Spore Forming Gram-Positive Bacilli

BACILLUS SPECIES

Large **aerobic**, occurring in chains (3-4 μm). Saprophytic, prevalent in soil, water, and air.

BACILLUS CEREUS

- Motile, no vaccine, high lecithenase activity, beta hemolysis, resistant to Penicillin and Cephalosporins and it is ubiquitous.
- Cause **food poisoning** (very common and self-limiting), endocarditis, eye infections, meningitis osteomyelitis, and pneumonia mainly in immunocomised patients.
- Pathogenesis→ secreted toxins: hemolysins, distinct phospholipases, an emesis inducing toxin, and three pore-forming enterotoxins: hemolysin BL(HBL), non-hemolytic enterotoxin (NHE), and cytotoxin K.
- Two clinical syndromes produced by the toxins:
  1 vomiting type [cerulide] → heat stable toxins [they survive flash frying], incubation period ½ - 6 hours, nausea, vomiting, abdominal cramps, sometimes diarrhea, connected with chines food especially rice and cereals, **it's formed outside the body**. The illness usually self-limiting.
  2 The Diarrheal type → heat labile, the incubation period 6–15 hours, diarrhea, abdominal cramps, sometimes nausea and vomiting, connected with contaminated meat, vegetables and sauces, **it's formed inside the colon** after ingesting bacillus spores.
- Diagnosis: clinical grounds, specimen from the suspect food and do culture + gram stain → Large feathery White Colonies, typical for Bacillus Cereus (using sheep blood agar plate).
- Treatment: antimicrobial therapy is NOT normally required.
  -Diarrheal type → vital signs +fluid & electrolyte replacement.
  -Food poisoning → self-limiting.

BACILLUS ANTHRACIS

- Non motile, low or no lecithenase activity, no beta hemolysis, sensitive to Penicillin and Cephalosporins
- Causes **anthrax**:
  1)cutaneous anthrax → start as malignant pustule over the skin.
  2)inhalational anthrax → "woolstorter's disease"
  3)GI anthrax (rare)→ ingestion of spores
- Culture: Medusa-head" colonies (typical for b. anthracis)
- Has a capsule consisting mainly of D-Glutamic acid → has a vaccine

CLOSTRIDIUM SPECIES

Motile (**except C. perfringens**) and possess peritrichous flagella, **anaerobes** (a few species are aerotolerant), grow well in the blood-enriched media or media used to grow anaerobes, prevalent in the environment and normal inhabitants of the human intestinal tract.
Spores of clostridia are usually wider than the diameter of the rods

CLOSTRIDIUM BOTULINUM

- Habitat: in the soil, it colonizes the GIT of fish, birds and mammals
- MOA: The most common offenders are spiced, smoked, vacuum packed, or canned alkaline foods that are eaten without cooking. In such foods, spores of C. botulinum germinate under anaerobic conditions, vegetative forms grow and produce Botulinum toxin.
- Botulinum toxin: highly toxic neurotoxin/coded for by a prophage/has 7serotypes (A-G). A&B&E are associated with human infections. Thus, we give patients Botulism Immune Globulin Intravenous (Human) (BIG-IV), that contains the three common serotypes (trivalent).
- The toxin then absorbed by the gut and carried by blood to peripheral nerve synapses and blocks the release of Ach at the myo-neuronal junction resulting in **reversible** flaccid paralysis (Botulism)
- Botulism: symmetrical, descending, flaccid paralysis of motor and autonomic nerves usually beginning with cranial nerves.
- There are five clinical categories of botulism:
  1. Foodborne botulism (ADULT BOTULISM): The toxin is already preformed outside of the body.
  2. Infant botulism (the most common form): The baby eats **honey** that contains spores → germinate later in the guts producing toxins (one of the causes of sudden infant death syndrome)
  3. wound botulism: contaminated wounds
  4. Inadvertent: following botulinum IM toxic injection.
- Clinical finding: 1. Initial symptoms can include nausea, vomiting, abdominal cramps or diarrhea. 2. Dry mouth, blurred vision, and diplopia are usually

CLOSTRIDIUM PERFRINGENS

- The most common in invasive disease including myonecrosis and gas gangrene.
- An enterotoxin of C. perfringens is a common cause of food poisoning (self-improvement)
- C. Perfringens is the least spore former among other spore forming species either in laboratory media or in tissue
- Double zone of hemolysis (alpha and beta).
- "Stormy fermentation": characteristic of C. perfringens coagulation of milk in litmus milk test in addition to gas production.
- Reservoir: soil and human's colon.
- Transmission: foodborne or traumatic implantation in the case of gas gangrene or myonecrosis or from the intestinal tract.
- Pathogenesis: Spores germinate at low oxidation reduction potential; vegetative cells multiply, ferment carbohydrates present in tissue, and **produce gas**. Toxins have lethal, necrotizing, and hemolytic properties→ αlpha (lecithinase)+ theta (necrotizing) theta (necrotizing)+ epsilon (edematous) toxins.
- Clinical findings:
  *From a contaminated wound the infection spreads in 1–3 days to produce **crepitation** in the subcutaneous tissue and muscle, **foul-smelling discharge**, rapidly progressing necrosis, fever, hemolysis, toxemia, shock, and death.

  *C perfringens food poisoning usually follows the ingestion of large numbers of clostridia that have grown in **warmed meat dishes**. The toxin forms when the organisms **sporulate in the gut**, with the

the earliest neurologic symptoms.
3. Inability to swallow, and speech difficulty.
4. In severe cases, extensive respiratory muscle paralysis leads to ventilatory failure.

*Infants in the first months of life develop poor feeding, weakness, **floppy baby**.

- Diagnosis: Clinical grounds+ Toxin may be found by ELISAs and PCR / Mouse bioassay is the **test of choice** for the confirmation of botulism
- Treatment: NO antibiotics! Supportive treatment, especially adequate mechanical ventilation+ IV trivalent (A, B, E) anti-toxin + Surgical debridement in wound botulism
- Prevention and control: Canned food must be sufficiently heated to ensure destruction of spores [boiled for more than 20 minutes].
  No honey for the first year infants.

onset of diarrhea—usually without vomiting or fever—in 7–30 hours. The illness lasts only 1–2 days; usually self-limiting

*C. perfringens causes **Endometritis**. Thus, it may cause post-partum syndrome or septic shock.

- Diagnosis:
  1. Gram stain (specimen from wounds/pus/tissue).
  2. Thioglycolate medium/ blood agar plates.
  3. Nagler test→ rests on toxin production and neutralization by specific antitoxin
- Treatment and prevention:
  1- Surgical debridement
  2- Administration of antimicrobial drugs
  3- Antitoxins (but should not be relied on)
  4- Food poisoning usually requires only symptomatic care (fluid & electrolyte replacement)

CLOSTRIDIUM DIFFICILE

- Ubiquitous in the environment and colonizes the intestine of 50% of healthy neonates and 4% of healthy adults.
- A major cause of health-associated infection; patients taking antibiotics (cephalosporins, clindamycin) are at an increased risk of developing C. difficile antibiotic associated diarrhea.
- The infection may be endogenous (this is due to suppression of the normal bowel flora and subsequent of C. difficile) or exogenous (ingestion of environmental spores).
- Produce 2 major toxins:
  Toxin A → induce cytokine production with hypersecretion of fluid.
  Toxin B → induce depolymerization of actin with loss of cytoskeleton.
- Hypervirulent, hypertoxin producing strains are now recognized (027,078)

CLOSTRIDIUM TETANI

- Cause rigid paralysis (tetanus)

- Diseases:
  1.Antibiotic associated diarrhea
  2. Pseudomembranous colitis, fulminant colitis (presence of whitish membrane covering the colon)

- Diagnosis:
  1. Diarrhea
  2. Detect the toxins by ELISA, latex agglutination, PCR or culture in selective media.
  3. Pseudo membranes in colon

- Treatment:
  1- discontinue other antibiotics therapy
  2- oral administration of vancomycin or metronidazole
  3- limited-spectrum drugs should be considered first
  4- Autoclave (temperature of 121°C & pressure) ensure the destruction of spores.
  5- fecal transplantation

DONE BY:
SHAHED ATIYAT

GOOD LUCK