

ANAEROBIC BACTERIA

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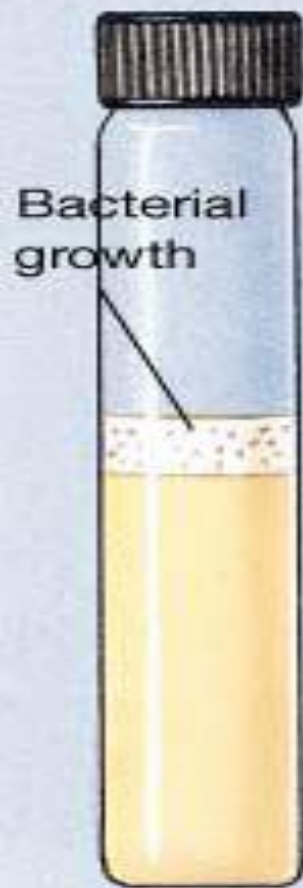
DEFENITION

■ A MICRBE THAT CAN ONLY GROW UNDER ANAROBIC CONDITION

■ SENSITIVE TO metronidazole (MTZ)

■ FAIL TO GROW IN AIR 10 % O₂





Obligate
aerobe



Obligate
anaerobe



Microaerophile



Facultative
anaerobe

Why can't anaerobic bacteria survive in oxygen?

- The presence of oxygen leads to the production in cells of the **superoxide radical** (a negatively charged O_2 molecule). Normally, the superoxide anion is lethal enough to kill almost any organism. Aerobic organisms and facultative anaerobes have the enzymes superoxide dismutase and catalase. These enzymes work together to convert superoxide to oxygen and hydrogen peroxide

CLASSIFICATION

■ A - NON SPORE FORMING

{MOR COMMN}

■ B - SPORE FORMING

A - NON SPORING

A -GRAM NEGATIVE BACILLI

- *bacteroides fragilis* (resistant to penicillin)
- *Prevotella spp*
- *Leptotricha buccalis*
- *fusobacterium spp f.nucleatum*
- *Viellonella sp.* GRAM NEGATIVE COCCI

■ B –GRAMME POSITIVE COCCI

- *Peptococci*
- *Peptostreptococci*

■ C –GRAMME POSITIVE BACILLI

- *Propionobacterium propionicum ,p.acne*
- *Bifidobacterium*
- *Euobacterium*
- LACTOBACILLUS
- *Actinomyces israelii*

■ D-MICROAEROPHILIC STREPT.

ବିଭାଗୀୟତା

- ଗ୍ରୁପ୍ = ବି. ଫ୍ରାଗ୍ମେଣ୍ଟସ୍, ବି. ୟୁଲ୍ଟ୍ରାସ୍, ବି. ଟେକ୍ନୋଲୋଜି, ବି. ୟୁଲ୍ଟ୍ରାସ୍, ବି. ଟେକ୍ନୋଲୋଜି, ବି. ୟୁଲ୍ଟ୍ରାସ୍
- ଉପାଦାନ ୧୦୦ ୧/୩ ଠାରୁ ଅଧିକ ଶ୍ରେଣୀ
- କମ୍ପାନୀ ୨୦% ବ୍ୟୟ
- କମ୍ପାନୀ ୨୦ ଉପାଦାନ ଉପାଦାନ
 - ଟେକ୍ନୋଲୋଜି, କମ୍ପାନୀ, ଉପାଦାନ, ଉପାଦାନ, ଉପାଦାନ - ଉପାଦାନ ଉପାଦାନ
- ୧୦ ପ୍ରତିଶତରୁ ଅଧିକ ଉପାଦାନ ୧୦
ଫୁଲ୍ଟାଇମ୍



ବିଭେଦନାତ୍ମକ ଉପାଦାନ ସମ୍ପ

- ବିଭେଦନାତ୍ମକ ସମ୍ପଦାତ୍ମକ ଉପାଦାନ ସମ୍ପଦା
ବି. ସମ୍ପଦାତ୍ମକ ଉପାଦାନ
- ବିଭେଦନାତ୍ମକ ଉପାଦାନ
- ବିଭେଦନାତ୍ମକ ଉପାଦାନ
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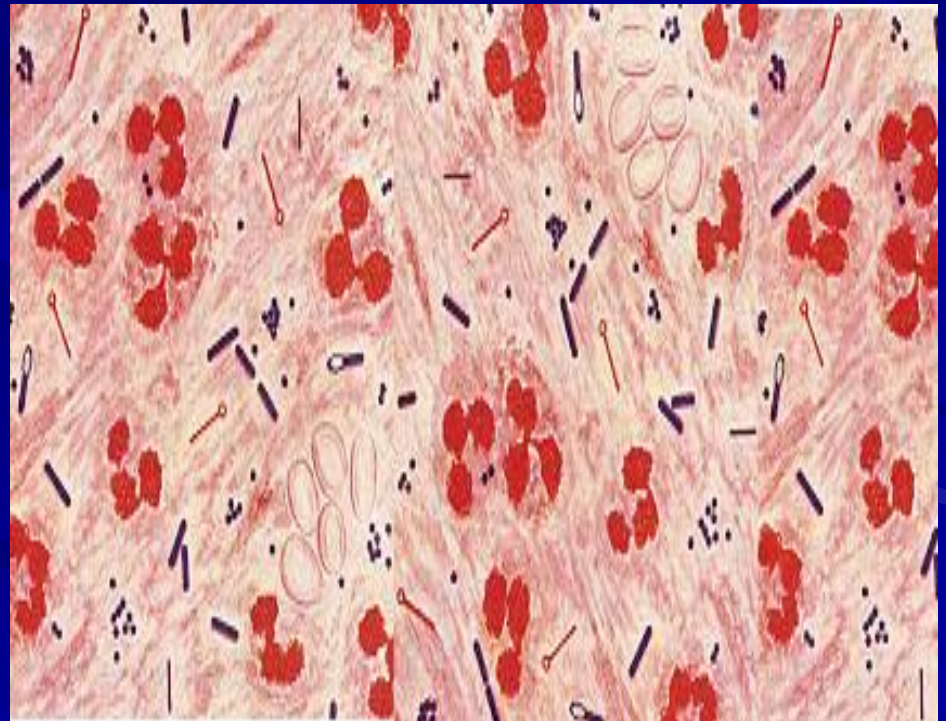
SPORE FORMING

■ GRAME POSITIVE BACILLI

- CL .perfringens*
- CL .Septicum*
- CL .novyi*
- CL .Histolyticum*
- CL .Difficile*
- CL .Tetani*
- CL .Botulinum*

IMPORTANCE

- Dominate the indigenous flora (colonization resistance)
- Commonly found in infection
- Easy to overlook
 - special precautions
 - Slow growth
 - Mixed infection
- Difficult treatment



PRESENCE AS NORMAL FLORA

- Skin
- Nose
- Mouth, throat
- Stomach
- Large intestine $>10^{11}$ / gram colonic contents
- Vagina
- Endocervix
- Urethra

MODIFIED BY

- Pathophysiologic states
- Antimicrobial agents ,H-Blockers ,antacids
- Hormonal changes
- Age

EPIDEMIOLOGY

- Almost all infections are indigenous except
 - Tetanus
 - Infant ,wound botulism
 - Gas gangrene { some cases }
 - Bites
 - C .difficile {nosocomial }

HABITAT :

These organism are normal flora in:

■ Oropharynx

Prevotella melaninogenica, Fusobacteria, Veillonella

■ Gastrointestinal tract

- Found mainly in the large colon in large numbers
- Total number of anaerobes = 10^{11}
- While all aerobes (including *E. coli*) = 10^4
- examples are (1) *Bacteroides fragilis*
- *Bifidobacterium species*

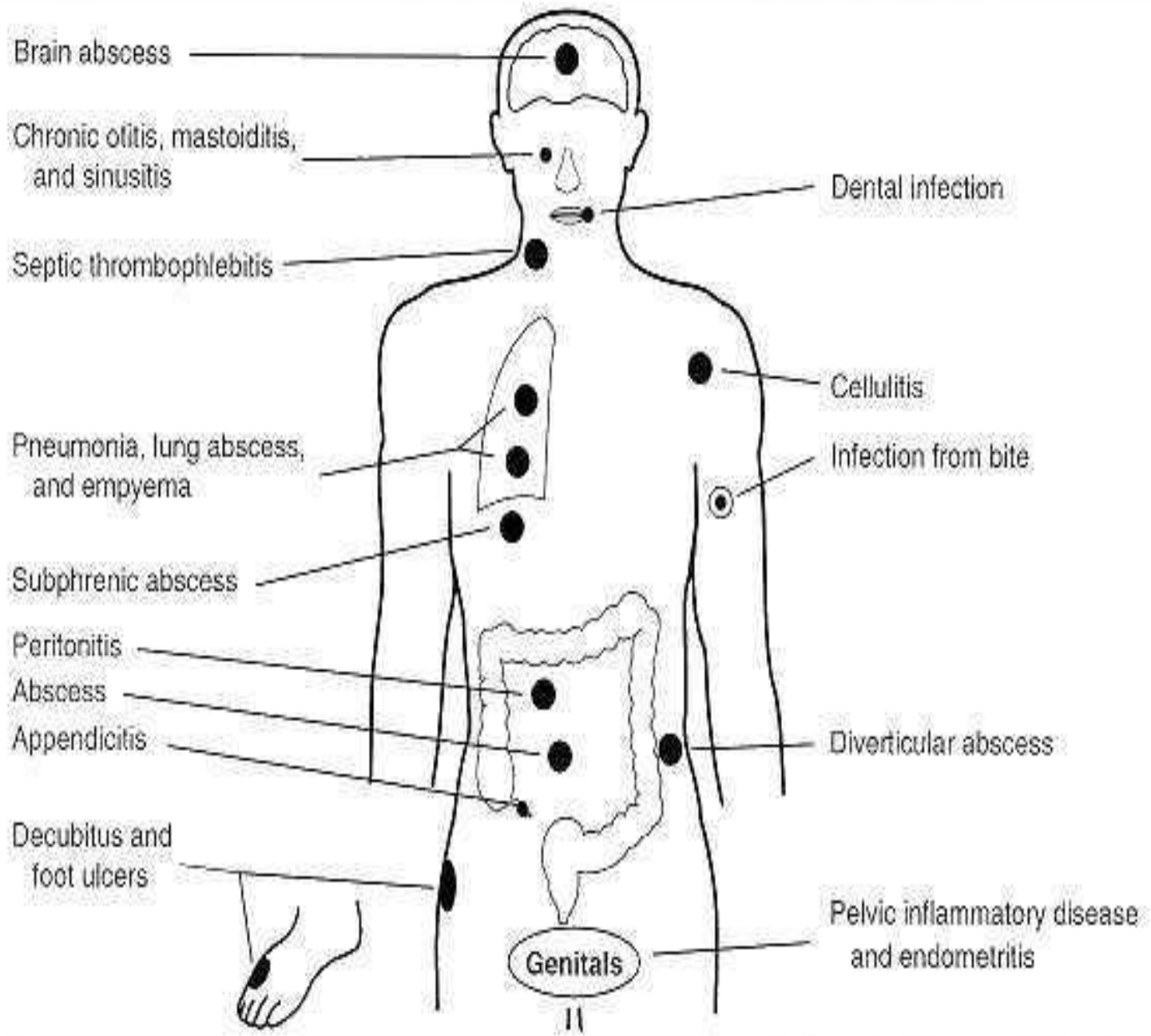
■ Female genital tract (mainly in the vagina)

CHARACTER OF ANAEROBIC INFECTION

- Suppuration
- Abscess formation
- Tissue destruction{gangrene}
- Septic thrombophlebitis
- Some have unique pathology 
 - Actinomycosis
 - Pseudomembranous colitis
 - Gas gangrene

PREDISPOSING FACTORS

- Low O tension {Eh}
- Trauma, dead tissue , deep wound
- Impaired blood supply
- Presence of other organisms
- Foreign bodies

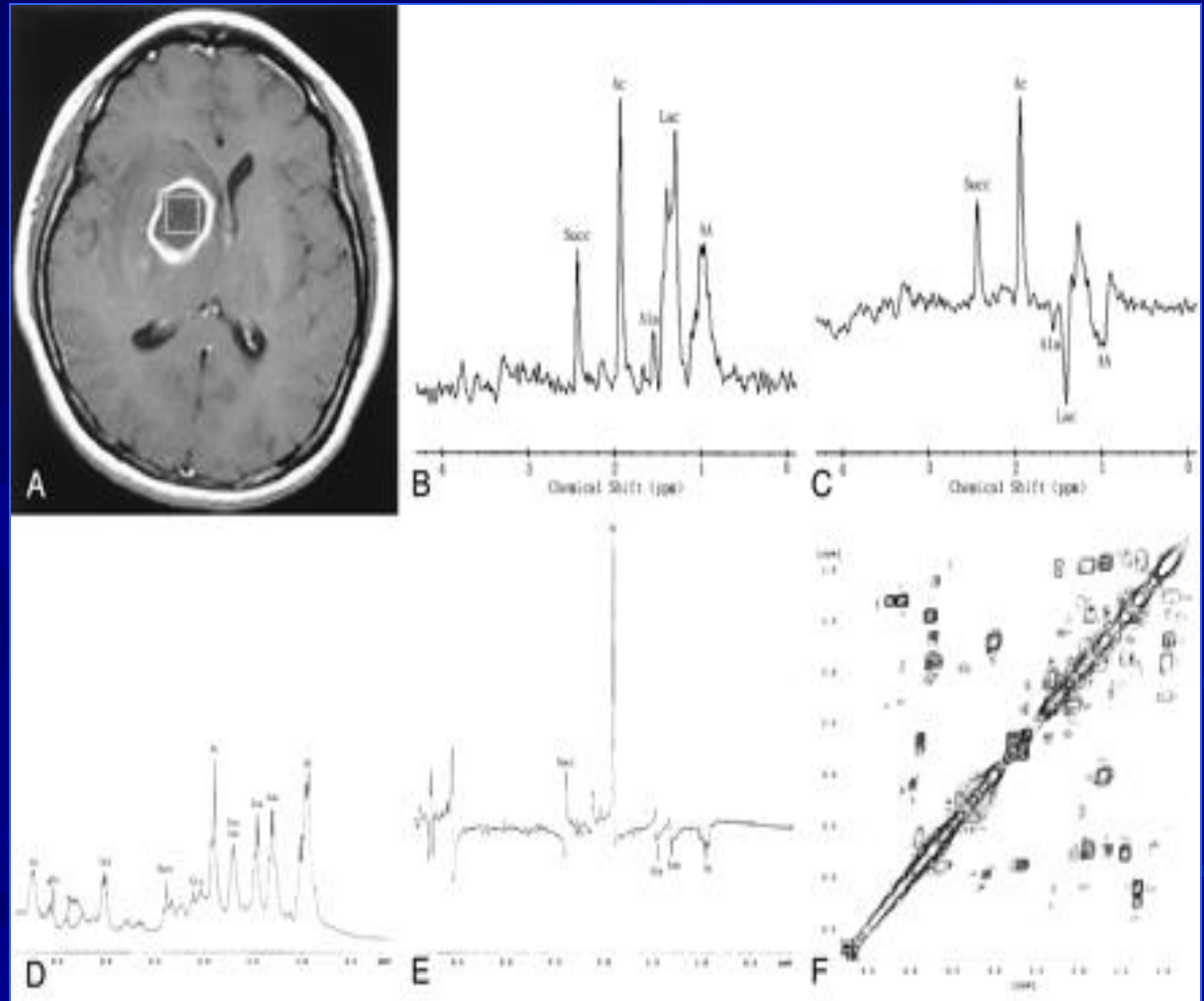


**Frequency with which Anaerobes
are Associated with Human Disease**

Infection	Anaerobes Involved (%)*
Bacteremia	10-20
Central nervous system	
Brain abscess	89
ENT-dental	52
Thoracic	
Aspiration pneumonia	93
Lung abscess	93
Empyema (nonsurgical)	76
Intraabdominal	
General infection	86
Liver abscess	50-100
Appendicitis	96
Obstetric-gynecologic	
Vulvovaginal abscess	74
Pelvic abscess	92

■ A 43-year-old man with surgically proved pyogenic brain abscess in the right basal ganglion secondary to *Eubacterium lentum* (obligate anaerobe) infection.

■ Axial contrast-enhanced T1-weighted MR image shows a ring-shaped cystic lesion and surrounding edema.



Predisposing factors

- Antibiotic therapy
- Neoplasm
- Trauma
- Cholecystitis
- Obstruction
- Ulceration
- Diabetes mellitus
- Pylephlebitis
- Diverticula formation

INFECTIONS BEGIN :

■ DISRUPTION OF BARRIERS

- TRAUMA
- OPERATIONS
- CANCEROUS INVASION OF TISSUES

■ DISRUPTION OF BLOOD SUPPLY

- DROPS OXYGEN CONTENT OF TISSUE
- DECREASE IN E_h POTENTIAL
- TISSUE NECROSIS

LABORATORY DIAGNOSIS:

- When anaerobic infection is suspected;
 - a) Specimens have to be collected from the site containing necrotic tissue.
 - b) Pus is better than swabs.
 - c) Specimens has to be send to the laboratory within 1/2 hour why?
 - d) Fluid media like cooked meat broth are the best culture media.
 - e) Specimens have to incubated anaerobically for 48 hours.



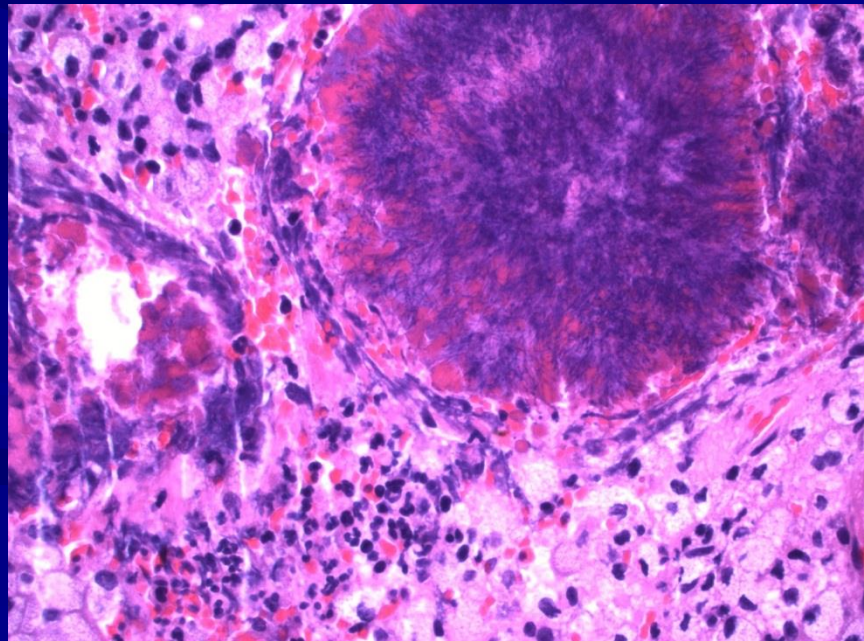
GasPak Envelope

Wire mesh containing
palladium catalyst



ACTINOMYCOSIS

- Actinomyces are branching anaerobic or microaerophilic Gram positive bacilli
- Source of the infection is normal flora and the host usually normal host
- Primary site of the infection is mouth, lung, appendix, uterus with IUD (chronic infection)
- Infection can spread to the brain, liver, bone and blood
- Diagnosis by Gram stain with sulfur granules and growth of molar tooth colonies
- Treatment penicillin, clindamycin or tetracycline



TREATMENT:

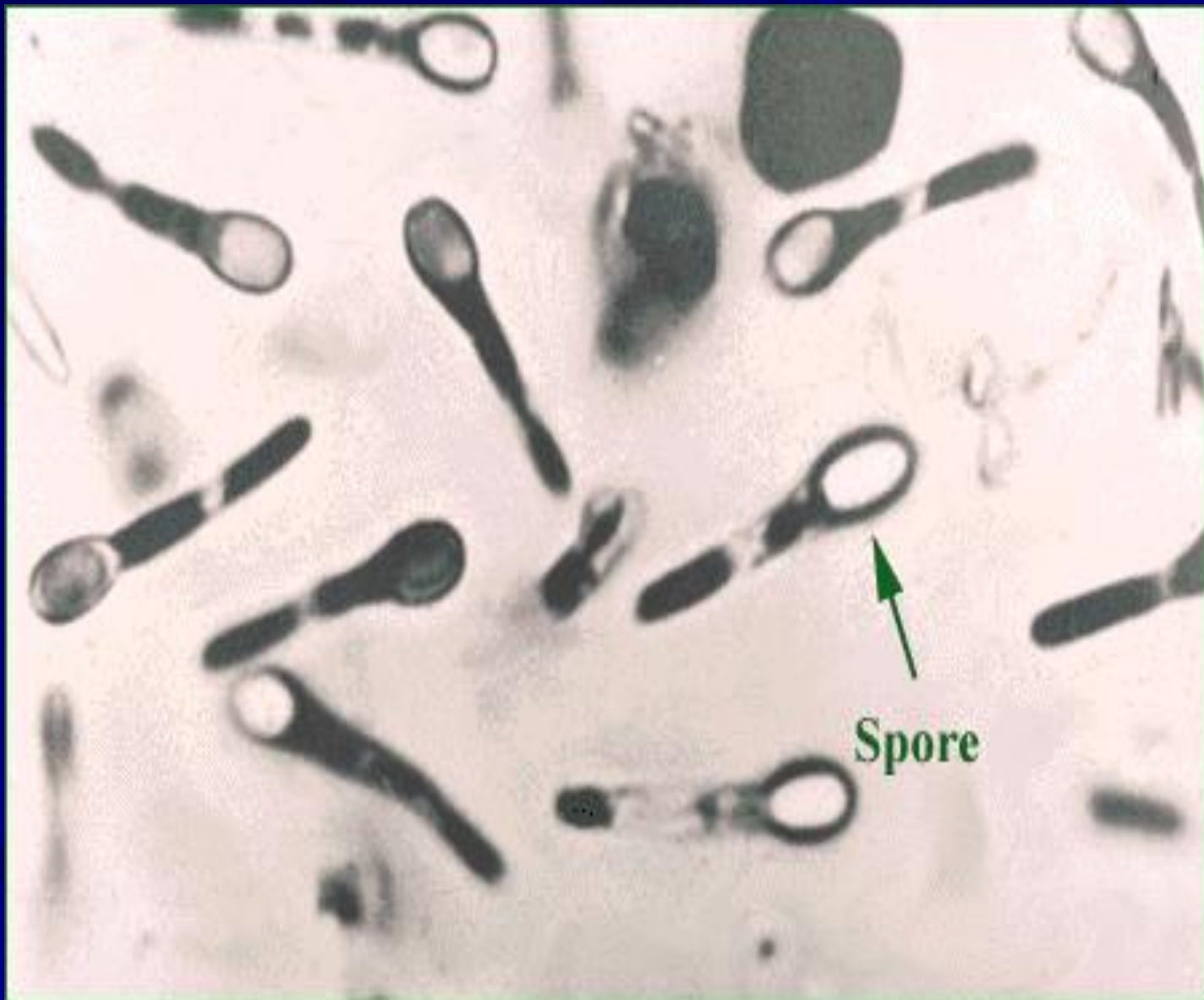
- *Bacteroides fragilis* is always resistant to penicillin.
- But penicillin can be used for other anaerobes
- Flagyl (metronidazole) is the drug of choice.
- Clindamycin can also be used.

TETANUS

TRIMUS

LOCKJAW 1884

Strict toxigenic disease





EPIDEMIOLOGY

- 1 Million/year > 60 yr .injection of drugs {young}
- 1/2 due to neonatal tetanus
- Cryptogenic t. {23%}
- Disease of non-immunized animals and humans {toxoid}

SOURCE

- Animals feaces {horses} ,soil
 - Contaminated wound {minor}
 - Compound fracture
 - Narcotic addicts
 - Unsterile injections
 - Burns , bites ,avulsions
 - Umbilical stump
- Face , neck , upper extremities wounds are more dangerous

TETANUS

■ PATHOGENESIS

- EXTOXIN
{TETANOSPASMIN}
- Presynaptic terminals of
LMN Inhibitory
impulses to MNs
- Persistent tonic spasm

■ Clinical picture

- Generalized
- Localized
- Cephalic
- Neonatal{
>90%}mortality
- IP 3-21 days









DIAGNOSIS

Clinical

Laboratory

{minor role}

TRATMENT

Supportive

Airway

Muscle relaxant

Wound care

Antitoxin

Antibiotics :MTZ , PG

TIG {500 UNITS}

PREVENTION

Complete active childhood immunization

Appropriate wound management

Type of wound

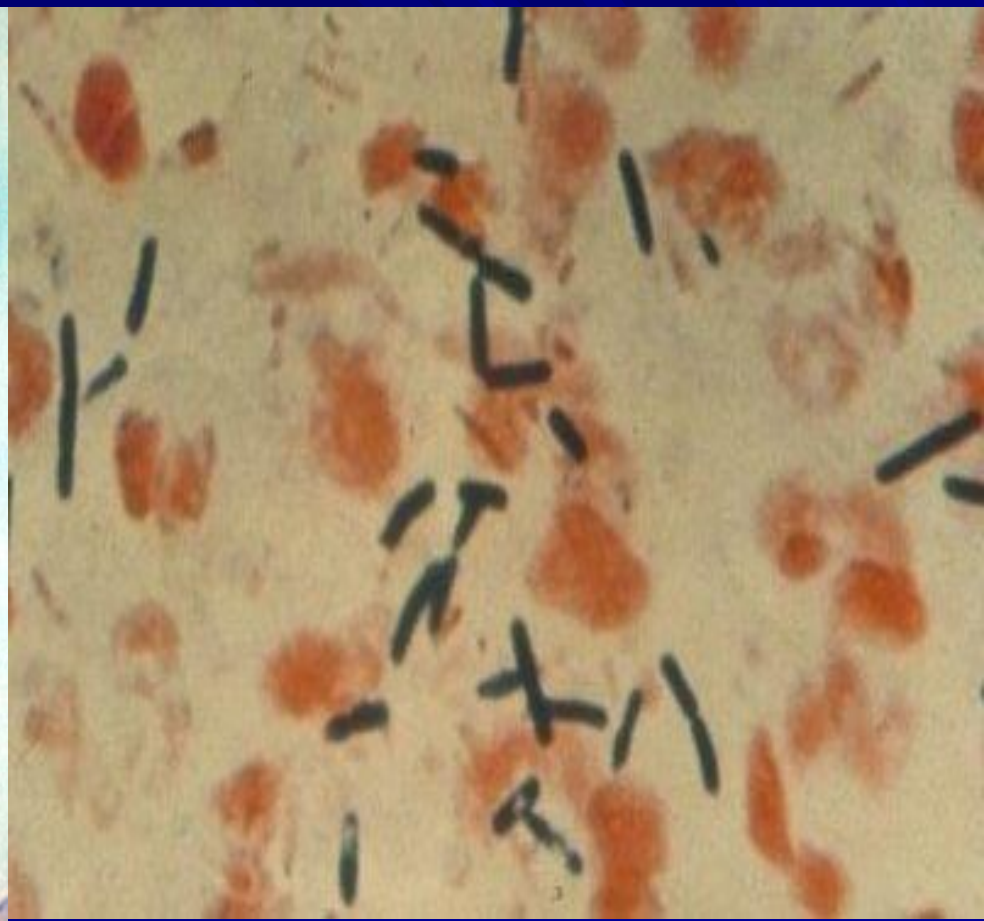
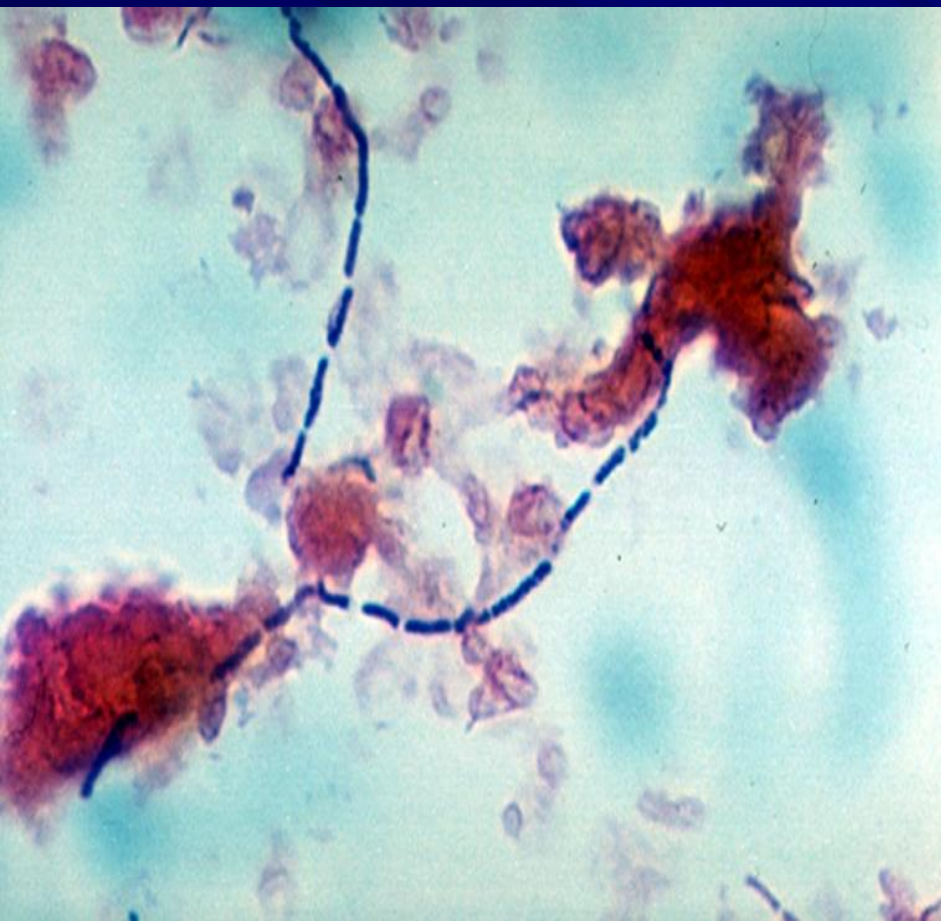
Immunization history

C. Perfringens {C. Welchii}

Histotoxic clostridia

Gas gangrene

Food Poisoning





NATURAL HABITATS

- Soil and intestinal tracts of animals and humans { 10^3 10^8 }
- Widespread occurrence
- Vagina of 1-9 % healthy women

CLINICAL SIGNIFICANCE

- Species most commonly isolated from clinical specimens
- Many clinical settings ranging from :-
 - Simple contamination of wounds – traumatic or non traumatic myonecrosis
 - C. Cellulitis
 - Intra-abdominal sepsis
 - Gangrenous cholecystitis
 - Post-abortion infections – septicemia
 - Bacteremia
 - Brain abscess

Gas gangrene

- Toxin mediated breakdown of muscle tissue
- Rapid progression { Uterus }
- Liquefactive necrosis of muscle , gas formation , toxemia
- Fulminant septicemia
- Intravascular hemolysis
- Hemoglobinuria
- Blood cultures positive in 15 % of patients



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PATHOGENESIS

- 5 – Toxins [A – E]
- Phospholipase C { alpha toxin }
 - Acts on membranes of muscle cells , leukocytes and platelets .
 - Play major role in the pathogenesis of C. myonecrosis
 - Has necrotizing activity
- Other toxins :- collagenase , proteinase , DNAs

Clinical picture

- Acute progressive pain , edema , skin discoloration
- Systemic – fever , tachycardia , hypotension , renal failure , crepitus , pulmonary edema , death

ETIOLOGY

- *C.perfringens* { 80% }
- *C.Novyi*
- *C.Septicum*
- *C.Histolyticum*

DIAGNOSIS

- CLINICAL

- SURGICAL

- MICRO.

- Gram stain :- G PB , absent leukocytes

- Culture { aerobic and anaerobic }

- Exudate , aspirates

- Tissue

- Blood

- Nagler reaction

FOOD POISONING

- One of most common bacterial causes of food –borne illness
- Sporadic cases and outbreaks
- Almost all due to type A
- Improperly cooked meat or meat product
- Ingestion of vegetative cells [10^8]
- Afebrile Crampy abdominal pain - diarrhea within 7-15 h
- Enterotoxin [SPORULATION]
- Mild illness , recovery after 2-3 days

TRATMENT

- Early and complete surgical excision of necrotic infected tissue { most important }
- High dose of :-
 - Penicillin G IV
 - Metronidazole
 - Clindamycin
- Management of shock , hemolysis , anemia

C . Difficile

Pseudomembranous colitis
Antimicrobial associated diarrhea
Hospital acquired diarrhea

Epidemiology

- *Clostridium difficile* causes **antibiotic associated diarrhea (AD)** and more serious intestinal conditions such as **colitis** and **pseudo membranous colitis** .
- Overgrowth of *Clostridium difficile* in the colon, usually after the normal flora has been disturbed by anti microbial chemotherapy

EPIDEMIOLOGY

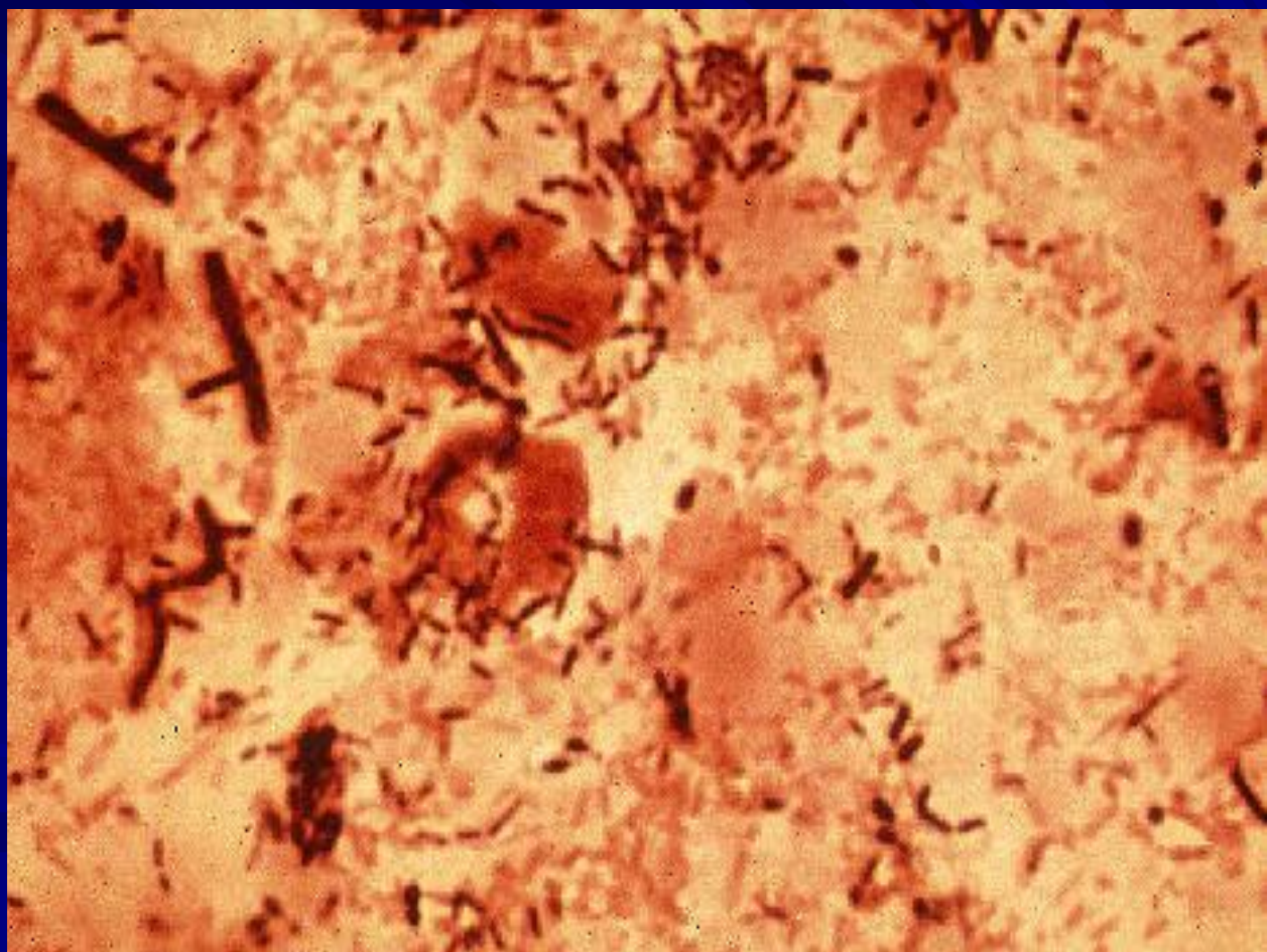
- Soil
- Human and animal feces
- Hospital environment {Reservoirs}
- Spores acquired –
 - Environment
 - Fecal – oral { colonized persons }
- Intestinal colonization rate
 - Healthy neonates , young infant [50 %]
 - Children > 2yrs , adults { 3 % }

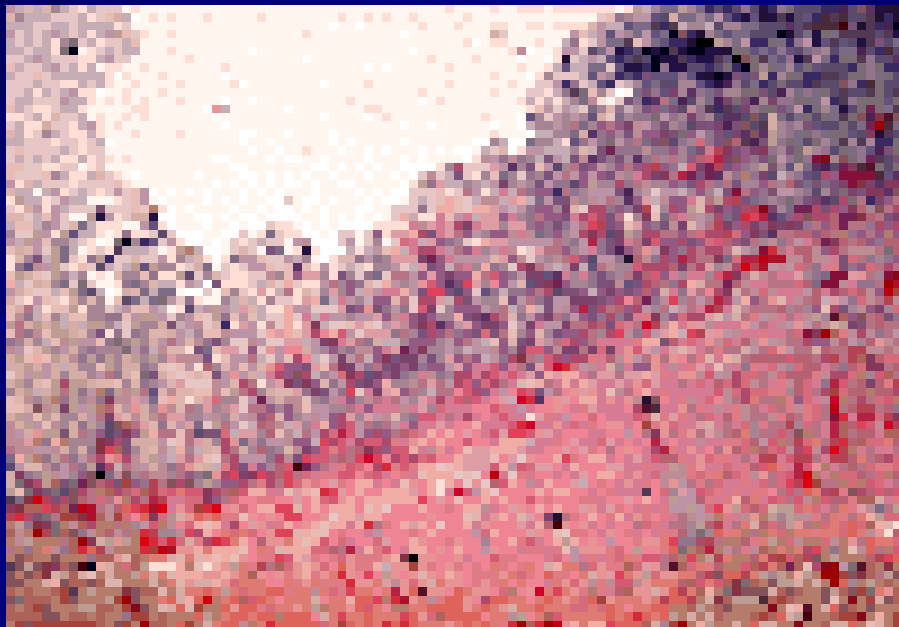
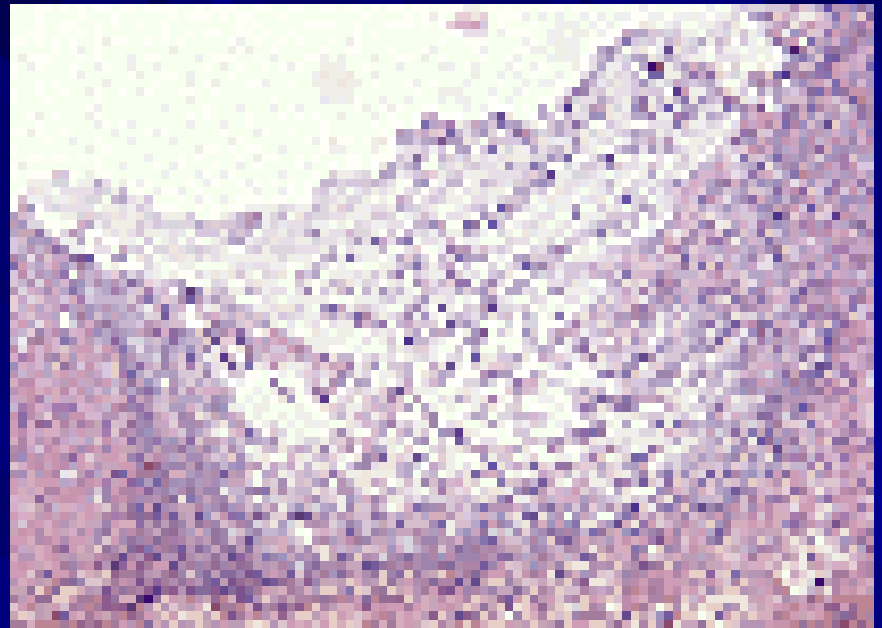
CLINICAL PICTURE

- Mild diarrhea , asymptomatic carriage – Toxic megacolon , bowel perforation and death
- Pseudomembranous colitis
 - Bloody diarrhea , abdominal cramps,
 - Fever , systemic toxicity
 - Colonic mucosa – yellowish plaques
- Severe disease – neutropenic , inflammatory bowel disease .

Control Measures

- Proper hand washing {contact precautions }
- Limiting use of antimicrobial agents
- Isolation of patients with diarrhea
- Disinfection of pt. rooms





TREATMENT

- Discontinue antimicrobial therapy { clinical significant diarrhea or colitis
- Antimicrobial therapy : severe toxicity , persistent diarrhea
- Metronidazole for 7-10 days , oral , IV
- Oral vancomycin : {emergence of VRE }
- 10-20 % relapse rate
- Antimotility drugs : contraindicated

Risk Factors

- Exposure to organisms
- Disturbed normal gut flora {proliferate – toxin}
 - Repeated enema
 - Prolonged NG tube
 - GI surgery
 - Bowel stasis
 - Antimicrobials : penicillins , clindamycin ,
Cephalosporins

PATHOGENESIS


■ TOXINS

● TOXIN A [Enterotoxin]

● TOXIN B [Cytotoxin] , more potent

■ Most strains produce both or no toxins

DIAGNOSIS

- Endoscopy : pseudomembranes and Hyperemic rectal mucosa
- Stool : toxins { EIA } , Cell culture  Confirm toxigenic strains
- Isolation of C. Difficile { not diagnostic }
- PCR

C.BOTULINUM

BOTULISM

TRANSMISSION

- SPORES

- VEGETABLES , MEATS , FISH

- CANNED FOOD

- PREFORMED TOXIN

**PATHOGENESIS
TOXIN (PHAGE)
MOST TOXIC SUBSTANCE**

GUT  **BLOOD** 
PERIPHERAL NERVE SYNAPSES 

BLOCKS RELEASE OF ACETYLCHLINE

 **FLACCID PARALYSIS**

CLINICAL

■ DESCENDING PARALYSIS

- DIPLOPIA

- DYSPHAGIA

- RESPIRATORY MUSCLE FAILURE

■ NO FEVER

■ WOUND , INFANT BOTULISM (honey)



Diagnosis : clinical (TOXIN ,FOOD SERUM)

■ **TREATMENT**

- ANTITOXIN
- A , B ,E

■ **RESPIRATORY
SUPPORT**

■ **PREVENTION**

- STERILIZATION
OF CANNED FOOD