

ANAEROBIC BACTERIA

Dr- Fawzia Al-otaibi

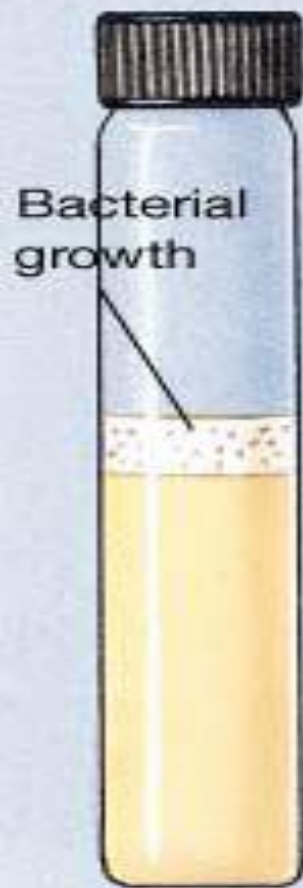
DEFENITION

■ A MICRBE THAT CAN ONLY GROW UNDER ANAROBIC CONDITION

■ SENSITIVE TO metronidazole (MTZ)

■ FAIL TO GROW IN AIR 10 % O₂





Bacterial growth

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 – GRAME POSITIVE COCCI

- *Peptococci*
- *Peptostreptococci*

■ C – GRAME POSITIVE BACILLI

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

■ D-MICROAEROPHILIC STREPT.

BACTEROIDES

- GROUP = *B. FRAGILIS*, *B. VULGARIS*,
B. THETAHOTAMICRON, *B. UNIFORMIS*
 - ACCOUNT FOR 1/3 OF ALL ISOLATES
 - RESISTANT TO 20% BILE
 - **RESISTANT TO MANY ANTIBIOTICS**
 - PENICILLIN, KANAMYCIN, VANCOMYCIN, COLISTIN – AND MANY MORE
 - NO PIGMENTATION OF COLONIES OR FLUORESCENCE

BACTEROIDES OTHER SP

■ BACTEROIDES SPECIES OTHER THAN B. FRAGILIS GROUP

- BILE SENSITIVE
- RESISTANT TO KANAMYCIN ONLY
- SOME PIGMENTED

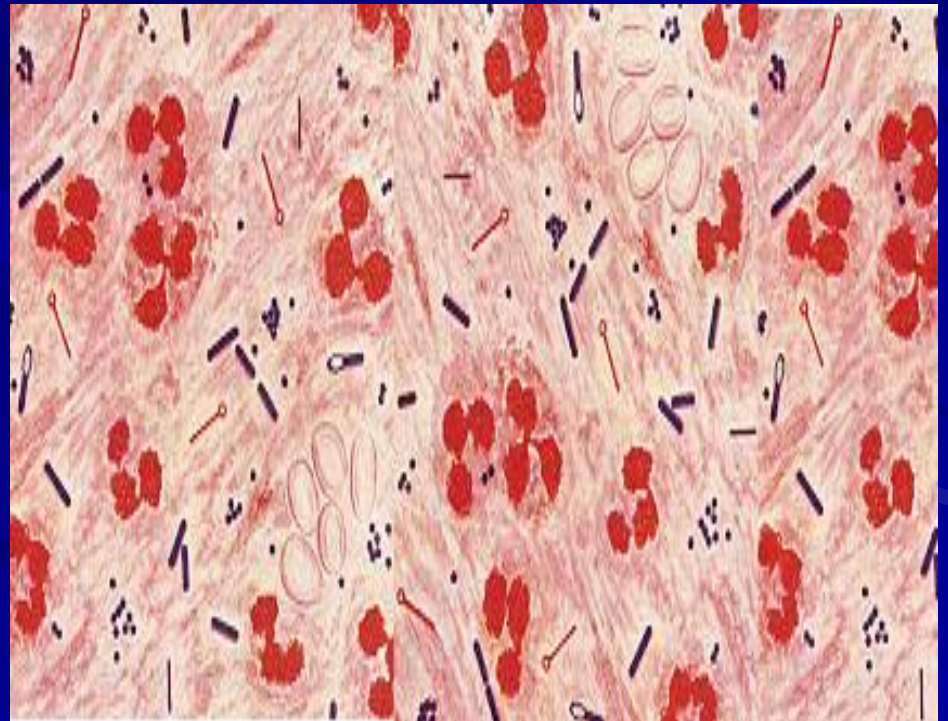
SPORE FORMING

■ 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*

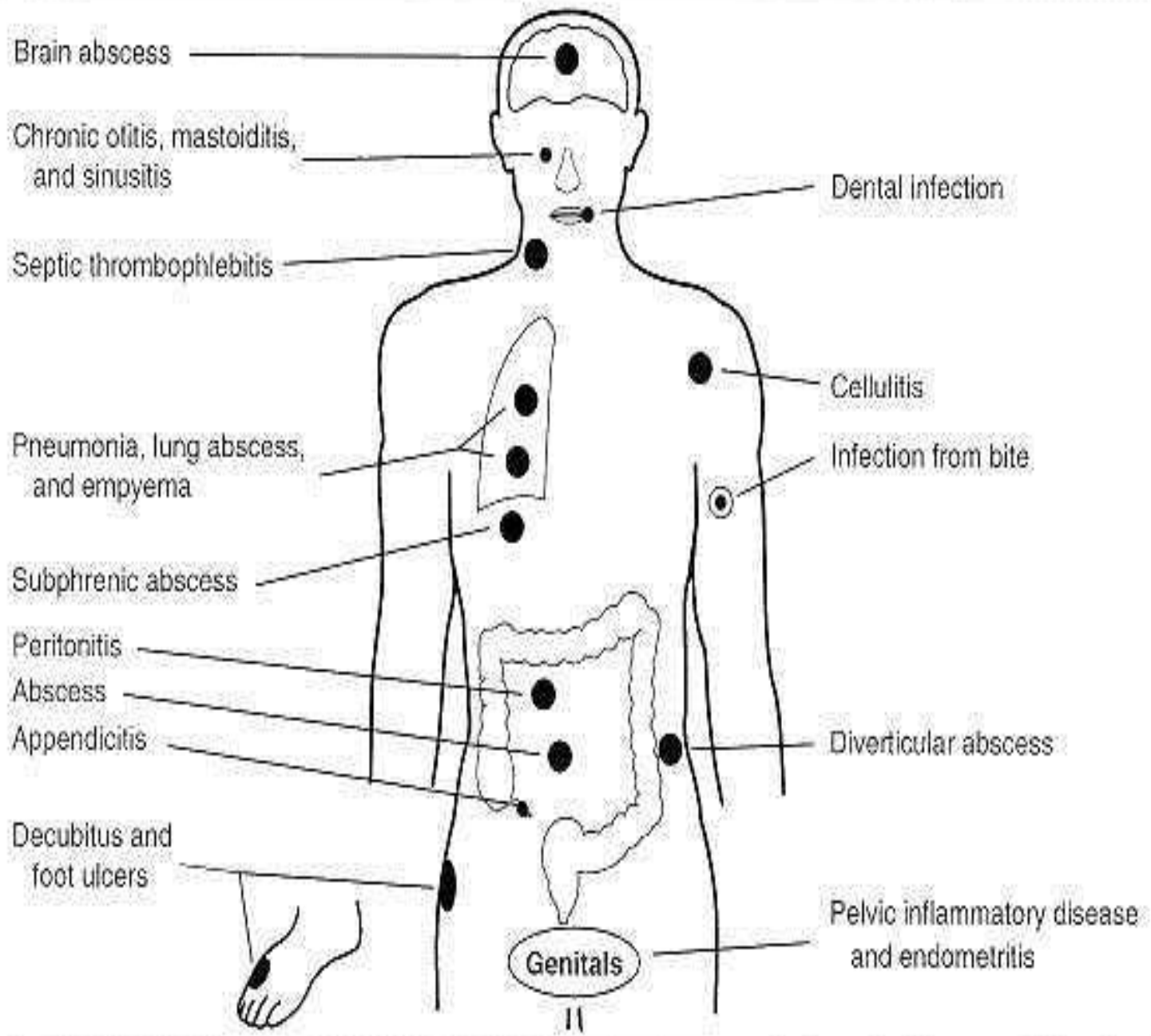
■ Female genital tract (mainly in the vagina)

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

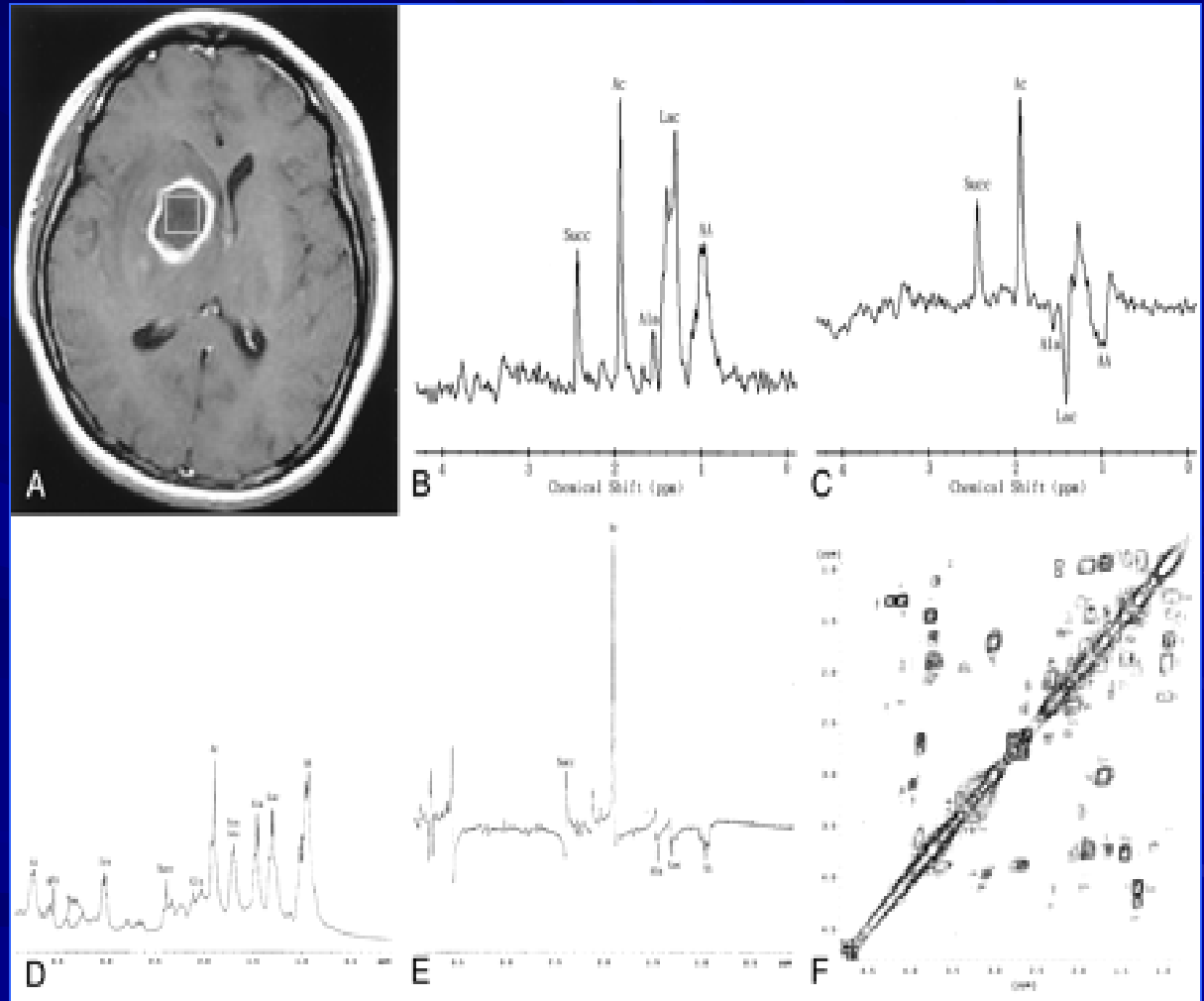


**Frequency with which Anaerobes
are Associated with Human Disease**

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.



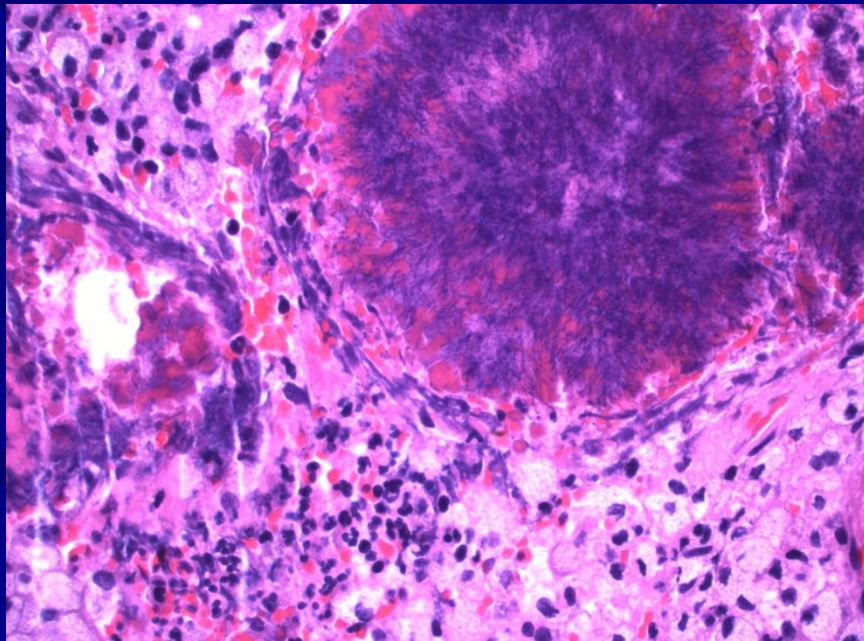
GasPak Envelope

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

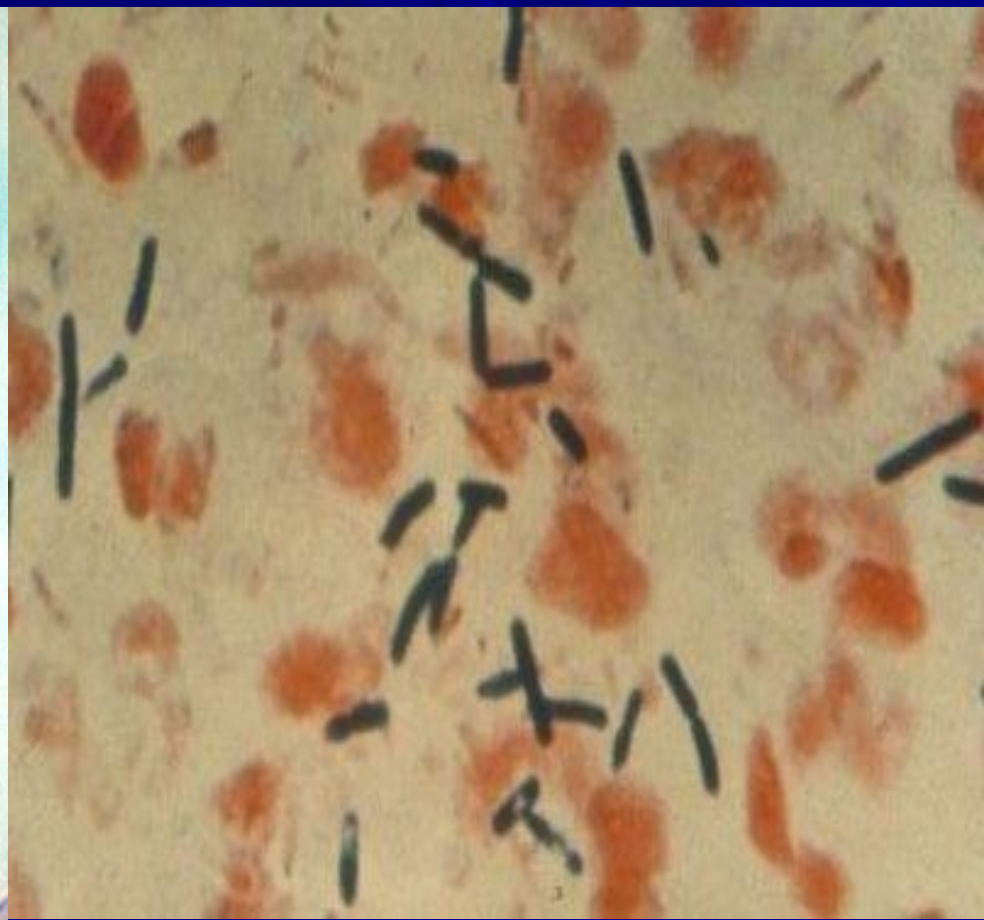
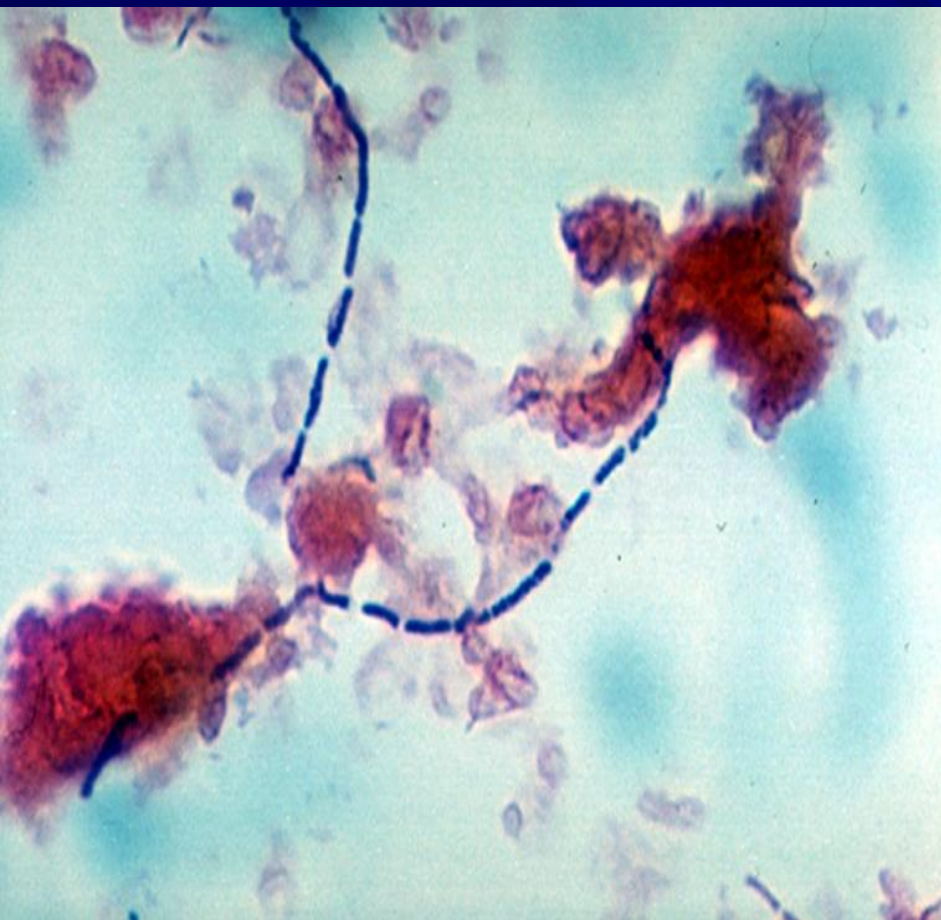
Immunization history

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









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

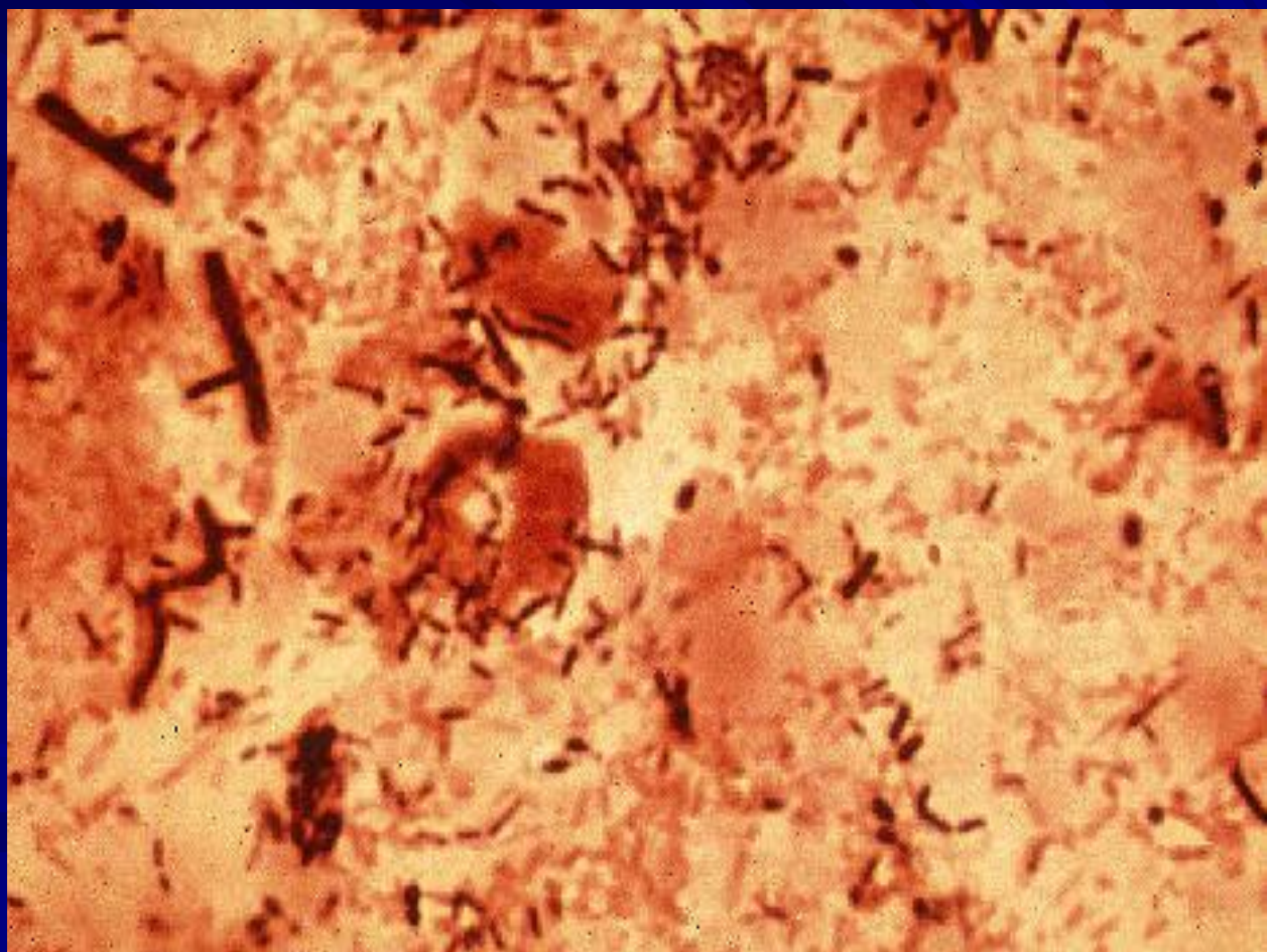
- Soil
- Human and animal feces
- Hospital environment {Reservoirs}
- Spores acquired –
 - Environment
 - Fecal – oral { colonized persons }
- Intestinal colonization rate
 - Healthy neonates , young infant [50 %]
 - Children > 2yrs , adults {3 % }

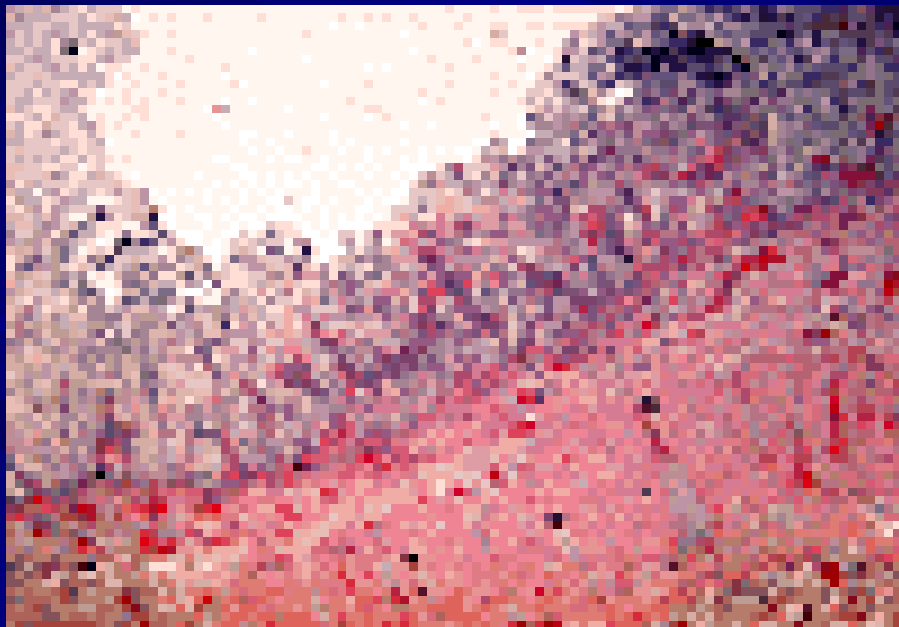
CLINICAL PICTURE

- 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