Acute Infectious Diarrheal Diseases and Bacterial food Poisoning

The wide range of clinical manifestations of acute gastrointestinal illnesses is matched by the wide variety of infectious agents involved, including viruses, bacteria, and parasites.

Frequency: 3 unformed stools per day

Volume: > 250 g/24h per stool

Duration:

- Acute diarrhea: <14 days (infectious cause)
- Persistent diarrhea: between 14-29 days
- Chronic diarrhea: > 30 days (parasitic or non- infectious cause)

- estimated 1.4 million deaths per year

Toxin

- Preformed in food
- Elaborated in the intestinal lumen

- Source

- Animals
- Human

PATHOGENESIS

Stages of infectious diarrhea:

1. The number of microorganisms that must be ingested to cause disease

- Shigella spp - 10-100 bact.
- Enterohemorrhagic E.coli, Salmonella typhi, Campylobacter jejuni - 1000 bact.
- Vibrio cholerae, Salmonella spp - 10^5 - 10^8

2. Natural defense barriers:

- Gastric acidity
- Intestinal motility
- Mucus layer
- Intestinal commensal flora
- Humoral immunity (local IgA and systemic IgM and IgG)
- Cellular immunity (provided by lymphocytes and macrophages)

3. Adhesion of MICRO to intestinal mucosal cells

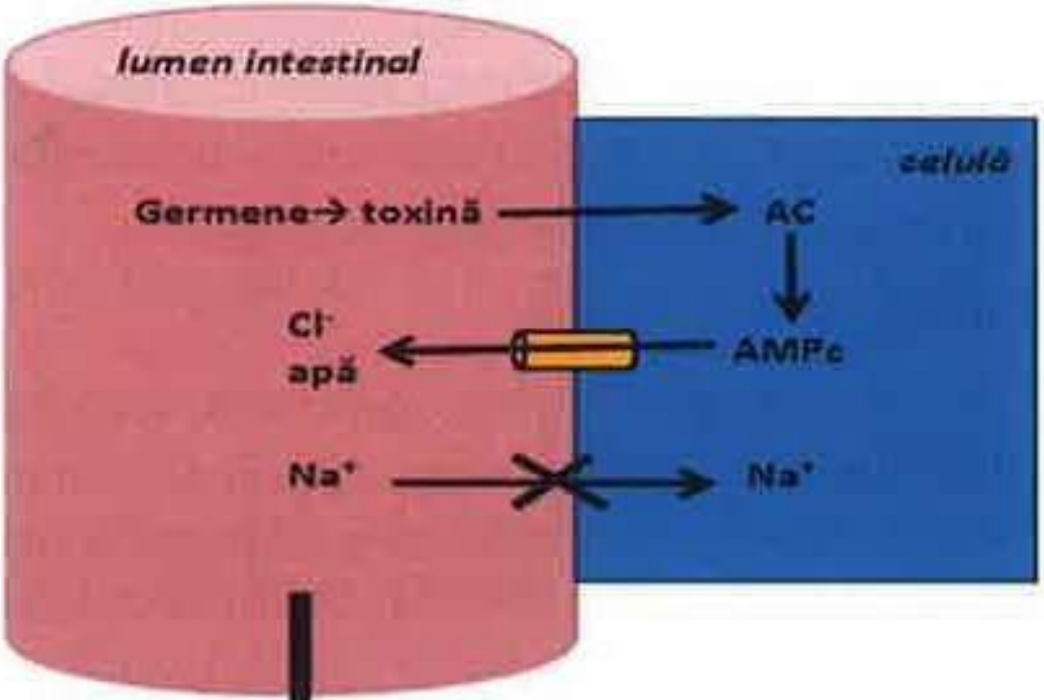
- Multiplication of infectious agents:
  - in the intesinal lumen (secretory-exudative mechanism)
  - intracellular (cytotoxic mechanism)
  - in the basement membrane (invasive mechanism)
  - Mixed

4. Toxicity and invasiveness:

- ***Enterotoxins*** – noninflammatory diarrhea by stimulating intestinal secretion (*vibrio cholerae, salmonella spp, s.aureus, clostridium perfringens, ETEC)*
- ***Cytotoxins*** - inflammatory diarrhea as a result of invasion of mucosal epithelial cells and destruction *(shigella spp, EHEC, clostridium difficile)*
- ***Neurotoxins*** with direct CNS or peripheral action (staphylococcus aureus, bacillus cereus).

Acute secretory diarrheal disease

Vibrio cholerae,
Staphylococcus aureus,
enterotoxigenic E.coli,
B.cereus, Cl.perfringens



Scaun voluminos

Watery diarrhea,
vomiting, no fever

dehydration

Cholera - acute diarrheal disease with pandemic potential, evolving into epidemics

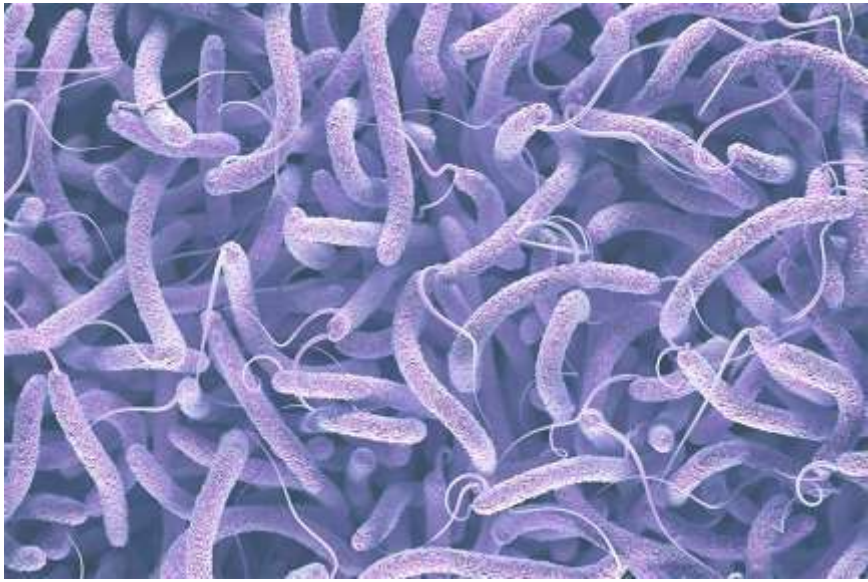
Etiological agent - ***Vibrio cholerae*****,** a gram-negative, aerobic bacillus, mobile by a single polar flagellum.

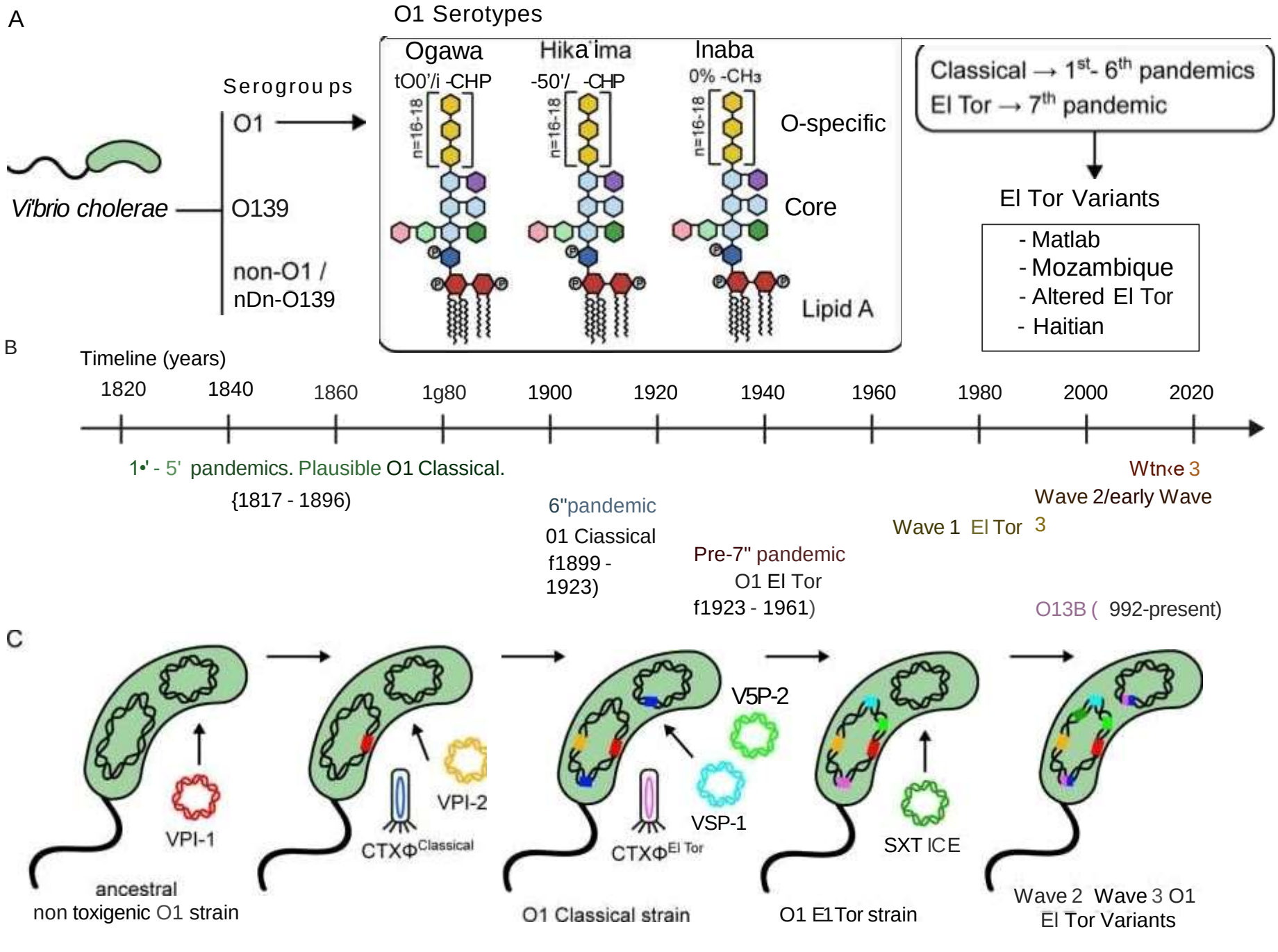
Flagellar antigen - H

Somatic antigen - O, according to which distinguish 3 serotypes:

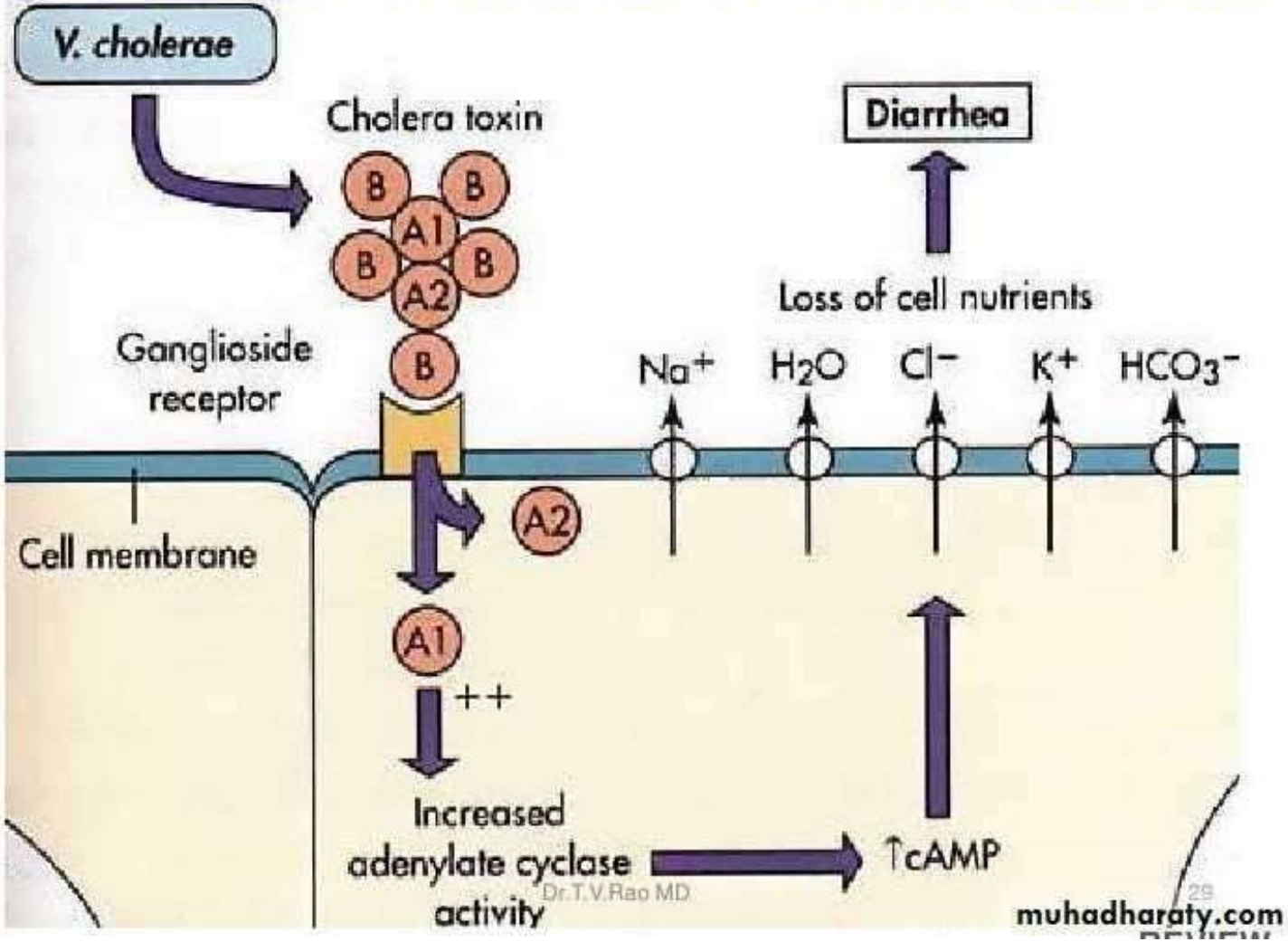
Ogava serotype (antigens A and C)
Inaba serotype (antigens A and B)
Hikojima serotype (antigens A, B, C)

Each serotype has 2 biotypes:
Classic biotype and **El Tor biotype**



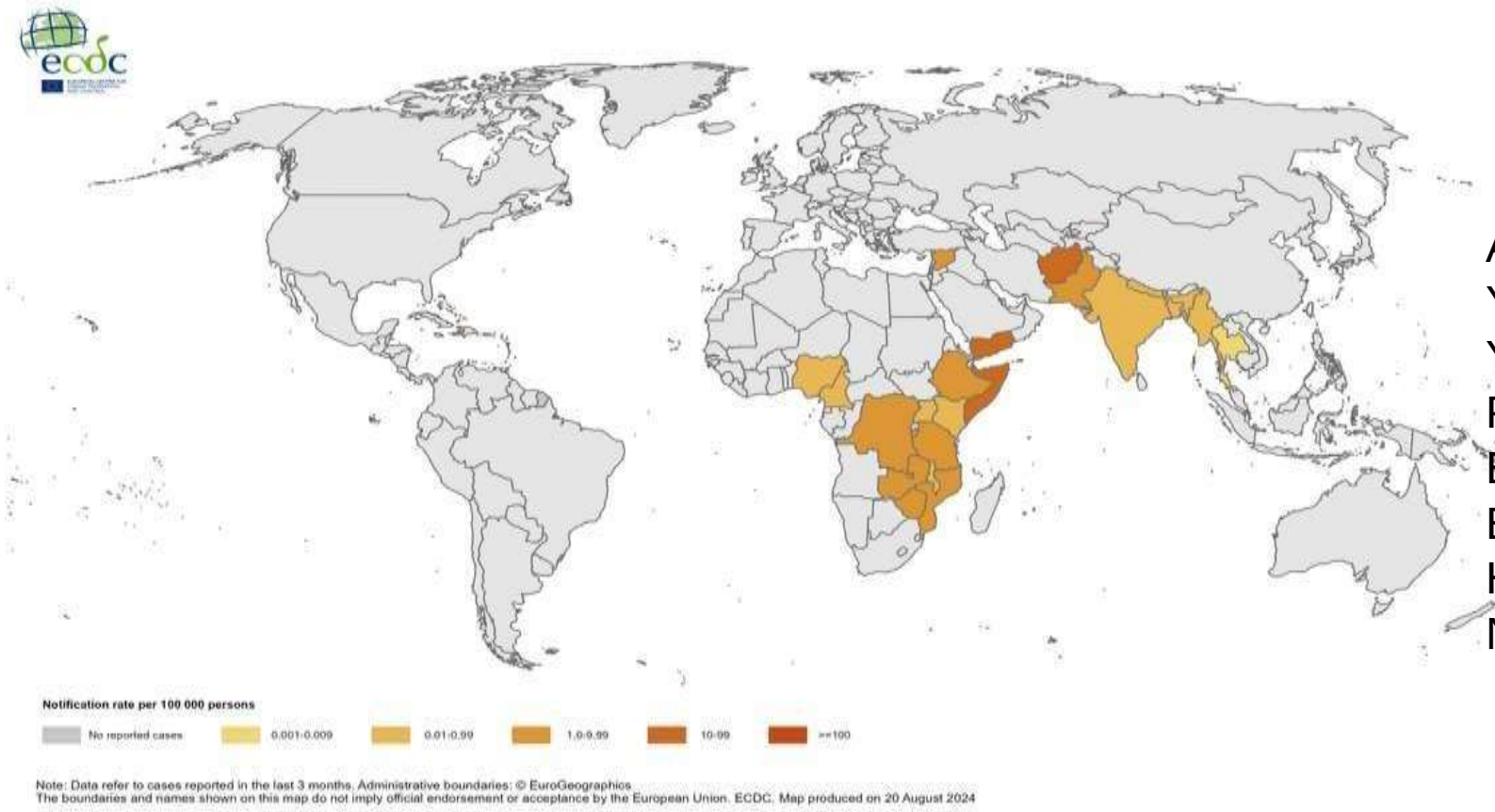

A
O1 Serotypes
Serogroups
O1
O139
non-O1 / nDn-O139
Vibrio cholerae
Ogawa
tO0'/i -CHP
Hika'ima
-50'/ -CHP
Inaba
0% -CH3
n=16-18
O-specific
Core
Lipid A
Classical → 1st- 6th pandemics
El Tor → 7th pandemic
El Tor Variants
- Matlab
- Mozambique
- Altered El Tor
- Haitian
B
Timeline (years)
1820
1840
1860
1g80
1900
1920
1940
1960
1980
2000
2020
1•' - 5' pandemics. Plausible O1 Classical.
{1817 - 1896)
6"pandemic
01 Ciassical
f1899 - 1923)
Pre-7" pandemic
O1 El Tor
f1923 - 1961)
Wave 1 El Tor 3
Wtnᵹe 3
Wave 2/early Wave
O13B (992-present)
C
VPI-1
ancestral
non toxigenic O1 strain
VPI-2
CTXΦClassical
CTXΦEl Tor
V5P-2
VSP-1
O1 Classical strain
SXT ICE
O1 E1Tor strain
Wave 2 Wave 3 O1
El Tor Variants

Mechanism of Action of Cholera Toxin



- Fluid losses occur in duodenum and the proximal jejunum, the ileum is less affected .
- The absorption in the colon is not affected .

- **Source of infection** – infected patients or chronic carrier
- **Transmission** - fecal-oral
- **Transmission factors** of cholera - water, contaminated food, dirty hands
- **Immunity after cholera** - serotype-specific, short-lived (2-3 months)



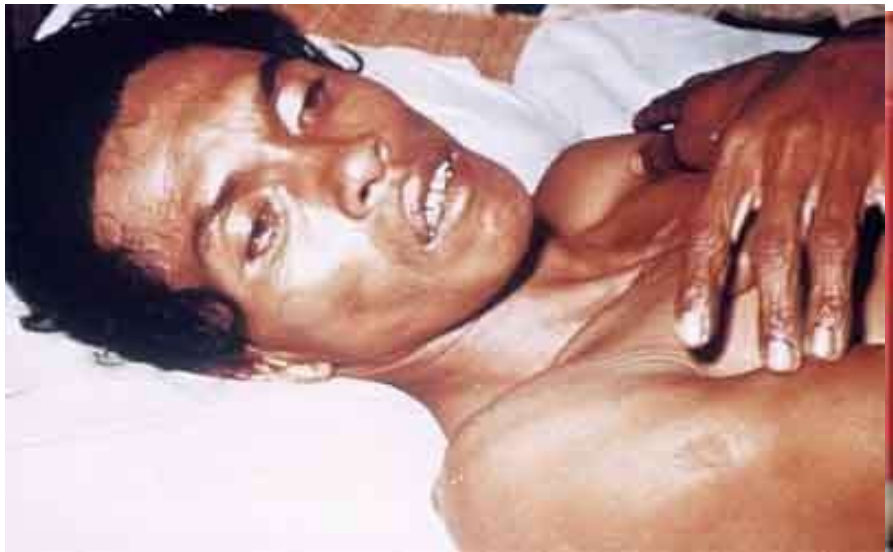
Afghanistan
Yemen
Yemen
Pakistan
Ethiopia
Ethiopia
Haiti
Nigeria

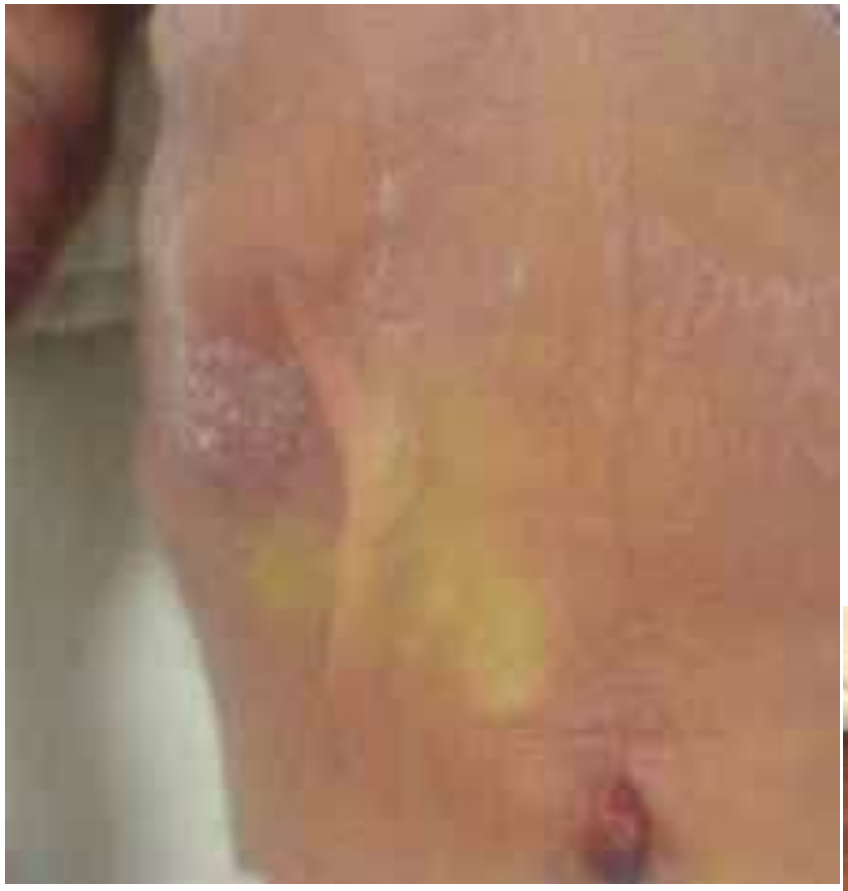
Clinical manifestation

Incubation period - 1-3 days

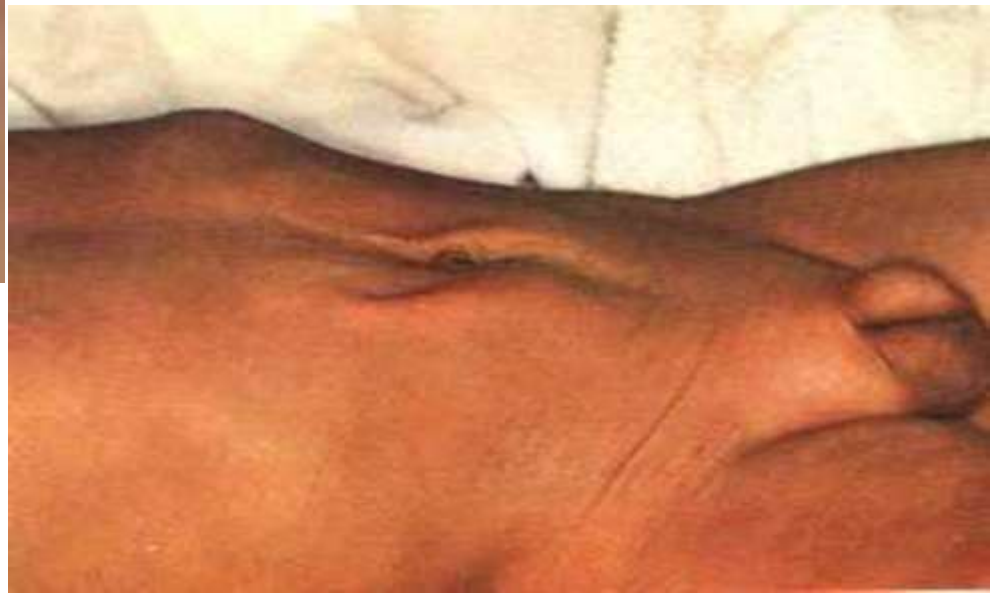
Period of clinical manifestation:

- **Asymptomatic forms**
- **Typical forms** – rice, watery diarrheal stools , painless abdomen, afebrile
- **Severe forms** - spontaneous vomiting without nausea, massive water loss and electrolytes, dehydration
- **Diarrhea sicca** - hypertoxic form of cholera, without diarrhea, paralytic ileus, abdominal distension, death



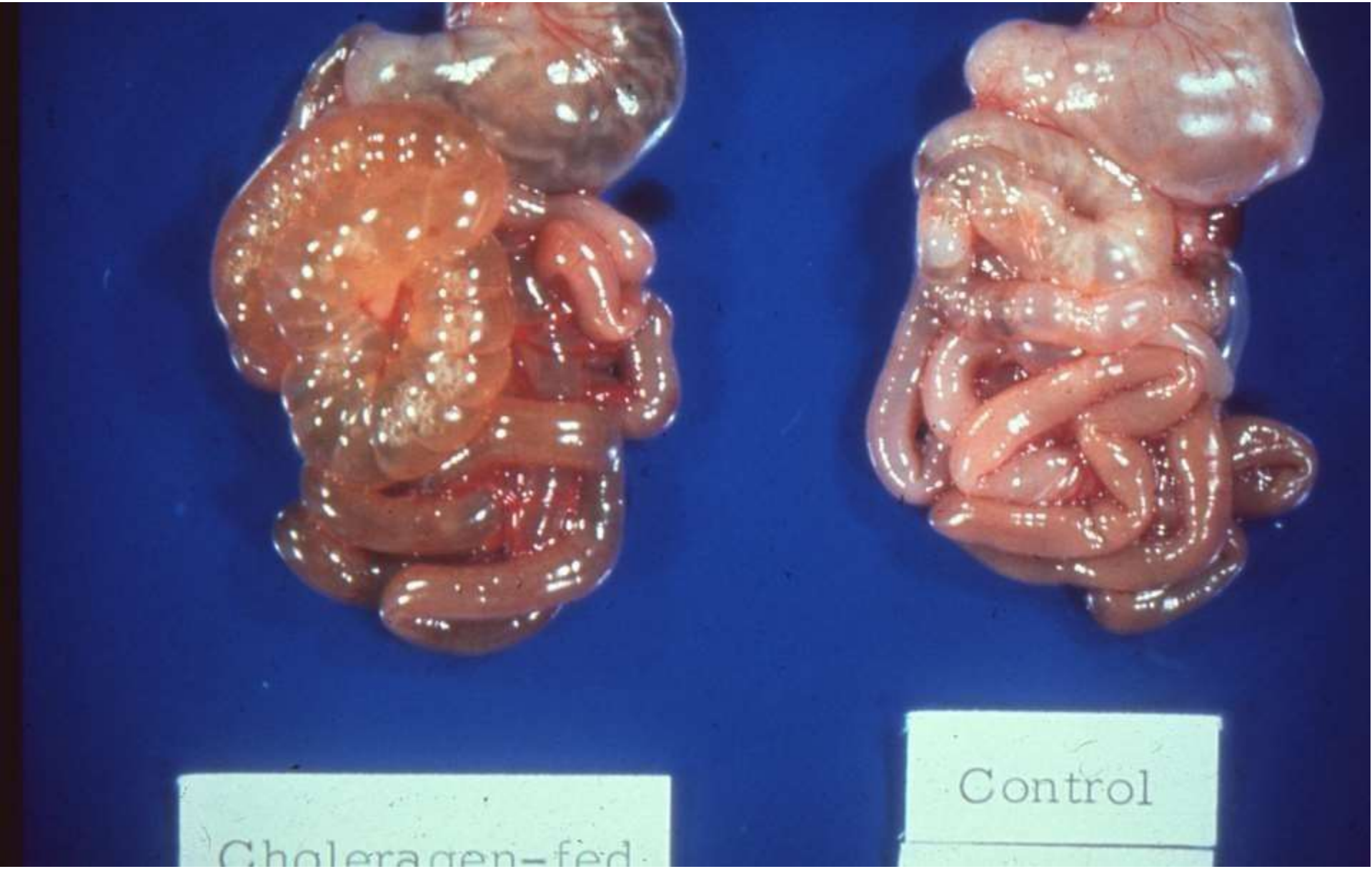


Persistent skin fold in an infant with SDA >10%



Circular eyes and dry mucous membranes in an infant with severe dehydration

Depressed anterior fontanel in a patient with severe dehydration


Control

Accepted classification of dehydration by percentage of fluids lost

	Mild	Moderate	Severe
Weight loss	Up to 5%	6-10%	More than 10%
Appearance	Active, alert	Irritable, alert, thirsty	Lethargic, looks sick
Capillary filling (compared to your own)	Normal	Slightly delayed	Delayed
Pulse	Normal	Fast, low volume	Very fast, thready
Respiration	Normal	Fast	Fast and deep
Blood pressure	Normal	Normal or low Orthostatic hypotension	Very low
Mucous memb.	Moist	Dry	Parched
Tears	Present	Less than expected	Absent
Eyes	Normal	Normal	Sunken
Pinched skin	Springs back	Tents briefly	Prolonged tenting
Fontanel (infant sitting)	Normal	Sunken slightly	Sunken significantly
Urine flow	Normal	Reduced	Severely reduced

Oral rehydration with WHO-recommended standard ORS

- For **adults** - **75 ml/kg in the first 4 hours**
- For **children** - up to **20 ml/kg/h**
- Reassess general condition in 1 hour, then after each stool administer ORS until normal hydration status is restored

Age	Quantity ORS
< 2 years	50-100 ml
2-9 years	100-200 ml
≥ 10 years and adults	How much it needs

Table 34.3 Composition of oral rehydration solution recommended by WHO

Solute	Grams/liter	Mmol/liter
NaCl	2.6	Na 75; Cl 65
Glucose	13.5	7.5
KCl	1.5	K 20
Trisodium citrate	2.9	Citrate 10
TOTALS	20.5	245 osmolality

I/V rehydration

If the patient is able to drink, ORS is administered until infusion.
In **children it is recommended 100 ml/kg /24h Ringer's Lactat solution** (or normal saline), divided as follows:
In **adults, up to 200 ml/kg/24h Ringer's lactate solution**

Age	Initially 30 ml/kg for:	Then 70 ml/kg for:
Children up to 1 year	1 hour	5 hours
Children over 1 year and adults	30minute	2 hours 30 minutes

➢ Reassess the general condition every 1-2 hours. If condition does not improve , he rehydration is repeated until BP normalizes and diuresis increases.

➢ In case of stabilization of blood pressure, vomiting stops, after 6 hours, children up to 1 year old and the rest after 3 hours can start rehydration with ORS.

Etiological treatment

	First-line	Alternative
Adults (including pregnant women)	doxycycline 300 mg p.o. single dose	azithromycin 1g p.o. single dose or ciprofloxacin 1g p.o. single dose
Children < 12 years old	doxycycline 2-4 mg/kg p.o. single dose	azithromycin 20 mg/kg (max 1g) p.o. single dose, or ciprofloxacin 20 mg/kg (max 1g) p.o. single dose

Staphylococcus aureus food poisoning

- Bacterial disease caused by an enterotoxin elaborated outside the host.

- Most cases of staphylococcal food poisoning are caused by contamination from infected human carriers or people with staphylococcal skin lesions.

- Outbreaks following picnics where potato salad, mayonnaise, and cream pastries have been served offer classic examples of staphylococcal food poisoning.

- Has the shortest incubation period (1–6 h) and generally lasts <12 h.

- **Clinical manifestations**

- Disease starts with nausea and vomiting, rarely diarrhea (may miss) and abdominal pain.

- Fever is absent or low-grade.

- **Treatment of choice**: Enterofuril, Nifuroxazid. Antibiotic therapy is not recommended.

- **Pathogenetic treatment** is adequate rehydration.

Gram-Positive Rods: Bacillus

B. Cereus

- Environmentally ubiquitous
- Beta-hemolytic
- Motile

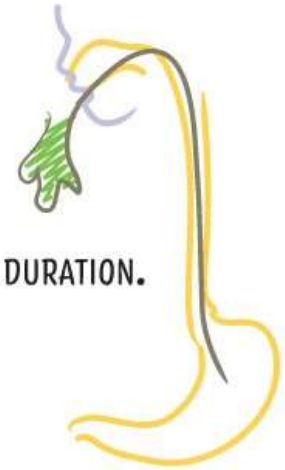
Gastroenteritis

Emetic

Heat-stable toxin

- Rice

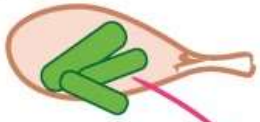
- Nausea, abdominal cramps, vomiting.
- Quick Incubation & duration.



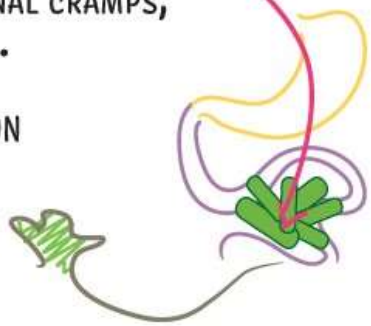
Diarrheal

Heat-labile enterotoxin

- Meat, vegetables



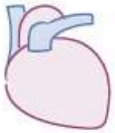
- Nausea, abdominal cramps, watery diarrhea.
- Longer incubation & Duration.



Ocular Infection

- Rapid progression, eye loss.
- Clindamycin/Vancomycin

Severe Pneumonia & Others

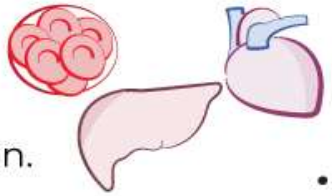


- IV catheter, CNS shunt infections Endocarditits, bacteremia, meningitis

Gram-Positive Rods: Clostridium

C. perfringens

- Spores rarely seen clincially.
- Flat, irregular colonies.
- β-hemolytic.
- Subtypes based on toxin production.



Alpha toxins (all types):
Hemolysis, vascular leakage, liver toxicity, cardiac dysfunction.

- Myonecrosis: Gas Gangrene

Necrosis & gas bubbles

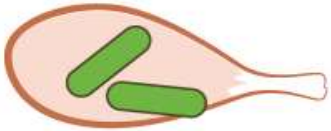
Other toxins: Pore-forming; necrosis.

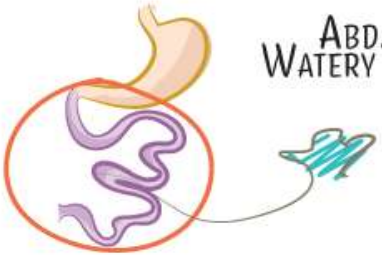
- Soft tissue infections:
  Cellulitis, fasciitis, myositis, myonecrosis.
- Rx: Antibiotics, surgical debridement

Enterotoxin: Fluid & ion loss (superantigen).

- NO antibiotics.
- Rehydration.
- Self-limiting disease.

Food poisoning





Abd. cramps,
Watery diarrhea

Clostridium Perfringens food poisoning

- Is widely distributed in feces, soil, air, and water.
- Contaminated meat has caused many outbreaks.
- **C. perfringens** spores survive cooking, can germinate and multiply, resulting in large numbers of bacteria when cooked meat that is contaminated is left at room temperature or even up to 60° C for a period of time.
- Once inside the gastrointestinal tract, C. perfringens produces an ***enterotoxin*** that acts on the small bowel.
- Only ***C. perfringens type A*** has been definitively linked to this food poisoning syndrome.
- Mild gastroenteritis is most common, with onset of symptoms 6 to 24 hours after ingestion of contaminated food.
- The most common symptoms are ***watery diarrhea and abdominal cramps***. Vomiting and fever are unusual.
- Symptoms typically resolve within 24 hours; severe or fatal cases rarely occur.

Escherichia Coli food poisoning

1*. Enterotoxigenic E. Coli (ETEC) and 2. Enteroaggregative E. Coli (EAEC)* - causes watery diarrhea and is often found in food and water in areas with poor sanitation. This is the type most responsible for traveler's diarrhea.

Clinical manifestation:

- Watery diarrhea, self-limiting in 2-4 days, mild and moderate forms
- Abdominal pain, nausea, vomiting
- Fever or underfebrile

3. *Enterohemorrhagic E. Coli* produce Shiga toxin, responsible for hemorrhagic colitis.

- In children diarrhea can be complicated by **hemolytic uremic syndrome** characterized by triad: *hemolytic anemia, thrombocytopenia and acute renal failure*

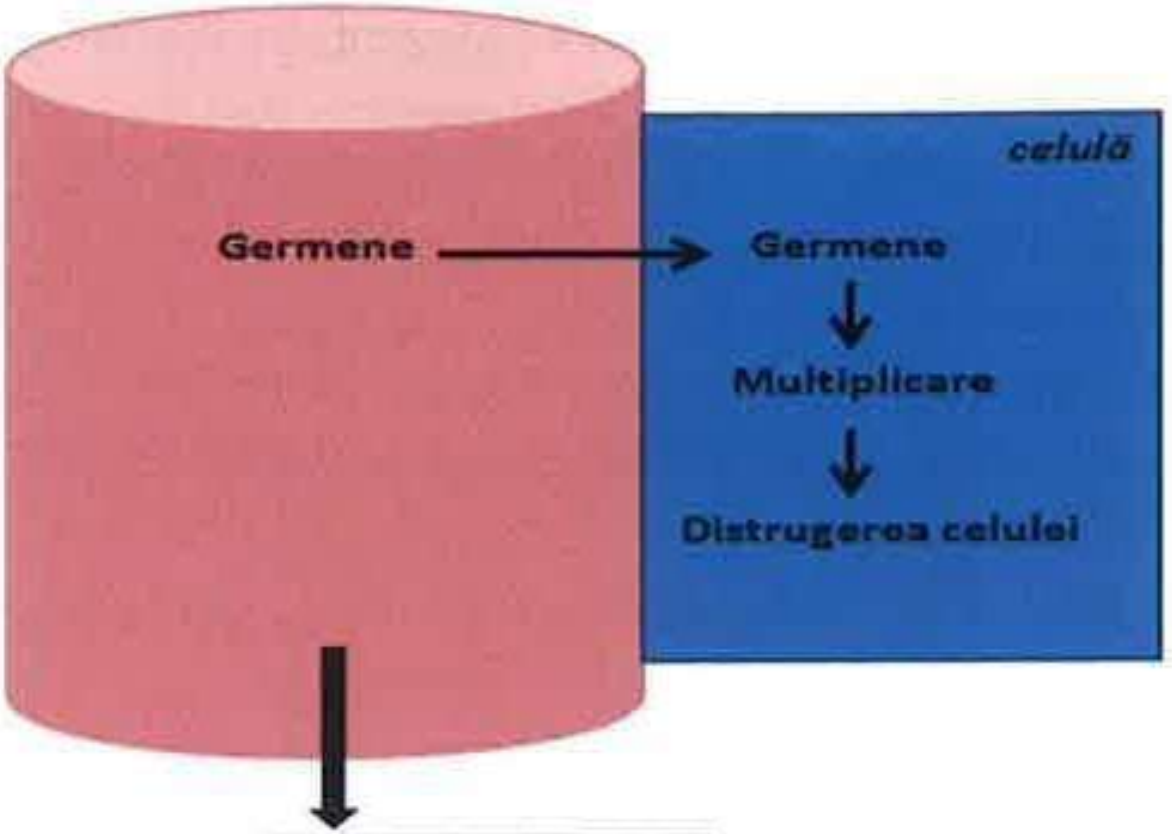
Diagnosis: 1. Coproculture
2. PCR identifies the ***shiga-like toxin*** in the stool

Treatment: adequate rehydration .

Mild and medium forms - antibiotic therapy not recommended *Severe forms - ciprofloxacin, cotrimoxazole, azithromycin*

Acute cytotoxic diarrheal disease

Salmonella, shigella,
Campylobacter jejunii,
Y.enterocolitica



Diarrhea with mucus and blood,
Abdominal pain

Febră

Risk of sepsis

Salmonellosis

Most common serotypes of ***non-typhi salmonellae***: *S. typhi murium,* ***S. enteritidis****, S.* ***enteritidis****, S. panama, S. infantis, S. newport, S. agona, S. derby, S. London, etc.*

Develops by **mixed mechanism**: cytotoxic + secretory

Incubation period - 6-36 hours (more commonly up to 24 hours)

Source of infection - sick birds and animals, sick or chronically carrier humans

Transmission:

- Duck or chicken eggs, foods prepared from raw eggs (creams, mayonnaise, ice cream)
- Undercooked meat, fish, shellfish
- Raw milk

Clinical manifestation:

Fever 38-40*C

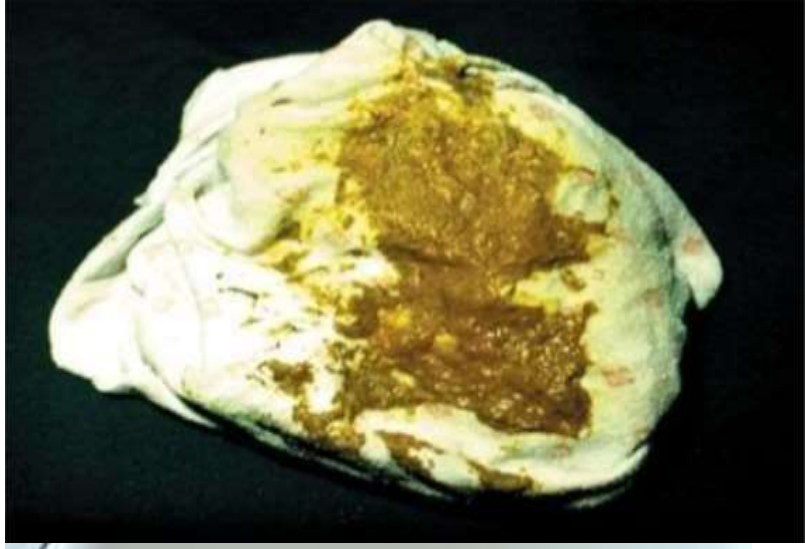
Frequent, watery, green diarrhea, with foam a fetid smell

Nausea, vomiting, abdominal pain

Localized gastrointestinal forms - gastritis, gastroenteritis, gastroenterocolitis and enterocolitis

Generalized forms with *typhoid* and *septic evolution*

Intestinal carriage: acute, chronic and transient



Hemocolitis in salmonella infection

Treatment:

Mild and moderate forms: hydroelectrolyte rebalancing, dietary treatment, antibacterial nitrofuranic drugs(Enterofuril, Nifuroxazid, Furazolidone)

Severe forms: Antibiotic therapy

Bacterial dysentery

Etiological agent - *Shigella* 4 species (serogroups)

- *Sh. flexneri* - in developed countries
- *Sh. sonnei* - in developing countries
- *Sh. boydii* - has a very low pathogenicity and spread.
- *S. dysenteriae* - causes epidemics

- *Sh. dysenteriae serotype 1* produces an exotoxin - **Shiga toxin** - associated with severe forms of the disease due to its neurotoxic, enterotoxic and cytotoxic effects.

- **Shiga toxin** is translocated from the intestines into the circulation, with binding to target cells in the kidneys, following the pathophysiological changes characteristic of **hemolytic-uremic syndrome**: *hemolytic anemia, thrombocytopenia and acute renal failure*.

Bacterial dysentery

It is endemic disease in developing countries

Source of infection - sick people

Transmission mechanism - fecal-oral

Ways of transmission: water, contaminated food or dirty hands

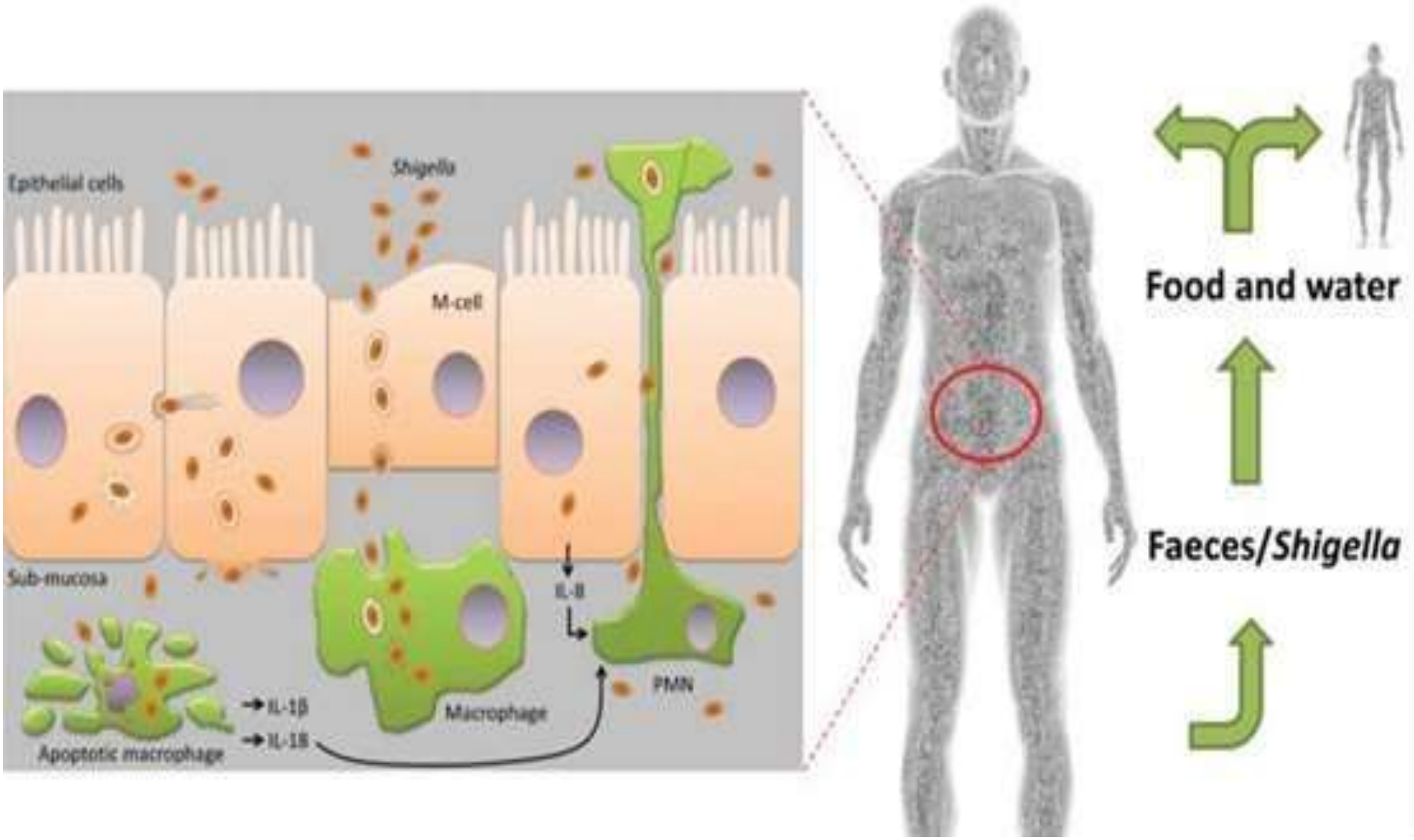
Receptivity to the disease is general

Disease leaves no immunity

Infection dose is low: < 200 units

Pathogenesis in Shigellosis

Mechanism - cytotoxic (inflammatory)



- ***Shigela*** penetrates and multiplies inside the intestinal mucosa → cell destruction → inflammatory phenomena

- It mainly affects the sigmoid and rectum

- Mucosal inflammation occurs with necrosis and ulceration

- Decreases mucus synthesis

- Menteric mucous plexuses are affected and submucosa, the shy nerve endings → sensation of imminent defecation and rectal tenesmus

- The stools in dysentery are low in quantity, affecaloid and contain mucus, pus and blood

Clinical manifestation

Incubation period: 1-3 days

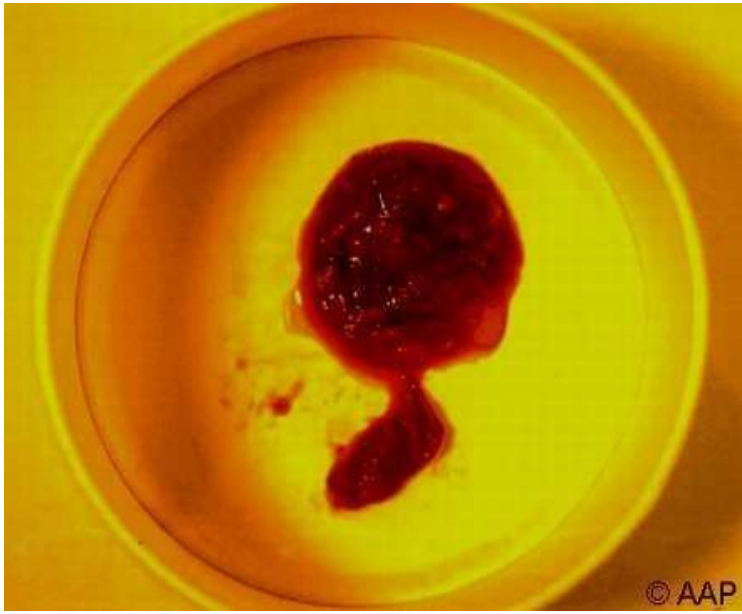
Sudden **onset** with: fever, chills, spastic abdominal pain, nausea, vomiting, severe rectal tenderness

Frequent, affecaloid **stools** contain mucus, pus and blood ('rectal sputum')

On palpation of the abdomen - colonic chord

Treatment:
Etiotropic antibiotics: Ciprofloxacin, Rifaximin, Ceftriaxone

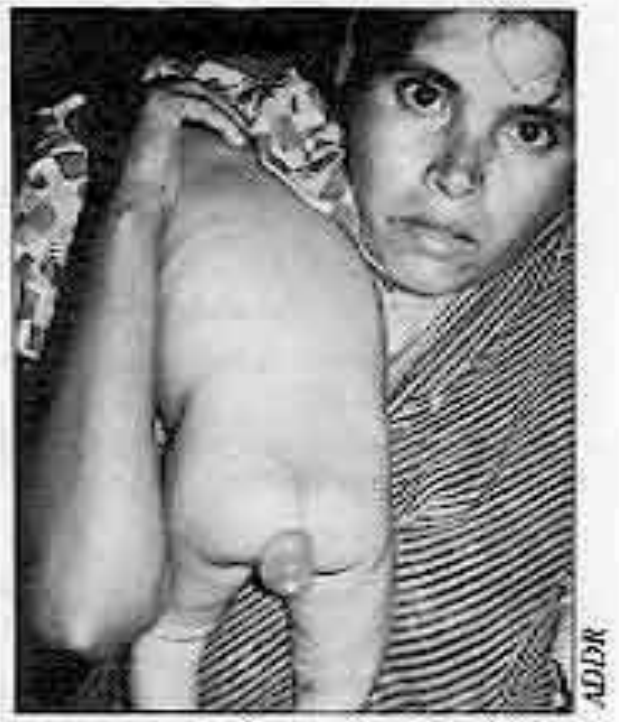
Pathogenetic: hydroelectrolyte rebalancing
Symptomatic: antispasmotics, antipyretics



Rectal sputum affecaloid stool

Possible complications In severe forms:

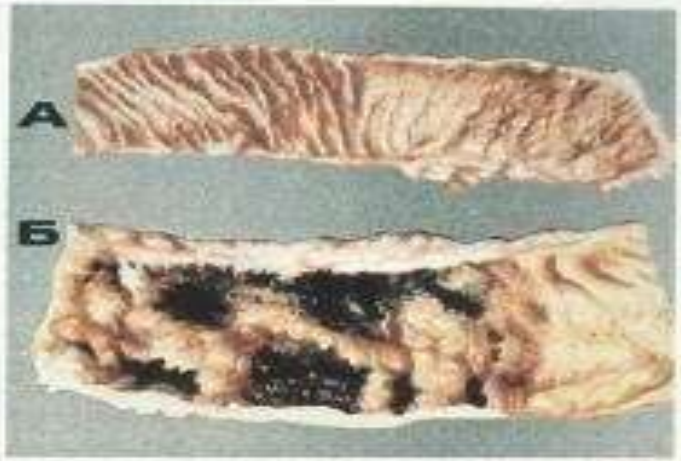
- neurotoxicosis and cerebral edema in children;
- toxicosis with exicosis; toxic-infectious shock;
- intestinal bleeding;
- intestinal perforation, peritonitis;
- paraproctitis;
- colonic dysbacteriosis;
- urinary tract infections;
- toxic megacolon;
- hemolytic uremic syndrome,
- rectal prolapse.



Inflammation and tissue damage causes painful straining to pass stools, which can lead to rectal prolapse.

- **Complications secondary to** chronic intestinal lesions: malabsorption syndrome and cachexia.
- **Remote complications** after apparent cure: chronic colitis; dyspepsia Rebellious; autoimmune reactive arthritis.
- There is the possibility of progression to **chronic or relapsing forms of** the disease in 3-4% of untreated cases.

1



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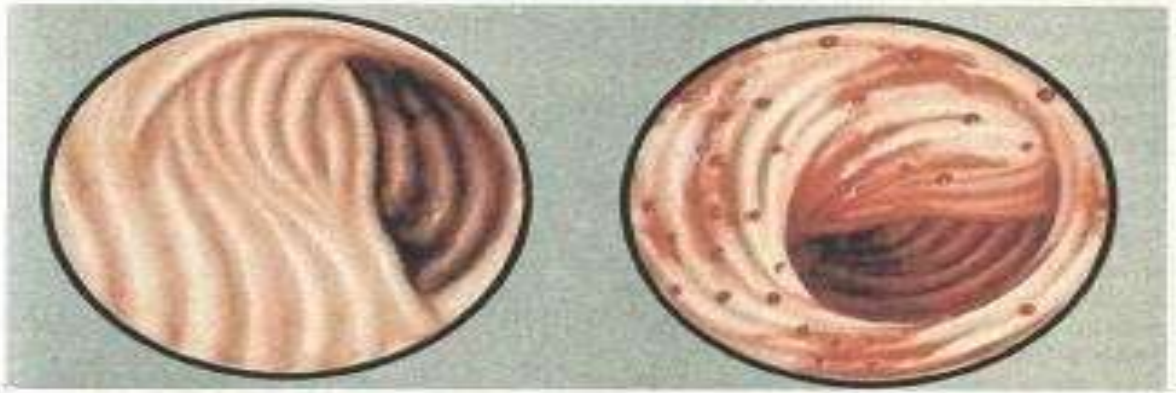

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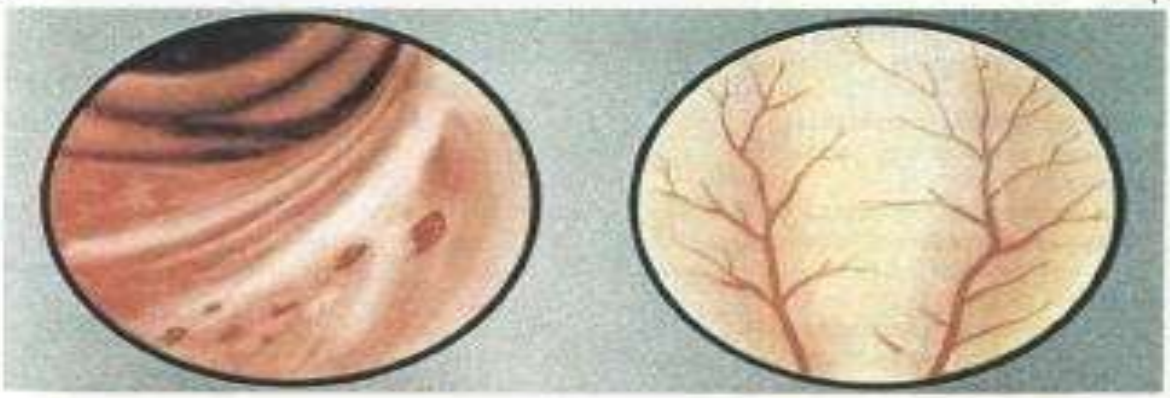
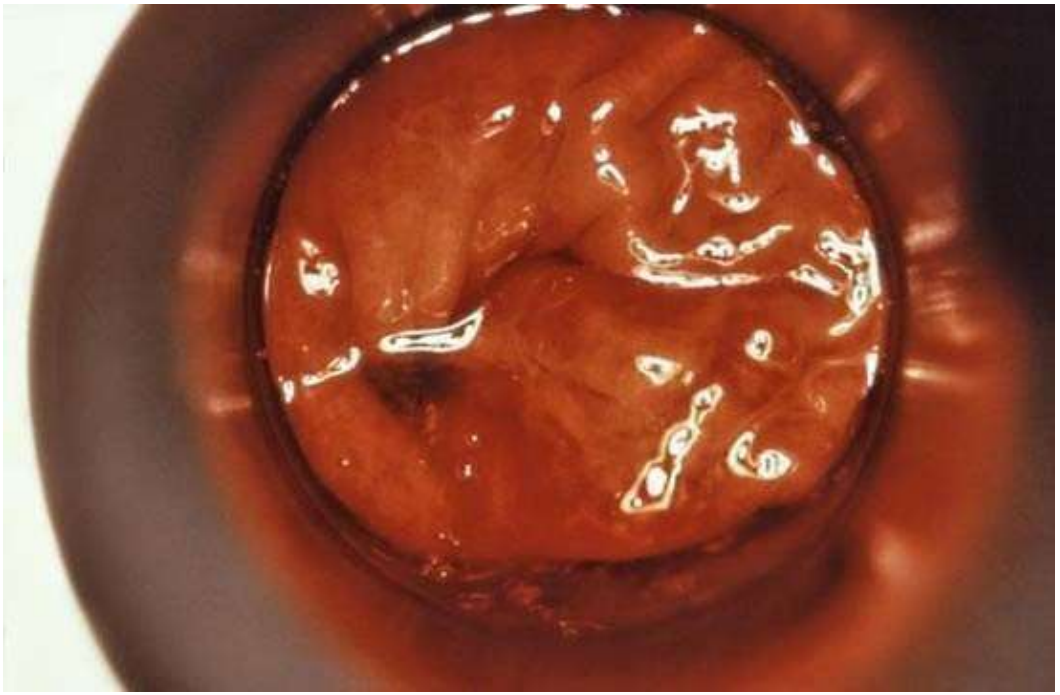
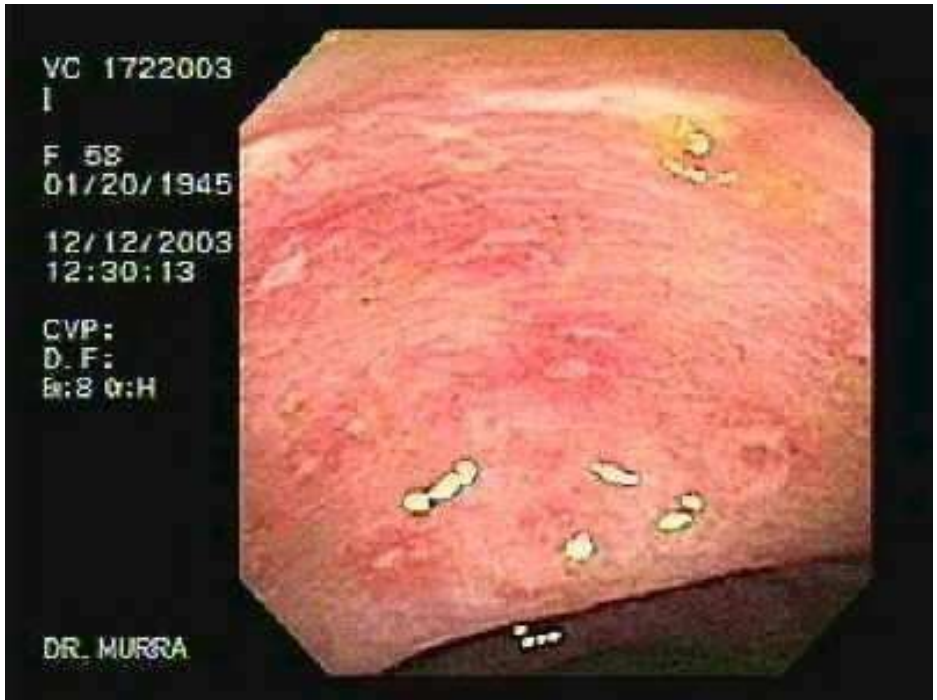
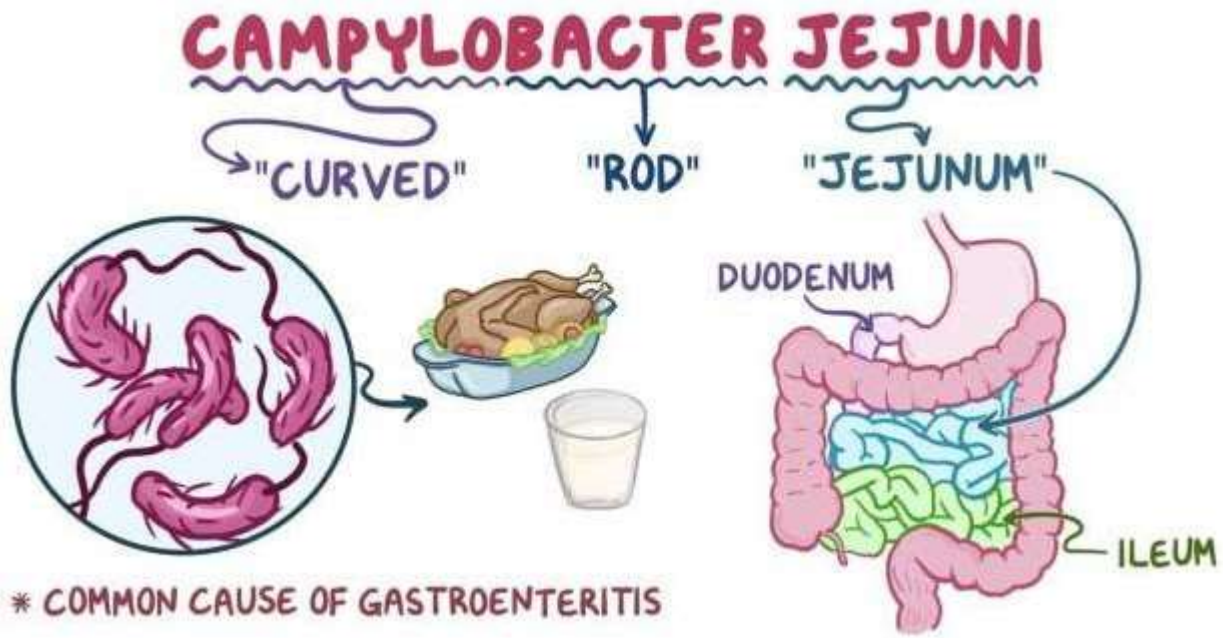




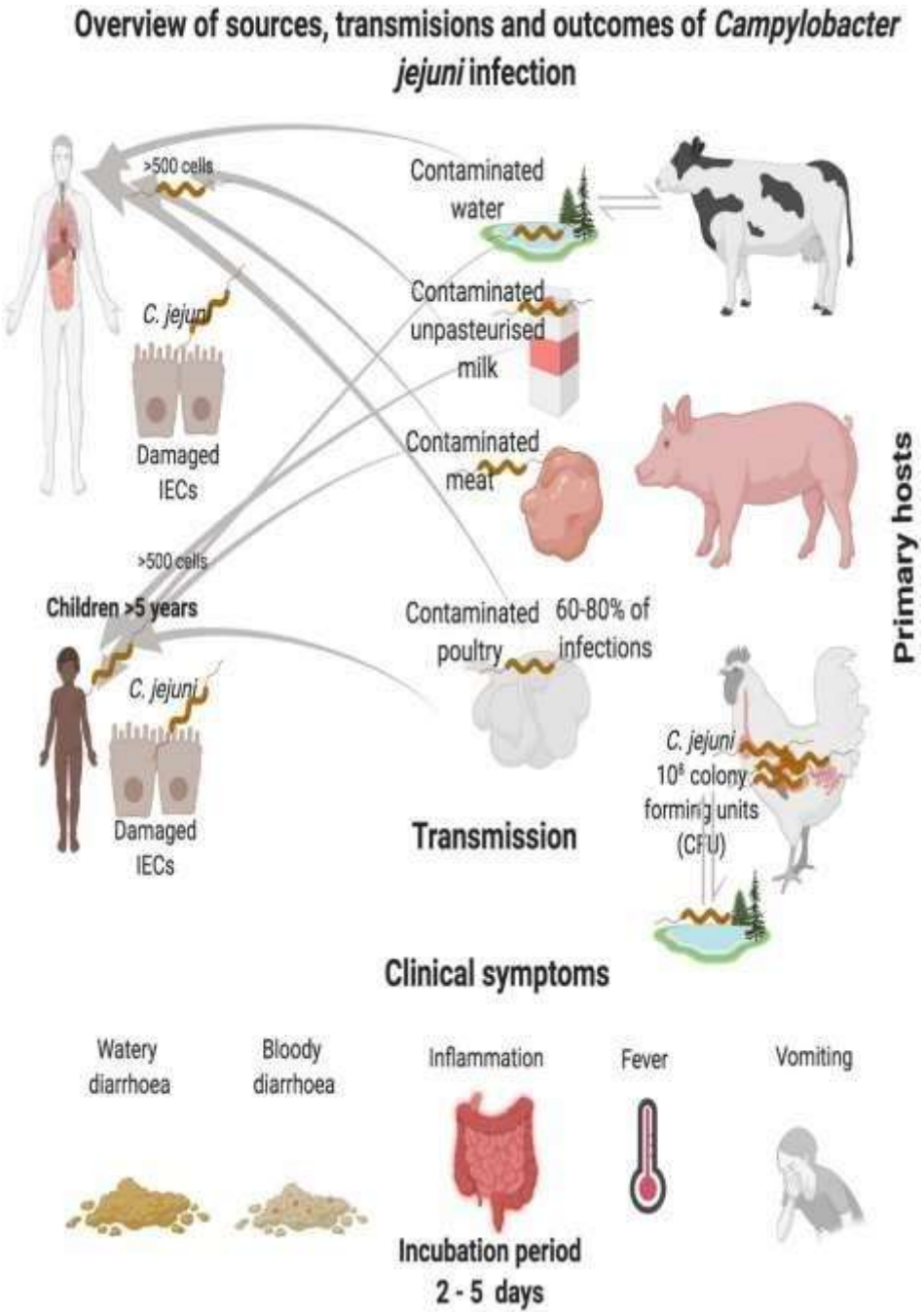


Instrumental investigations - rectoromanoscopy:
signs of inflammation of the distal colon (proctosigmoiditis in mild catarrhal or catarrhal-hemorrhagic form,
erosive, ulcerative and fibrinous - in severe forms).

	cholera	dysentery	salmonellosis
The chair	Aqueous, abundant, often colorless	Poor, with a mixture of mucus and streaks of blood	Green with undigested debris, sometimes colorless
Defecation	painless	tenesmus	Tenesmus can be
Pain in abdomen	-	+	+
Dehydration grade III-IV	+	-	-
Chill	-	+	+
Body temperature	Normal or low	grown	grown
Blood pressure	Low	Moderately low or normal	Moderately low or normal
Debut	With diarrhea	Often begins with vomiting, then diarrhea	Often begins with signs of infectious impregnation, then vomiting and diarrhea
Abdominal noise	+	-	-
Sigmoid painful spasms	-	+	+
Haemoconcentration	expressed	missing	missing
Oligoanuria	expressed	More often missing	More often missing



- Campylobacter is the main cause of bacterial food-borne gastroenteritis in the world.
- C. jejuni is responsible for 80%-90% of diagnosed Campylobacter infections.
- A feeder of birds and animals
- 50-70% chicken or turkey
- It mainly affects the jejunum and ileum, but can extend to the colon and rectum



- The flagellum ensures motility and secretion of proteins that favor mucosal invasion.
- Plasmid pVir is associated with bloody stools
- Mebrane contains antigen O with endotoxic activity.
- Some O antigens contain sialic acid involved in the pathogenesis of the syndrome Giullain-Barre.

Front. Cell. Infect. Microbiol., 03 February 2021
Sec. Molecular Bacterial Pathogenesis

Clinical manifestation

- **Incubation period** 12-24 hours.
- Fever, enterocolitis, watery stools, hematochezia.
- Colicky abdominal pain relieved by defecation.
- **Complications:**

  Bacteremia (1%),

  Toxic megacolon

  Pseudoappendicitis

  Peripheral SN involvement (Guillain Barre Syndrome)

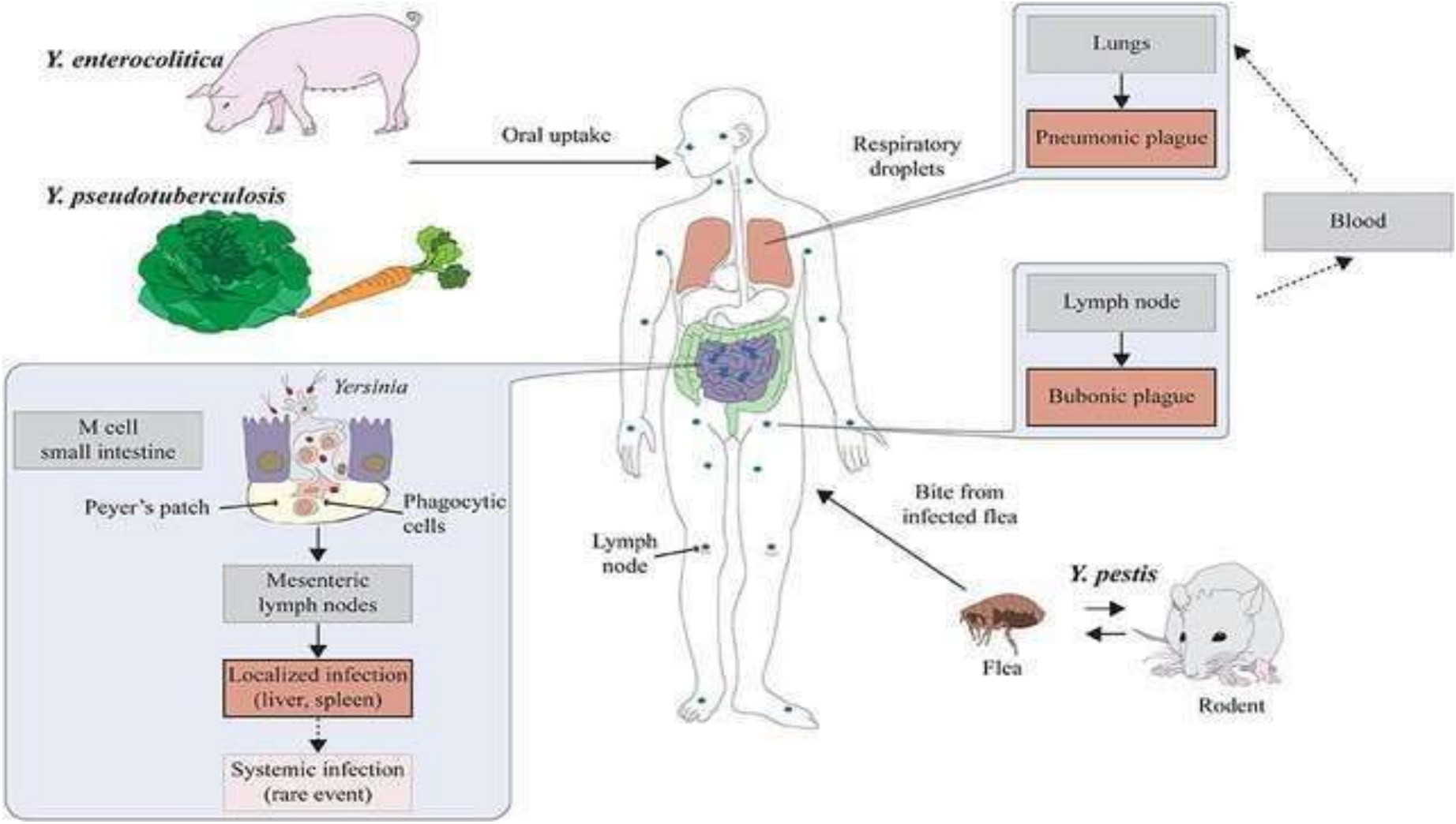
  Reactive arthritis (Reiter's syndrome)

  Bacteremia with secondary localizations

- **Treatment:**
- Naturally resistant to cephalosporins.
- Recommended azithromycin or erythromycin, amoxicillin/clavulanic, nitrofurans.
- Increasing resistance to fluorquinolone.

Yersinioza

- develops at T +4ºC, so in refrigerated foods
- seasonality - winter



SURSA: Coregulation of host-adapted metabolism and virulence by pathogenic yersiniae October 2014Frontiers in Cellular and Infection Microbiology 4(146):146DOI: 10.3389/fcimb.2014.00146

Clinical manifestation

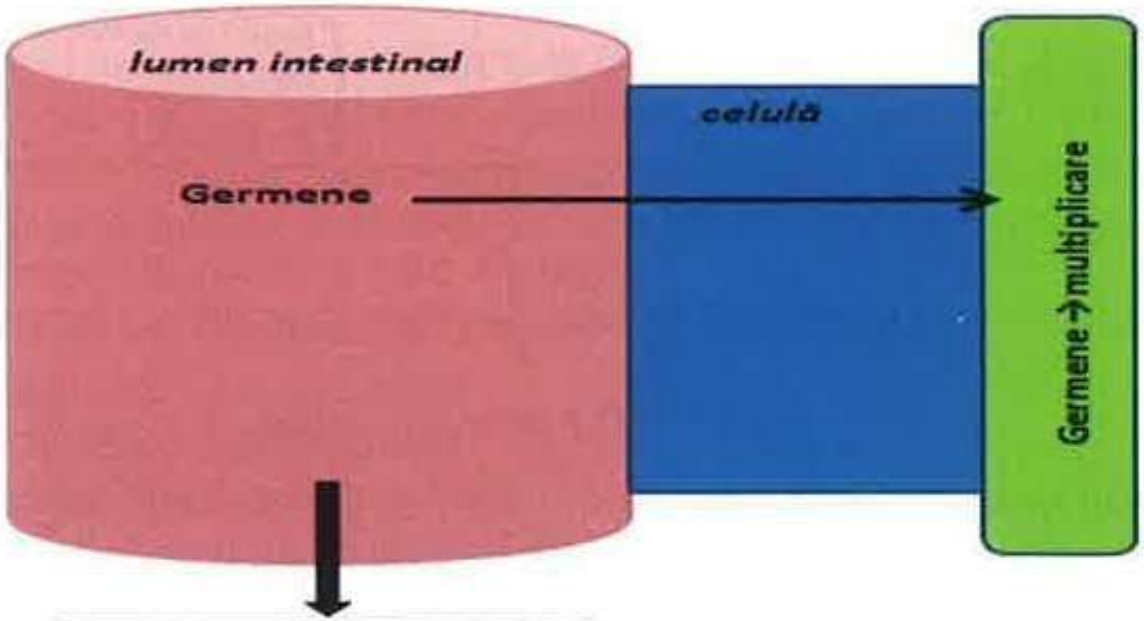
- Incubation period: 4-7 days
- ***Yersinia enterocolitica*** usually causes an acute febrile enteritis, rarely ulcerative colitis. Diarrhea may be bloody.
- ***Yersinia pseudotuberculosis*** - pseudoappendicular picture with ileitis, mesenteric adenitis, intussusception, ileocecal pseudotumor, ileum perforation.
- Persist 1-4 weeks
- At 2-20 days 1/3 patients erythema nodosum at the trunk or lower limbs.
- Another 15% are complicated by reactive polyarthritis
- Rare complications: mesenteric artery thrombosis and intestinal necrosis.

- **Diagnosis:**
- Coproculture - positive within 2 weeks of onset.
- IgG and IgA antibodies - in chronic inflammatory and reactive arthritis.
- PCR has higher sensitivity than bacteriologic method

- **Treatment:**
- Ciprofloxacin, Cotrimoxazole and Doxycycline in usual doses for 7 days,
- in severe forms - Cephalosporins gen.III and Gentamicin

Invasive BDA

Salmonella typhi and paratyphi



Small volume diarrhea, fever, signs of impregnation

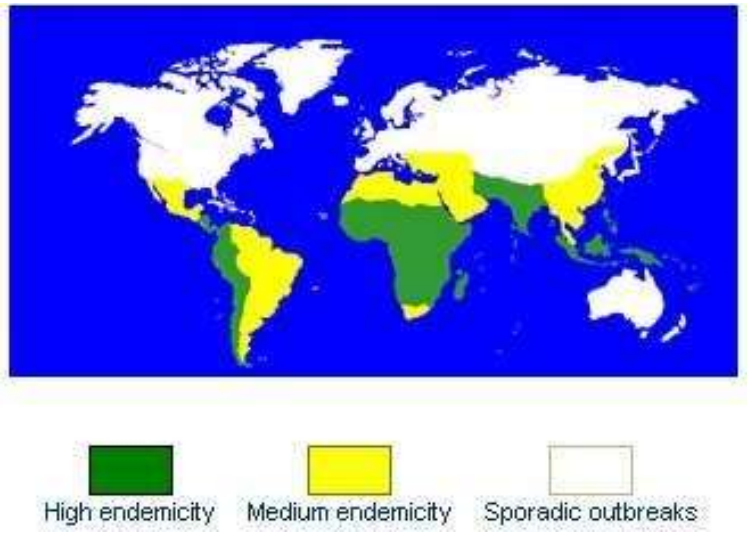
Risk of sepsis

After Gavriliu L-C.

Typhoid fever

- Acute systemic infectious disease characterized by bacteremia, prolonged fever, typhoid, digestive disorders, splenomegaly

- Etiological agent - ***Salmonella typhi*, *Salmonella paratyphi A*, *B or C***
- It is gram-negative, aerobic, motile, flagellate bacillus
- It is resistant in the external environment
- In South-East Asia, sub-Saharan Africa, sub-Saharan South - endemic with epidemic outbreaks



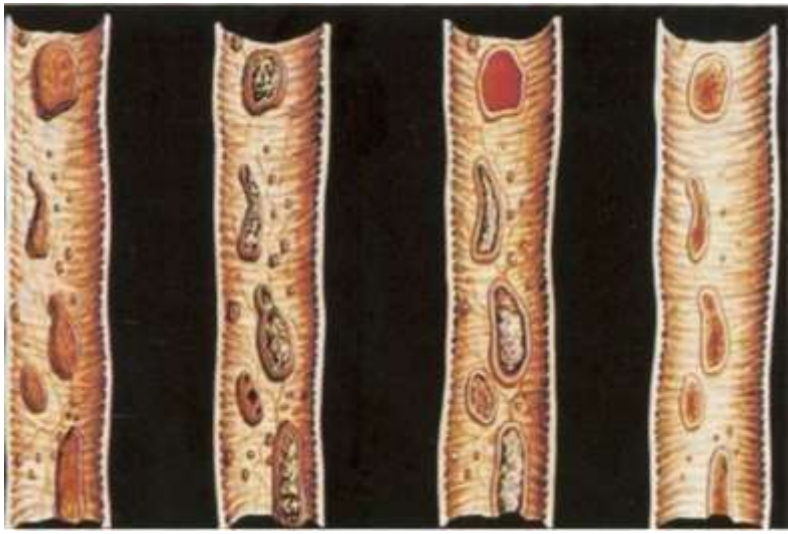
- **Source of Infection** – sick people with TF, asymptomatic chronic carriers
- **Transmission** - fecal-oral
- **Routes of transmission** - direct contact through dirty hands or indirect contact - through contaminated food or water

- **Immunity after illness** - durable but not absolute

Pathogenesis

Ingestion of the bacterial inoculum (10^5 -10^9 bacteria) → gastric barrier → multiplication at the ileo-cecal level, penetrate lymphoid tissue and Peyer's patces.

- **Primary bacteremia** → reticulo-endothelial system (liver, spleen, bone marrow), where replication occurs for 10-14 days (incubation period)
- **Secondary bacteremia** → systemic dissemination ≈ (I week FT) (Peyer's patces hyperplasia, spleen, gallbladder and bile duct involvement)
- II week ≈ necrotic lesions
- III week ≈ ulcerative lesions
- IV week ≈ scarring changes
- S.typhi can remain in cholecyst, kidneys - explains relapses occurrence
- ***SalmonellaeTyphi*** release **endotoxin** - explains fever, myocarditis, toxic hepatitis, meningoencephalitis, hematologic manifestations (leukopenia, thrombocytopenia), Disseminated intravascular coagulation



I week **II week** **III week** **IV week**

Clinical manifestation

Onset of illness (1st week of illness)

- *Fever gradually rises to 40°C*
- *Frontal headache, insomnia, abdominal pain*
- *Constipation, rarely diarrhea*
- *Pulse-temperature dissociation*

Period of illness (II week of illness)

- *High fever, 39-40°C, remains on the plateau*
- *Severe headache*
- *Typhoid rash (maculopapular, on abdomen and chest), appears on day 10 of illness*
- *Digestive manifestations:* edematous tongue with dental impressions

Catarrhal tonsillitis (Duguet's tonsilitis)

Abdominal pain, constipation, bloating, intestinal paresis , Pea-like diarrhea or constipation

Hepatosplenomegaly

- ***Respiratory*** *manifestations: bronchitis or pneumonia*
- ***Cardiovascular*** *manifestations: hypotension, bradycardia*
- ***Neurological*** *manifestations: typhoid-like state with drowsiness, confusion, obnubilization,*
- ***Hematologic*** *manifestations: anemia, leukopenia, thrombocytopenia*

Clinical manifestation

Period of endotoxin complications (3rd week of illness)

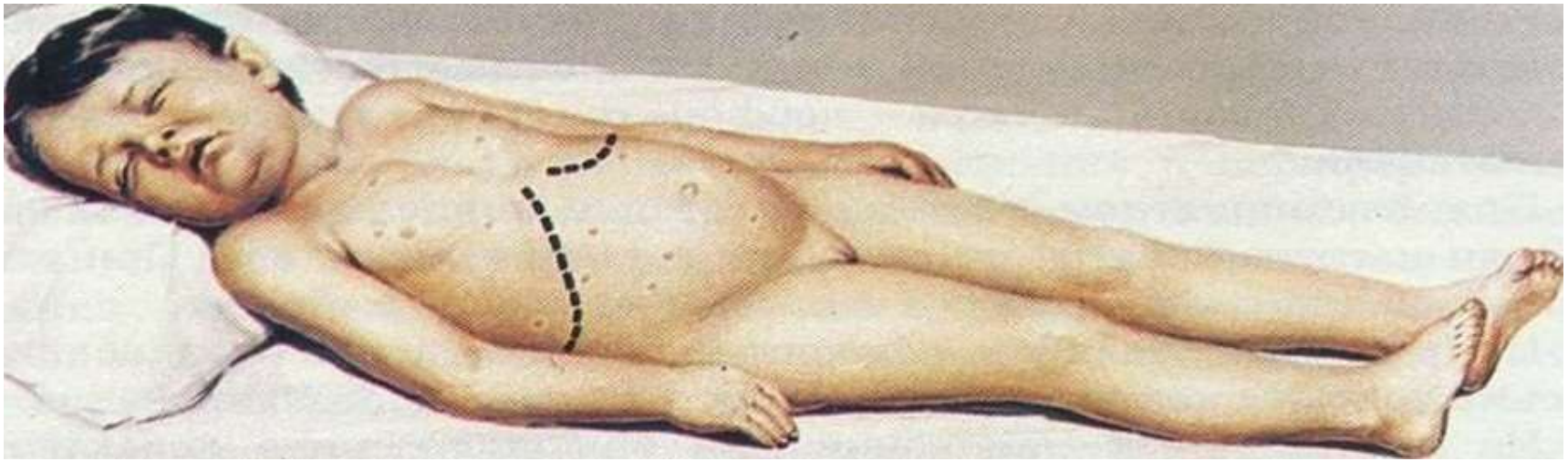
- *Tachycardia setting in* place of bradycardia
- *Digestive complications*: bleeding, intestinal perforation, melena
- *Heart complications*: myocarditis
- *Neurological complications*: altered consciousness, severe encephalitis
- *DIC*

Period of decline (IV week of illness)

- Fever gradually decreases
- The typhoid situation is regressing
- Appetite is back

The convalescence period - is long

Chronic (biliary) carrier - 1-5% of all infections



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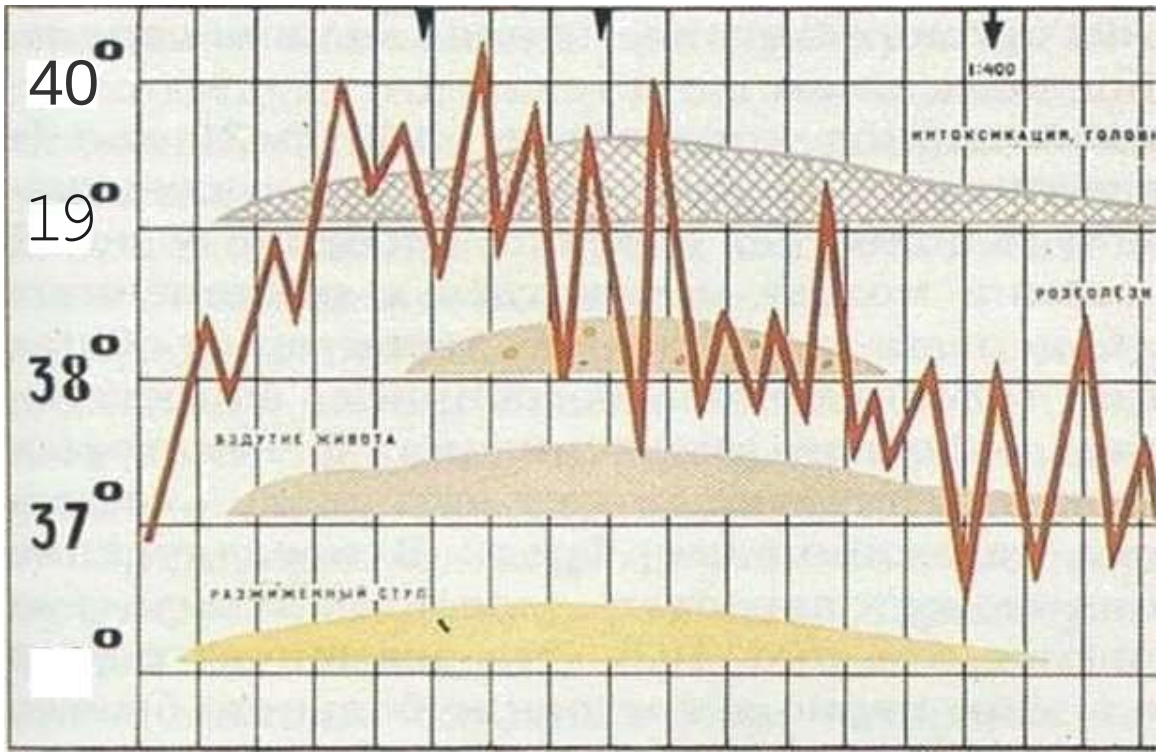
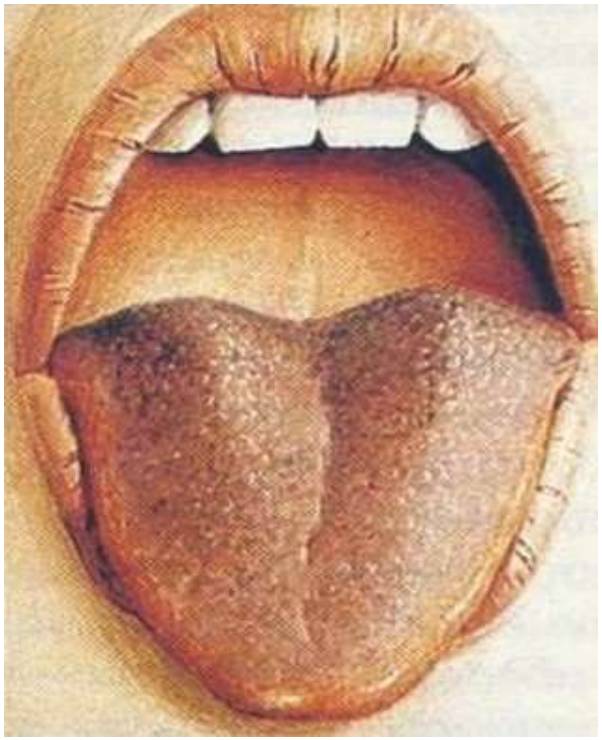

40°
19°
38°
37°
1:400
ИНТОКСИКАЦИЯ, ГОЛОВ
ВЗДУТИЕ ЖИВОТА
РАЗЖИЖЕННЫЙ СТУЛ



Treatment

Etiotrop:

- **Ciprofloxacin 500 mg x 2 times/day 7 days -** in uncomplicated forms, 10-14 days - in complicated forms and 28 days - for chronic carrier
- **Ceftriaxone 60-70 mg/kg/day (not to exceed 4g/day), 7 days**, In severe forms - 10-14 days

Pathogenetic:

- Corticosteroid therapy in severe forms (myocarditis, meningoencephalitis)
- Transfusions - in bleeding complications
- Surgical treatment - in intestinal perforations

Positive diagnosis of food -borne infections (FBI)

- ***Clinical criteria:***

- Acute onset (1-2 hours or a few days)
- Liquid stools, watery, bulky, without pathologic elements, without fever (secretory mechanism)
- Stool with blood, fever - in FBI with mixed mechanism

- ***Epidemiologic criteria:***

- Consumption of foods at increased risk of contamination
- Group sickness of several people eating the same food

- ***Laboratory criteria:***

- ***Non-specific:***
- CBC - leukocytosis with neutrophilia
- Increased hematocrit
- Metabolic acidosis
- Elevated urea and creatinine (acute kidney failure)

- ***The coprocytologic examination:***

- Absence of leukocytes in stool (FBI with secretory mechanism)
- Presence of leukocytes in stool (mixed mechanism)

Laboratory confirmation of FBI

- ***Bacteriological examination*** of feces - Culture identification and antibiotic susceptibility testing
- ***Serologic examination*** (AT, IHAT) - not useful in the acute stage of the disease, confirms the retrospective diagnosis.

In Typhoid Fever and generalized forms of FBI:

- ***Blood culture*** - positive from week 1 of illness
- ***Urine culture*** - positive within 2-3 weeks of illness
- ***Stool culture*** - positive after week 2 of illness
- ***Bile culture*** - positive during convalescence
- ***Bone marrow culture -*** positive in late-stage disease

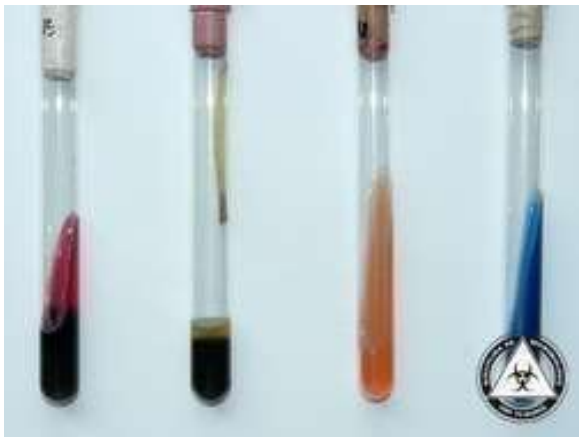


TABLE 160-4 BACTERIAL FOOD POISONING

Incubation Period, Organism	Symptoms	Common Food Sources
1–6 h		
Staphylococcus aureus	Nausea, vomiting, diarrhea	Ham, poultry, potato or egg salad, mayonnaise, cream pastries
Bacillus cereus	Nausea, vomiting, diarrhea	Fried rice
8–16 h		
Clostridium perfringens	Abdominal cramps, diarrhea (vomiting rare)	Beef, poultry, legumes, gravies
B. cereus	Abdominal cramps, diarrhea (vomiting rare)	Meats, vegetables, dried beans, cereals
>16 h		
Vibrio cholerae	Watery diarrhea	Shellfish, water
Enterotoxigenic *Escherichia coli*	Watery diarrhea	Salads, cheese, meats, water
Enterohemorrhagic *E. coli*	Bloody diarrhea	Ground beef, roast beef, salami, raw milk, raw vegetables, apple juice
Salmonella spp.	Inflammatory diarrhea	Beef, poultry, eggs, dairy products
Campylobacter jejuni	Inflammatory diarrhea	Poultry, raw milk
Shigella spp.	Dysentery	Potato or egg salad, lettuce, raw vegetables
Vibrio parahaemolyticus	Dysentery	Mollusks, crustaceans