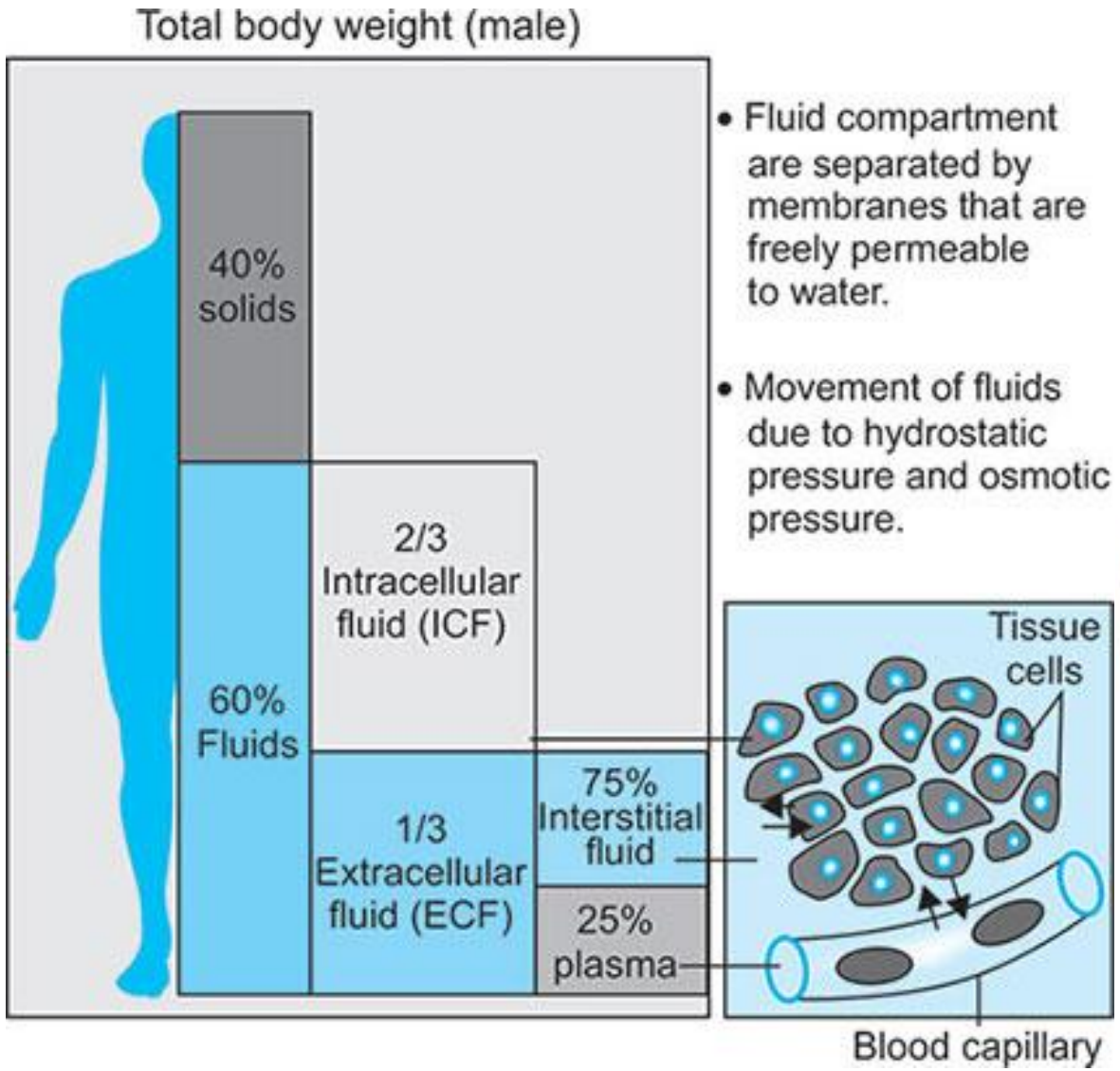
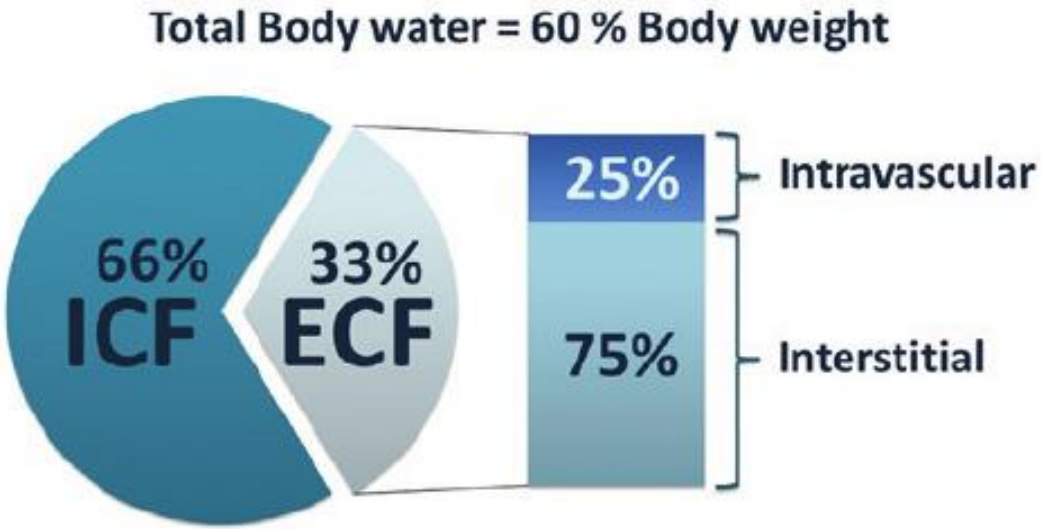
Fluid therapy

In Gastroenteritis & Food Poisoning

Dr.M.Jafarzadeh Infectious disease specialist



- Fluid compartment are separated by membranes that are freely permeable to water.
- Movement of fluids due to hydrostatic pressure and osmotic pressure.



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Adults need 2-3 liters of water daily

Water Loss

- Breathing : 600 ml
- Feces : 200 ml
- Sweating : 400ml
- Urine : 1000-2000 ml

In Gastroenteritis & Food Poisoning

Water Loss : Diarrhea & Vomitig

1. Viral Gastroenteritis
2. Bacterial Gastroenteritis
3. Parasitic Gastroenteritis

- Diarrhea
- Nausea & Vomiting
- Abdominal cramps
- Fever & chills

Fluid Therapy

1 Viral Gastroenteritis

- Group A Rotavirus
- Norovirus
- Sapovirus
- Astrovirus
- Adenovirus

2 Bacterial Gastroenteritis

- Campylobacter jejuni
- Vibrio cholerae
- Enterotoxigenic E.coli
- Salmonella spp
- Shigella
- Y.enterocolitica

3 Parasitic Gastroenteritis

- Giardia lamblia
- Cryptosporidium spp
- Entamoeba histolytica
- Strogyloides stercoralis

TABLE 203-2 Characteristics of Gastroenteritis Caused by Viral and Bacterial Agents

FEATURE	VIRAL GASTROENTERITIS	BACTERIAL GASTROENTERITIS
Setting	Incidence similar in developing and developed countries	More common in settings with poor hygiene and sanitation
Infectious dose	Low (10–100 viral particles) for most agents	High (>10^5 bacteria) for *Escherichia coli, Salmonella, Vibrio;* medium (10^2–10^5 bacteria) for *Campylobacter jejuni;* low (10–100 bacteria) for *Shigella*
Seasonality	In temperate climates, winter seasonality for most agents; year-round occurrence in tropical areas	More common in summer or rainy months, particularly in developing countries with a high disease burden
Incubation period	1–3 days for most agents; can be shorter for norovirus	1–7 days for common agents (e.g., *Campylobacter, E. coli, Shigella, Salmonella);* a few hours for bacteria producing preformed toxins (e.g., *Staphylococcus aureus, Bacillus cereus*)
Reservoir	Primarily humans	Depending on bacterial species, human (e.g., *Shigella, Salmonella*), animal (e.g., *Campylobacter, Salmonella, E. coli*), and water (e.g., *Vibrio*) reservoirs exist
Fever	Common with rotavirus and norovirus; uncommon with other agents	Common with agents causing inflammatory diarrhea (e.g., *Salmonella, Shigella*)
Vomiting	Prominent and can be the only presenting feature, especially in children	Common with bacteria producing preformed toxins; less prominent in diarrhea due to other agents
Diarrhea	Common; non-bloody in almost all cases	Prominent and occasionally bloody with agents causing inflammatory diarrhea
Duration	1–3 days for norovirus and sapovirus; 2–8 days for other viruses	1–2 days for bacteria producing preformed toxins; 2–8 days for most other bacteria
Diagnosis	This is often a diagnosis of exclusion in clinical practice. Commercial enzyme immunoassays are available for detection of rotavirus and adenovirus, but identification of other agents is limited to research and public health laboratories.	Fecal examination for leukocytes and blood is helpful in differential diagnosis. Culture of stool specimens, sometimes on special media, can identify several pathogens. Molecular techniques are useful epidemiologic tools but are not routinely used in most laboratories.
Treatment	Supportive therapy to maintain adequate hydration and nutrition should be given. Antibiotics and antimotility agents are contraindicated.	Supportive hydration therapy is adequate for most patients. Antibiotics are recommended for patients with dysentery caused by *Shigella* or diarrhea caused by *Vibrio cholerae* and for some patients with *Clostridium difficile* colitis.

Diarrheal disease

mortality has decreased substantially in the past three decades.

Nevertheless, acute diarrheal disease is still a leading cause of illness globally and is associated with an estimated 1.7 million deaths per year.

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** .

Gastrointestinal Pathogens Causing Acute Diarrhea

MECHANISM	LOCATION	ILLNESS	STOOL FINDINGS	EXAMPLES OF PATHOGENS INVOLVED
Noninflammatory (enterotoxin)	Proximal small bowel	Watery diarrhea	No fecal leukocytes; mild or no increase in fecal lactoferrin	*Vibrio cholerae*, enterotoxigenic *Escherichia coli* (LT and/or ST), enteroaggregative *E. coli*, *Clostridium perfringens*, *Bacillus cereus*, *Staphylococcus aureus*, *Aeromonas hydrophila*, *Plesiomonas shigelloides*, rotavirus, norovirus, enteric adenoviruses, *Giardia lamblia*, *Cryptosporidium* spp., *Cyclospora* spp., microsporidia
Inflammatory (invasion or cytotoxin)	Colon or distal small bowel	Dysentery or inflammatory diarrhea	Fecal polymorphonuclear leukocytes; substantial increase in fecal lactoferrin	*Shigella* spp., *Salmonella* spp., *Campylobacter jejuni*, enterohemorrhagic *E. coli*, enteroinvasive *E. coli*, *Yersinia enterocolitica*, *Listeria monocytogenes*, *Vibrio parahaemolyticus*, *Clostridium difficile*, *A. hydrophila*, *P. shigelloides*, *Entamoeba histolytica*, *Klebsiella oxytoca*
Penetrating	Distal small bowel	Enteric fever	Fecal mononuclear leukocytes	*Salmonella* Typhi, *Y. enterocolitica*

Abbreviations: LT, heat-labile enterotoxin; ST, heat-stable enterotoxin.

PATHOGENIC MECHANISMS

- Enteric pathogens have developed a variety of tactics to overcome host defenses.
- Understanding the virulence factors employed by these organisms is important in the diagnosis and treatment of clinical disease .

PATHOGENIC MECHANISMS

INOCULUM SIZE

as few as 10–100 bacteria or cysts

Shigella, Enterohemorrhagic E.coli, Giardia lamblia, Entamoeba

$10^5 - 10^8$ Vibrio cholerae

PATHOGENIC MECHANISMS

Many organisms must adhere to the gastrointestinal mucosa as an initial step in the pathogenic process; thus, organisms that can compete with the normal bowel flora and colonize the mucosa have an important advantage in causing disease.

ADHERENCE

The production of one or more exotoxins is important in the pathogenesis of numerous enteric organisms.

TOXIN PRODUCTION

Dysentery may result not only from the production of cytotoxins but also from bacterial invasion and destruction of intestinal mucosal cells.

INVASION

HOST DEFENSES

- Given the enormous number of microorganisms ingested with every meal, the normal host must combat a constant influx of potential enteric pathogens.
- Studies of infections in patients with alterations in defense mechanisms have led to a greater understanding of the variety of ways in which the normal host can protect itself against disease.

HOST DEFENSES

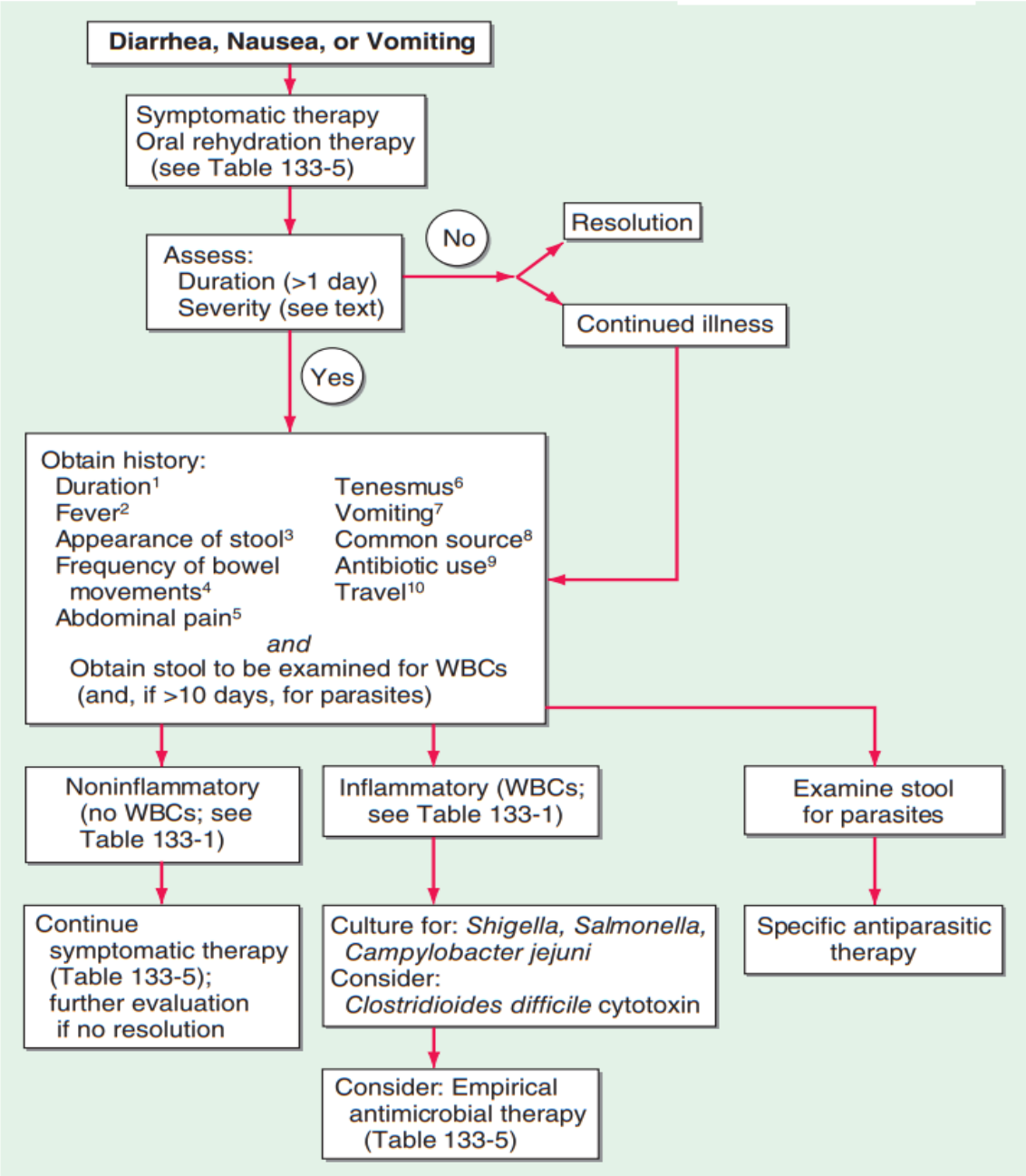
Preventing colonization by potential enteric pathogens.	INTESTINAL MICROBIOTA
The acidic pH of the stomach is an important barrier to enteric pathogens	GASTRIC ACID
Clearance of bacteria from the proximal small intestine.	INTESTINAL MOTILITY
glycoproteins and a range of antimicrobial molecules and secreted immunoglobulins directed against specific microbial antigens.	INTESTINAL MUCIN
Both cellular immune responses and Humoral immunity	IMMUNITY

GENETIC DETERMINANTS

Host genetic variation influences susceptibility to diarrheal diseases. People with blood group O show increased susceptibility to disease due to V. cholerae, Shigella, E. coli O157, and norovirus. Polymorphisms in genes encoding inflammatory mediators have been associated with the outcome of infection with enteroaggregative E. coli, enterotoxin-producing E. coli, Salmonella, C. difficile, and V. cholerae.

APPROACH TO THE PATIENT

- HISTORY
- PHYSICAL EXAMINATION
- DIAGNOSTIC APPROACH
- POSTDIARRHEA COMPLICATIONS



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Traveler’s Diarrhea

Of the several million people who travel from temperate industrialized countries to tropical regions of Asia, Africa, and Central and South America each year, 20–50% experience a sudden onset of abdominal cramps, anorexia, and watery diarrhea; thus, traveler’s diarrhea is the most common travel-related infectious illness .

LOCATION

AGE

HOST IMMUNE STATUS

BACTERIAL FOOD POISONING

If the history and the stool examination indicate a noninflammatory etiology of diarrhea and there is evidence of a common-source outbreak, questions concerning the ingestion of specific foods and the time of onset of diarrhea after a meal can provide clues to the bacterial cause of the illness.

TABLE 133-4 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

LABORATORY EVALUATION

- Many cases of noninflammatory diarrhea are self-limited or can be treated empirically, and in these instances, the clinician may not need to determine a specific etiology.
- Potentially pathogenic E. coli cannot be distinguished from normal fecal flora by routine culture, and tests to detect enterotoxins are not available in most clinical laboratories.

TREATMENT
Infectious Diarrhea or Bacterial Food Poisoning

- In many cases, a specific diagnosis is not necessary or not available to guide treatment.
- The clinician can proceed with the information obtained from the history, stool examination, and evaluation of dehydration severity .

Treatment of Traveler's Diarrhea on the Basis of Clinical Features

CLINICAL SYNDROME	SUGGESTED THERAPY
Watery diarrhea (no blood in stool, no fever), 1 or 2 unformed stools per day without distressing enteric symptoms	Oral fluids (oral rehydration solution, Pedialyte, Lytren, or flavored mineral water) and saltine crackers
Watery diarrhea (no blood in stool, no fever), 1 or 2 unformed stools per day with distressing enteric symptoms	Bismuth subsalicylate (for adults): 30 mL or 2 tablets (262 mg/tablet) every 30 min for 8 doses; or loperamide[b]: 4 mg initially followed by 2 mg after passage of each unformed stool, not to exceed 8 tablets (16 mg) per day (prescription dose) or 4 caplets (8 mg) per day (over-the-counter dose); drugs can be taken for 2 days. Antibacterial drug[c] can be considered in selected circumstances.
Dysentery (passage of bloody stools) or fever (>37.8°C)	Antibacterial drug[c]
Vomiting, minimal diarrhea	Bismuth subsalicylate (for adults; see dose above)
Diarrhea in infants (<2 years old)	Fluids and electrolytes (oral rehydration solution, Pedialyte, Lytren); continue feeding, especially with breast milk; seek medical attention for moderate dehydration, fever lasting >24 h, bloody stools, or diarrhea lasting more than several days

PROPHYLAXIS

- Improvements in hygiene to limit fecal–oral spread of enteric pathogens will be necessary if the prevalence of diarrheal diseases is to be significantly reduced in developing countries.
- Travelers can reduce their risk of diarrhea by eating only hot, freshly cooked food; by avoiding raw vegetables, salads, and unpeeled fruit; and by drinking only boiled or treated water and avoiding ice.

Vibrio Cholerae

- Cholera is an acute diarrheal disease that can, in a matter of hours, result in profound, rapidly progressive dehydration and death.
- prompt aggressive fluid repletion and supportive care can obviate the high mortality that is historically associated with cholera.
- It is possible that >2–3 million cases of cholera occur yearly (of which only ~200,000 are reported to the WHO) and that these cases result in >50,000–100,000 deaths annually (of which < 2000 are reported to the WHO).

TABLE 163-1 Assessing the Degree of Dehydration in Patients with Cholera

DEGREE OF DEHYDRATION	CLINICAL FINDINGS
None or mild, but diarrhea	Thirst in some cases; <5% loss of total body weight
Moderate	Thirst, postural hypotension, weakness, tachycardia, decreased skin turgor, dry mouth/tongue, no tears; 5–10% loss of total body weight
Severe	Unconsciousness, lethargy, or “floppiness”; weak or absent pulse; inability to drink; sunken eyes (and, in infants, sunken fontanelles); >10% loss of total body weight

TABLE 163-2 Treatment of Cholera, Based on Degree of Dehydration[a]	
DEGREE OF DEHYDRATION, PATIENT'S AGE (WEIGHT)	**TREATMENT[b]**
None or Mild, but Diarrhea[c]	
<2 years	1/4–1/2 cup (50–100 mL) of ORS, to a maximum of 0.5 L/d
2–9 years	1/2–1 cup (100–200 mL) of ORS, to a maximum of 1 L/d
≥10 years	As much ORS as desired, to a maximum of 2 L/d
Moderate[c,d]	
<4 months (<5 kg)	200–400 mL of ORS
4–11 months (5–<8 kg)	400–600 mL of ORS
12–23 months (8–<11 kg)	600–800 mL of ORS
2–4 years (11–<16 kg)	800–1200 mL of ORS
5–14 years (16–<30 kg)	1200–2200 mL of ORS
≥15 years (≥30 kg)	2200–4000 mL of ORS
Severe[c]	
All ages and weights	Undertake IV fluid replacement with Ringer's lactate (or, if not available, normal saline). Give 100 mL/kg in the first 3-h period (or the first 6-h period for children <12 months old); start rapidly, then slow down. Give a total of 200 mL/kg in the first 24 h. Continue until the patient is awake, can ingest ORS, and no longer has a weak pulse.

Clostridioides Difficile

- DEFINITION Clostridioides difficile infection (CDI) is a unique colonic disease that is acquired most commonly in association with antimicrobial use and the consequent disruption of the normal colonic microbiota.
- The most commonly diagnosed diarrheal illness acquired in the hospital, CDI results from the ingestion of spores of C. difficile that vegetate, multiply, and secrete toxins, causing diarrhea and, in the most severe cases, pseudomembranous colitis (PMC) .

ETIOLOGY AND EPIDEMIOLOGY

- C. difficile is an obligately anaerobic, gram-positive, spore-forming bacillus whose spores are found widely in nature, particularly in the environment of hospitals and chronic-care facilities.
- CDI occurs frequently in hospitals and nursing homes (or shortly after discharge from these facilities) where the level of antimicrobial use is high and the environment is contaminated by C. difficile spores.

PATHOLOGY AND PATHOGENESIS

Spores of toxigenic C. difficile are ingested, survive gastric acidity, germinate in the small bowel, and colonize the lower intestinal tract, where they elaborate two large toxins: toxin A (an enterotoxin) and toxin B (a cytotoxin).

GLOBAL CONSIDERATIONS

- Rates and severity of CDI in the United States, Canada, and Europe increased markedly in the early 2000s.
- Rates in U.S. hospitals tripled between 2000 and 2005.
- Hospitals in Montreal, Quebec, reported rates in 2005 that were four times higher than the 1997 baseline, with directly attributable mortality of 6.9% (increased from 1.5%).

CLINICAL MANIFESTATIONS

- Diarrhea is the most common manifestation caused by C. difficile.
- Stools are almost never grossly bloody and range from soft and unformed to watery or mucoid in consistency, with a characteristic odor.
- Clinical and laboratory findings include fever in 28% of cases, abdominal pain in 22%, and leukocytosis in 50%.
- When adynamic ileus (which is seen on x-ray in ~20% of cases) results in cessation of stool passage, the diagnosis of CDI is frequently overlooked.

DIAGNOSIS

- The diagnosis of CDI is based on a combination of clinical criteria: (1) diarrhea (≥3 unformed stools per 24 h for ≥2 days) with no other recognized cause plus (2) detection of toxin A or B in the stool, detection of toxin-producing C. difficile in the stool by nucleic acid amplification testing (NAAT; e.g., polymerase chain reaction [PCR]) or by culture, or visualization of pseudomembranes in the colon.
- Most laboratory tests for toxins, including enzyme immunoassays (EIAs), lack sensitivity.
- However, testing of multiple additional stool specimens is not recommended.

Relative Sensitivity and Specificity of Diagnostic Tests for Clostridioides difficile Infection (CDI)

TYPE OF TEST	RELATIVE SENSITIVITY[a]	RELATIVE SPECIFICITY[a]	COMMENT
Stool culture for *C. difficile*	++++	+++	Most sensitive test; specificity of ++++ if the *C. difficile* isolate tests positive for toxin; turnaround time too slow for practical use
Cell culture cytotoxin test on stool	+++	++++	With clinical data, is diagnostic of CDI; highly specific but not as sensitive as stool culture; slow turnaround time
Enzyme immunoassay for toxins A and B in stool	++ to +++	+++	With clinical data, is diagnostic of CDI; rapid results, but not as sensitive as stool culture or cell culture cytotoxin test
Enzyme immunoassay for *C. difficile* common antigen in stool	+++ to ++++	+++	Detects glutamate dehydrogenase found in toxigenic and nontoxigenic strains of *C. difficile* and other stool organisms; more sensitive and less specific than enzyme immunoassay for toxins; requires confirmation with a toxin test; rapid results
Nucleic acid amplification tests for *C. difficile* toxin A or B gene in stool	++++	+++	Detects toxigenic *C. difficile* in stool; widely used in United States for clinical testing; more sensitive than enzyme immunoassay toxin testing; marked increase in CDI diagnoses when implemented
Colonoscopy or sigmoidoscopy	+	++++	Highly specific if pseudomembranes are seen; insensitive compared with other tests

TREATMENT

PRIMARY CDI

- When possible, discontinuation of any ongoing antimicrobial administration is recommended as the first step in treatment of CDI.
- Earlier studies indicated that 15–23% of patients respond to this simple measure.

TREATMENT

RECURRENT CDI

- Overall, ~15–30% of successfully treated patients experience recurrences of CDI following treatment.
- CDI recurrence is significantly lower in patients treated with fidaxomicin than in those treated with vancomycin.
- Vancomycin and metronidazole have comparable recurrence rates, and metronidazole is not recommended for treatment of recurrent CDI.

TREATMENT

SEVERE COMPLICATED OR FULMINANT CDI

- Fulminant (rapidly progressive and severe) CDI presents the most difficult treatment challenge.
- Patients with fulminant disease often do not have diarrhea, and their illness mimics an acute surgical abdomen.
- Sepsis (hypotension, fever, tachycardia, leukocytosis) may result from fulminant CDI.

PROGNOSIS

- The mortality rate attributed to CDI, previously found to be 0.6–3.5%, has reached 6.9% in recent outbreaks and is progressively higher with increasing age.
- Most patients recover, but recurrences are common.

PREVENTION AND CONTROL

Strategies for the prevention of CDI are of two types :

1.those aimed at preventing transmission of the organism to the patient

2.those aimed at reducing the risk of CDI if the organism is transmitted.


Thank
you!