### APPROACH TO DIARRHOEA

# ACCOUNTS FOR UP TO 5% PRESENTATIONS, BUT MOST ARE SELF-LIMITED

#### **INCIDENCE:**

- Worldwide, remains a major problem → 4% deaths, mostly kids in developing countries
- Rotavirus accounts for 25-65% childhood cases, norovirus ~90% adult cases
  - At risk groups:
    - Extremes of age
    - $\circ$  Immunocompromised
    - Significant comorbidity

#### **DEFINITION AND CATEGORISATION:**

- TWO MAIN GROUPS → INFECTIOUS AND NON-INFECTIOUS
  - INFECTIOUS  $\rightarrow$  ~85% CASES
    - Viral ~70%
    - Bacterial 24%
      - Invasive → campylobacter, C-difficile, enteroinvasive E coli, salmonella, shigella, Yersinia enterocolitica
      - Toxigenic → pre-formed (staph aureus, Bacillus cereus, clostidium botulinum. Toxin formed after colonization → clostridium perfringens, aeromonas, enterohaemorrhagic E coli (0157:H7), shigella, vibrio cholerae
    - Parasitic ~6%
      - Protozoa → entamoeba histolytica, giardia, cryptosporidium
      - Helminthes  $\rightarrow$  hookworms, schistosoma
- ACUTE VS CHRONIC:
  - Acute  $\rightarrow \leq 14$  days
    - Usually self-limited and infectious in origin
  - Persistent  $\rightarrow$  lasts longer than 14 days:
    - Enteric pathogen other than viral
  - Chronic ≥30 days
    - Usually non-infectious cause
- PATHOPHYSIOLOGAL CLASSIFICATION:
  - $\circ$  Any state that reduces water absorption by 1% can cause diarrhoea
  - SECRETORY:
    - Oversecretion of water and electrolytes as a result of increased cellular permeability
    - Account for most ED cases
  - INFLAMMATORY:
    - Aka invasive → results from cellular DAMAGE to intestinal mucosa → hypersecretion of water electrolytes, blood, mucous and plasma proteins
    - Most commonly caused by invasive bacterial and parasitic pathogens that produce dysenteric illnesses

- Non-infectious causes → radiation therapy, hypersensitivity reactions, ischaemic colitis, IBD
- OSMOTIC:
  - Ingestion or malabsorption of osmotically active solutes
  - Cause osmotic movement of water into intestinal lumen
- ABNORMAL MOTILITY:
  - Generally seen in patients with chronic diarrhoea, but is always a component of acute diarrhoea
  - Hypermotility decreases contact time between solute and mucosa

## **CLINICAL APPROACH:**

- Initial focus is on assessment of volume status and fluid resuscitation
- Associated septic shock may contribute to hypotension and hypoperfusion
- Beware non-anion gap acidosis from massive bicarbonate loss
- SECONDARY SURVEY:
  - Physical exam should assess the patient's overall health, toxicity, fever, volume state and signs of a surgical abdomen
  - In kids  $\rightarrow$  look for sunken eyes/fontanelle,  $\downarrow$  urine output, lethargy
- CHARACTERISATION OF THE DIARRHOEAL SYNDROME:
  - ACUTE INFECTIOUS:
    - Most viral and many bacterial agents cause a self-limited secretory diarrhoea that lasts less than 14 days → 90% caused by norovirus → symptomatic treatment
    - Evaluate for factors that may change the probability of it being 'NOT NOROVIRUS' → with one or more of these findings → empirical antibiotics to be considered

Table 23-1 Factors Increasing Probability of Nonbenign Diarrhea

FACTOR	SPECIFIC PATHOGEN(S)/OTHER CONSIDERATIONS
Presentation to a health care facility	Degree of illness overall greater in patients presenting for evaluation; increased probability of "not norovirus" etiology to 50%
Travel history	Especially foreign travel and to endemic areas of dysenteric disease
Recent hospitalization	C. difficile from antibiotic exposure
Day care attendance	Rotavirus, Shigella, Giardia
Nursing home residence	C. <i>difficile</i> , medication side effects, tube feedings, ischemic colitis, fecal impaction, and overflow diarrhea
Wilderness exposure	Giardia or Cryptosporidium
Antibiotic therapy	C. difficile, antibiotic side effects
Raw shellfish, farm animals and fair livestock, pet reptiles or amphibians, petting zoos	Salmonella spp., E. coli O157:H7 and non-O157 Shiga toxin-producing E. coli, Vibrio spp.
Epidemic of multiple patients with a short time of onset	Norovirus; less commonly, Campylobacter jejuni, Salmonella spp., Cryptosporidium
Acute vomiting and diarrhea after suspected contaminated food	Bacillus cereus, Clostridium botulinum, Staphylococcus aureus
Epidemic of severe gastroenteritis traced to eggs, poultry, meat, or dairy products	Campylobacter jejuni, Salmonella spp.
Homosexual lifestyle (males)	Giardia lamblia, Entamoeba hystolytica
Abdominal pain Nausea, vomiting	Severe bacterial infections: Salmonella, Campylobacter, Shigella, EPEC, Yersinia or Vibrio spp.
Bloody stool	Also consider surgical abdomen, GI bleeding
Pever Destal agin	Inflammatory bowel disease
Tenesmus	
Diarrhea >7–14 days' duration	Protozoa and microsporidia, Clostridium difficile, Campylobacter, Shiga toxin- producine E. coli
Hemolytic uremic syndrome	E. coli O157:H7 or other species
Stool WBC count	Not reliable for diagnosis of bacterial etiology
Colonic ulcerations	Inflammatory bowel disease
Proctitis	Bacterial etiology highly probable
Pseudomembranes	Toxic megacolon, Clostridium difficile
Chronic disease (e.g., cirrhosis, DM)	Complicated course expected with any form of diarrheal illness
Organ transplantation	Abnormally severe illness from rotavirus and adenovirus
	Increased frequency of cytomegalovirus
	Severe illness from dysenteric diarrhea
	Spore-forming protozoa and microsporidia
HIV intection, other immunodeficiency disorders	bevere liness from common bacteria/spore-forming protozoa and
	Increased frequency of outomegalouirus and Muchagterium guine complex

#### • CHRONIC INFECTIOUS:

- Consider bacterial, parasitic causes and treat accordingly
- NON-INFECTIOUS:
  - TOXINS:
    - Multitude of drugs
    - Dietetic foods  $\rightarrow$  sorbitol, mannitol
    - Fish/plant associated toxins
  - OPIATE WITHDRAWAL
  - GI PATHOLOGY → obstruction, cirrhosis, melaena, appendicitis, ischaemic gut, IBD, malabsorption
  - ENDOCRINE-RELATED → carcinoid, hyperthyroidism, the weird and the wonderful (e.g. Zollinger-Ellison)
  - SYSTEMIC
- The distinction between infectious and non-infectious may not always be apparent
  - Always consider surgical pathology

#### **EMPIRICAL MANAGEMENT:**

- Oral rehydration is the treatement of choice for mild to moderate fluid losses
- The concept of BOWEL REST HAS BEEN ABANDONED because it may worsen diarrhoea
  - BRAT diet (bananas, rice, apples and toast)
  - o Avoid caffeine

- Avoid lactose as many pathogens cause a transient lactase deficiency
- High fat may delay gastric emptying and should be avoided
- Because the specific pathogen is rarely identified in the ED, any antimicrobial treatment must be empirical and guided by knowledge of the common cases of infectious diarrhoea
  - Directed against invasive bacterial and parasitic organisms that cause the greatest harm
  - Initiate antibiotics if:
    - Suspected invasive process/severe diarrhoea
    - Systemic symptoms
    - Fever/toxic appearance/abdominal pain
  - CIPROFLOXACIN currently advocated  $\rightarrow$  not to pregnant women
  - If possible, treatment for paediatric patients should be on the basis of culture results as antibiotic treatment of severe gastroenteritis in kids has been associated with development of HUS/TTP if E coli 0157:H7 the cause.
  - If amoebic dysentery is a concern  $\rightarrow$  metronidazole
- Use of antimotility agents is controversial:
  - $\circ$  Patients with simple, acute viral gastro seem to benefit from antimotility agents  $\rightarrow$  LOPERAMIDE
  - In kids, use of these agents has rarely been associated with HUS and toxic megacolon
- Probiotics effective in restoring normal flora, especially in travelers' diarrhoea and in nonspecific diarrhoea in kids