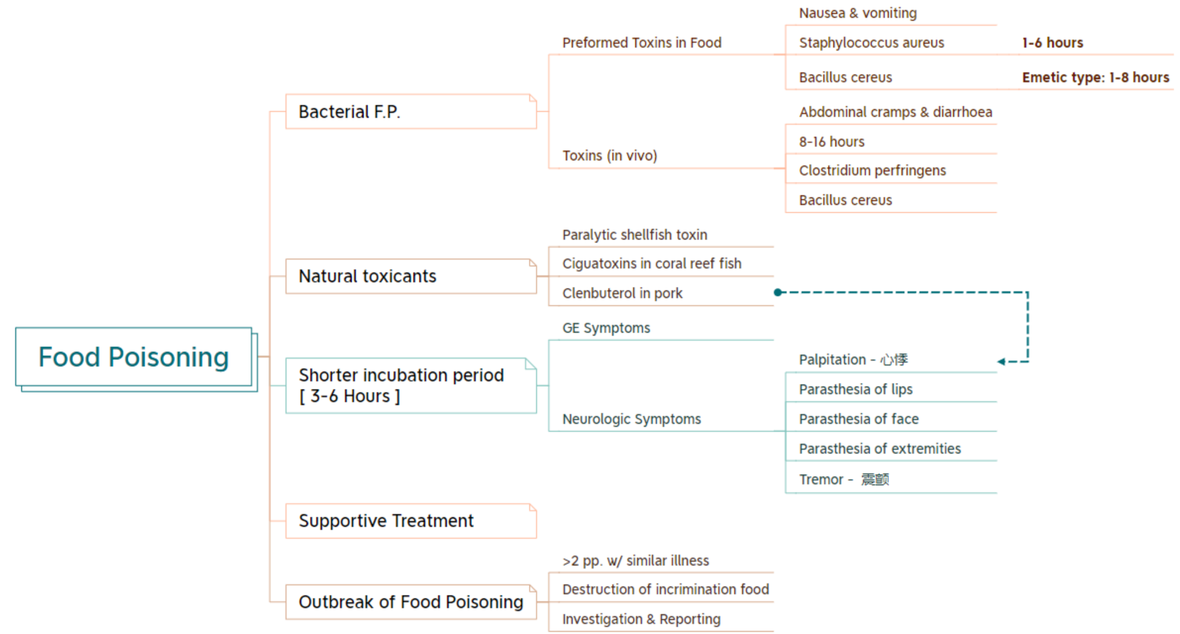
- Fluid replacement
- Close monitoring of fluid and electrolyte status
- Prevent complications
- Antibiotics generally not required (except for dysentery and enteric fever)
- Anti-motility agents not recommended

Food poisoning - Gastrointestinal Tract Infection

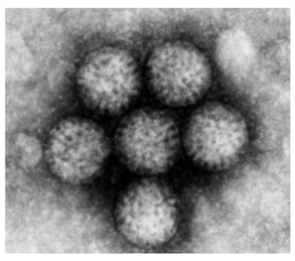
> An acute illness arising from consumption of food contaminated with Microbial agents, Toxins, Chemicals, Naturally occurring toxicants

Natural toxicants	Paralytic shellfish toxin
	Ciguatoxins in coral reef fish
	Clenbuterol in pork
Shorter incubation period - [3-6 Hours]	GE Symptoms
	Palpitation - 心悸
	Parasthesia of lips
	Parasthesia of face
	Parasthesia of extremities
	Tremor - 震顫
Supportive Treatment	
Outbreak of Food Poisoning	>2 pp. w/ similar illness
	Destruction of incrimination food
	Investigation & Reporting

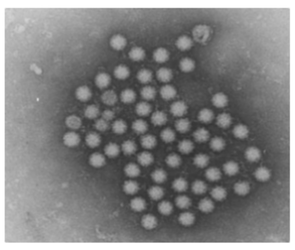
Bacterium	**Mechanism**	**Incubation Period**	**Clinical Presentation**
Staphylococcus aureus	Preformed toxin in food	1-6 hours	Nausea & vomiting
Bacillus cereus	Pre-formed Toxins (in vivo)	Emetic type: 1-8 hours	Nausea & vomiting
	Toxins (in vivo)	Long incubation type: 8-16 hours	Abdominal cramps & diarrhoea
Clostridium perfringens	Toxins (in vivo)	8-16 hours	Abdominal cramps & diarrhoea



Viral Gastroenteritis - Gastrointestinal Tract Infection



Rotavirus



Norovirus

	Rotavirus - Rota = wheel	**Norovirus - Calicivirus family**
Commonly found in	Infants less than 3 years of age	All ages
Transmission → **Live in water for months** → **Esp.: Shellfish, a Filter-feeders** → **Worldwide, esp. Winter**	Fecal-oral route	Fecal-oral route | 10e+5~10e+11 particles/g Vomitus: droplets Respiratory droplets
Symptoms → **Rehydration treatment**	Acute onset of fever	Vomiting in >50% cases; typically projectile
	Vomiting	
	Diarrhea (watery +/- mucus)	
Severity of Illness	Varies from subclinical to severe	Spontaneous and rapid recovery
Incubation Period	24-72 hours after exposure	24-48 hours after exposure
Duration of Illness	3-8 days	12-60 hours
Leading Cause of	Nil	Acute gastroenteritis
		Nosocomial infection
		Food-borne outbreaks [Oysters, Strawberries]
Most Severe in	Infants	Young and elderly
Potential to Cause Death	Rare, but severe cases → Dehydration & Complications	Can cause death in elderly

Remarks:

- Filter-feeders: concentrate viruses from water
- Not air-borne transmission for Norovirus: < 5 μm
- Disinfection
  - × **Moderate heat 60°C for 60 min**
  - × **alcohol hand rubs→ Washing hand is better**
  - √ **bleach/hypochlorite**

Enteric Fever - Typhoid/paratyphoid fever

Symptoms	Febrile illness
	Abdominal pain
	Headache
	Relative bradycardia
	Skin rash
	Splenomegaly
Pathogens	Salmonella typhi
	Salmonella paratyphi
Pathogenesis	From GI to Systemic Infection
Transmission	1. Penetration of ileal mucosa→ → infection
	2. Mesenteric lymph nodes - 肠系膜淋巴结
	3. Bloodstream causing systemic Infection
Complications	Sepsis, intestinal hemorrhage, perforation
Diagnosis of unknown origin	Culture of blood / stool
	Widal test - A Serology: O and H antibody titres

Bacterial GE Infection

Cholera - Vibrio cholerae

Spread	Incubation Period	Clinical Presentation	Complications
Contaminated water	A few hours to 5 days	Acute onset of severe watery diarrhea	Severe dehydration
Contaminated Shellfish / Food		Vomiting	Salt depletion → Renal failure

Cholera Toxins

1. Cholera toxin stimulates the secretion of chloride ions into the intestinal lumen.
2. This creates a high concentration of chloride ions in the lumen.
3. The high concentration of chloride ions creates an osmotic gradient.
4. Water flows from the interstitial space into the lumen due to the osmotic gradient.
5. Cholera toxin inhibits the absorption of sodium ions from the lumen into the epithelial cells.
6. Reduced absorption of water further promotes the secretion of water into the lumen.

> Summary:
> - A net movement of water and electrolytes from the body into the intestinal lumen.
> - Watery diarrhea & Dehydration

Escherichia coli

Type of Infection	Toxins	Recognized by
Gastroenteritis		
Entero-toxigenic E coli (ETEC)	LT / ST toxins [Similar to Cholera Toxin]	Toxin or Toxin gene detection
Entero-pathogenic E coli (EPEC)		O serotypes or Associated genes
Dysentery → Affects large and distal small intestine		
Entero-invasive E coli (EIEC) → Escherichia coli O157:H7	Resembles Shigella	Invasion-associated genes
Entero-hemorrhagic E coli (EHEC) → Shigella dysenteriae → Shigella flexneri	Shiga toxin	Shiga or VT toxin or serotypes O157

More about **Dysentery - Amoebic Dysentery:**

Caused by	Protozoan Entamoeba histolytica			
Acquired by	Amoebic cysts			
Infection site	Large intestine			
Symptoms	Mucosal ulceration	Dysentery	Bloody diarrhea	Bucus production
Complications	Liver abscess	Bowel perforation - 肠穿孔		
Diagnosis	Detection of cysts and trophozoites in stool	serology testing by indirect hemagglutination		
Treatment	Luminal agent: Metronidazole + diloxanide furoate			

▼ Life Cyle of Entamoeba histolytica

- Trophozoite - This is the actively feeding and dividing stage. Trophozoites live in the lumen of the large intestine. They have a single nucleus and ingest bacteria, cells, and debris.
  滋养体 - 这是活跃进食和分裂的阶段。滋养体生活在大肠腔内。它们有一个单核，并吞噬细菌、细胞和碎片。
- Cyst - When conditions become unfavorable, the trophozoite forms an inactive, hardened cyst with four nuclei. Cysts are passed in feces and can survive outside the body.
  囊 - 当条件变得不利时，滋养体形成一个不活跃、硬化的囊，囊内有四个核。囊通过粪便排出，并能在体外存活。
- Excystation - If cysts are ingested by a human host, enzymes in the small intestine allow excystation to release trophozoites.
  囊脱囊 - 如果囊被人体寄生宿主摄入，小肠中的酶能够使囊脱囊并释放滋养体。
- Trophozoites multiply by binary fission in the large intestine. Some burrow into the intestinal lining and cause ulceration and dysentery.
  滋养体在大肠内通过二分裂繁殖。其中一些滋养体会钻入肠壁并引起溃疡和痢疾。
- Encystation - As trophozoites progress down the intestine, they form cysts again which exit in feces, completing the cycle.
  囊化 - 随着滋养体在肠道中的进展，它们再次形成囊并通过粪便排出，完成生命周期。
- Extraintestinal infection - In some cases, trophozoites can spread through the bloodstream to other organs like the liver, lungs and brain. This can cause amebic abscesses.
  肠外感染 - 在某些情况下，滋养体可以通过血液传播到其他器官，如肝脏、肺部和脑部。这可能导致阿米巴脓肿。

Salmonellosis - GE / Septicemia / Enteric fever

Characteristic	Gram (-) bacilli	Flagellated (motile)	Facultative anaerobe
Common in	Raw meat, poultry, and Shellfish	Fermented meats (salami)	Milk and milk products
	Egg and egg-products (pudding)	Confectionery (chocolate)	Pasta, salads

Helicobacter pylori

Characteristic	Hydrolyze urea local production of ammonia and bicarbonate	G (-) bacilli	—
Associated w/	peptic ulcer disease	chronic superficial gastritis	adenocarcinoma of the stomach
Diagnosis	culture and urease test on biopsied tissue	Serology - Stool antigen detection	Urease breath test
Treatment	Triple therapy - Proton-pump inhibitors ★ **Omeprazole**	Triple therapy - **bismuth subsalicylate**	Triple therapy - Antibiotics ★ **metronidazole** ★ **clarithromycin** ★ **amoxicillin** ★ **tetracycline**