GAS GANGRENE

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INTRODUCTION

- AKA ***clostridial myonecrosis, malignant edema***

- During WW I and WW II, gas gangrene was found in 5% of wounds, but with improvement in wound care, antisepsis and the use of antibiotics the incidence had fallen to 0.1% hence also called previously as ***disease of war.***

- Nowadays, More commonly due to road traffic accidents with contaminated open wounds and crush injuries and gunshot wounds.

DEFINITION

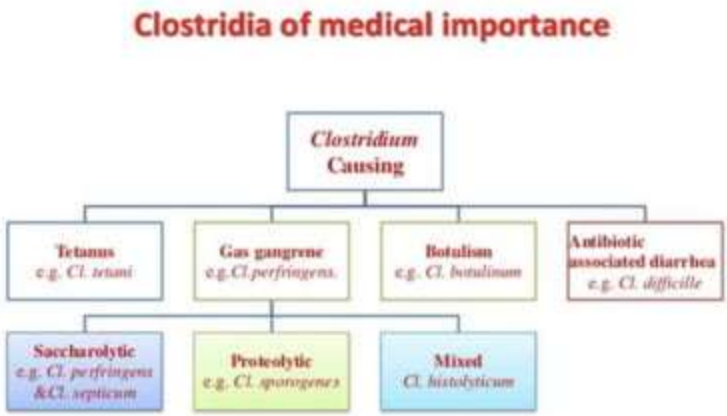
- It is an infective gangrene caused by clostridial organisms involving mainly skeletal muscle as oedematous myonecrosis.

ETIOLOGY AND RISK FACTORS

- POST TRAUMATIC
  - Road traffic accidents
  - Crush injuries
  - Severe Burn injuries and frost bite
  - Gun shot injuries
  - Contaminated Wounds

- POST SURGERY
  - Bowel resection surgeries
  - Hepatobiliary surgeries
  - Premature wound closures

MICROBIOLOGY



CAUSITIVE ORGANISM

CLOSTRIDIAL SPECIES

C. PERFRINGES (WELCHII) – 90%

C. NOVYI

C. SEPTICUM

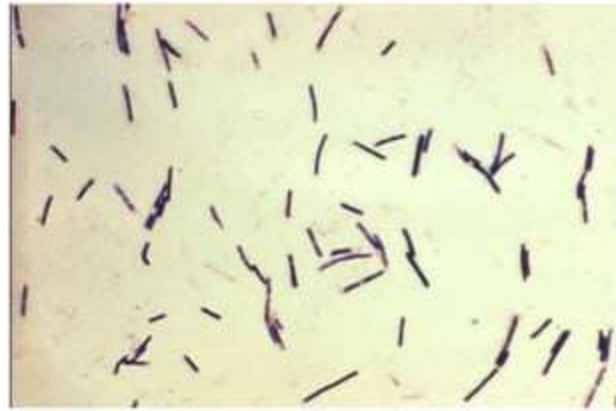
C. HISTOLYTICUM

OTHER SPECIES

- ESHERICHIA COLI
- PROTEUS
- PSEUDOMONAS AERUGINOSA
- KLEBSIELLA PNEUMONIA

CLOSTRIDIUM PERFRINGES

- GRAM POSITIVE
- ROD BACILLI
- SPORE FORMING (CENTRAL)
- CAPSULATED
- MOTILE
- ANAEROBIC
- SOURCE – SOIL, DEAD VEGETATIONS, GUT FLORA
- SEROTYPES – A,B,C,D,E



PATHOGENESIS

EXOTOXINS

***Clostridium welchii* produce toxins**

- Alpha (most common)
- Beta
- Epsilon
- Iota
- *Phi toxin*—myocardial depressant
- *Kappa toxin*—destruction of connective tissue and blood vessels
- **Bursting factor** and circulating factor

ENZYMES

- ***Lecithinase*** is important toxin which is haemolytic, membranolytic and necrotic causing ***extensive myositis.*** It splits lecithin into phosphocholine.
- ***Haemolysin*** causes extensive haemolysis.
- ***Hyaluronidase*** helps in rapid spread of gas gangrene.
- ***Proteinase*** causes breaking down of proteins in an infected tissue.

Spores enter through the devitalised tissues commonly in road traffic accidents, crush injury

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Spores germinate

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Released bacteria will multiply

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Exotoxins are released cause their effects

- Incubation period is 1-2 days.
- The gas is produced through glucose fermentation, and is usually composed of 5.9% hydrogen, 3.4% carbon dioxide, 74.5% nitrogen, 16.1% oxygen
- Muscle glycogen is broken down into lactic acid, CO2 and hydrogen. Proteinase released by organism forms amino acids which further releases ammonia and hydrogen sulphide.
- Clostridial infections are caused not by the extra virulence of the organism, but rather by unique local conditions, such as necrosis of muscles.

PRESENTATION

- Typically begins with the sudden appearance of pain in the region of the wound.
- Features of toxaemia, fever, tachycardia (out of proportion to fever) pallor.
- Wound is under tension with foul smelling discharge (sickly sweety/decaying apple odour).
- Khaki brown/black coloured skin due to haemolysis.
- Crepitus can be felt.
- Jaundice may be ominous sign and also oliguria signifies renal failure.
- Frequent sites are adductor region of the lower limb and buttocks and subscapular region in upper limb.
- Can infect limbs, abdominal wall, appendix, gallbladder, common bile duct, intestine, uterus (during septic abortion).

Types

- ***Fulminant type*** causes rapid progress and often death due to toxaemia, renal failure or liver failure or MODS or ARDS.
- ***Massive type*** involving whole of one limb containing fully dark coloured gas filled areas.
- ***Group type:*** Infection of one group of muscles, extensors of thigh, flexors of leg.
- ***Single muscle type*** affecting one single muscle.
- ***Subcutaneous type*** of gas gangrene involves only subcutaneous tissue (i.e. superficial involvement). It is mainly of *anaerobic cellulitis type* without muscle involvement usually caused by less virulent clostridial organisms other than clostridial welchii. It is usually superficial but may spread and involve fascial planes. It causes necrosis with foul smelling seropurulent discharge.

INVESTIGATIONS

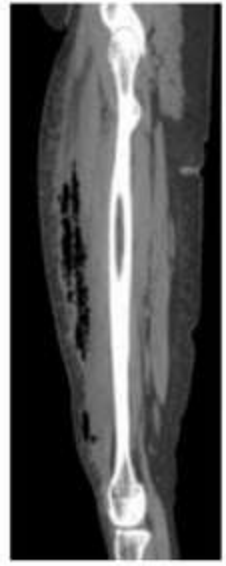
Diagnosis is on basis on clinical examination rather than on investigations

CLINICALLY

1. Septicemic patient
2. Cold and discolored extremities
3. Skin changes and blisters
4. Foul smelling , dark discharge
5. Abnormal peripheral pulses
6. Black and dead necrotic tissue if wound present

IMAGING

1. XRAY - shows gas in muscle plane or under the skin
2. USG – Gas in submuscular and subfascial planes
3. CT SCAN – Helpful, especially in abdominal cases of gas gangrene
4. MRI – To rule out other pathologies



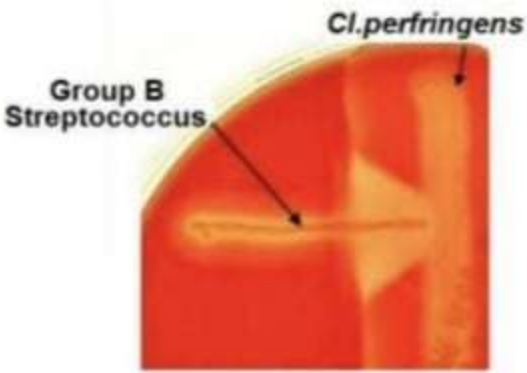
SEROLOGICAL

1. CBC – anemia and highly raised leucocytes
2. Renal function - deranged creatinine
3. Liver function – hypoproteinemia
4. Electrolyte imbalance – potassium loss
5. HHH – more chances in IV drug abusers
6. Serum LDH – deranged
7. Deranged coagulation profile – PT/INR, BT/CT

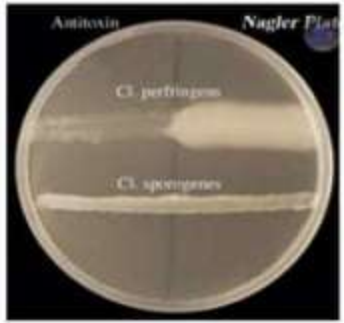
SPECIAL TESTS; TO ISOLATE THE CAUSATIVE ORAGNISM

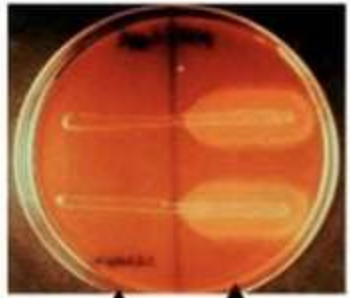
1. GRAM STAIN - POSITIVE
2. ROBERTSONS COOKED MEAT CULTURE – turns pink
3. BLOOD AGAR – TARGET HEMOLYSIS
4. cAMP TEST – PERPENDILAR TO ARROWHEAD HEMOLYSIS
5. NAGLERS REACTION
6. Ferments glucose, lactose and maltose

– Robertsons cooked meat broth


Cl.perfringens
Group B
Streptococcus

- Rapid detection of *Cl. perfringens* from clinical sample
- Done to detect the lecithinase activity of alpha toxin
- Characteristics opalescence is produced around colonies in +ve test due to breakdown of lipoprotein complex in the medium





+ Antitoxin No antitoxin

COMPLICATIONS

- Septicaemia, toxaemia.
- Renal failure
- liver failure.
- Circulatory failure
- MOD
- DVT
- secondary infection.
- Death occurs in critically ill patients.

DIFFERENTIALS

- NECROTIZING FASCITIS
- CELLULITIS

PREVENTION

- Proper debridement of devitalised crushed wounds
- Devitalised wounds should not be sutured
- Adequate cleaning of the wounds with H2O2 and normal saline
- Prophylactic antibiotic.

MANAGEMENT

- The combination of aggressive surgical debridement and effective antibiotic therapy is the determining factor for successful treatment of gas gangrene.

MEDICAL MANAGEMENT

1. Penicillin G in dosages of 10-24 million U/d was the drug of choice.

- A combination of penicillin and clindamycin
- Large intravenous doses of penicillin, 3 million units every 3 hours, should be administered.
- Combination of clindamycin and metronidazole is a good choice for patients allergic to penicillin.
- Third generation cephalorsporin

ADJUVANTS

2. Polyvalent antiserum 25,000 units given intrav enously after a test dose and repeated after 6 hours (Welchii 10,000 IU, oedematiens 10,000 IU, and septicum 5,000 IU).

3. Hyperbaric Oxygen therapy – administration of 100% oxygen at 2.5-3 absolute atmospheres for 90-120 minutes 3 times a day for 48 hours, then twice a day as needed.

4. Rehydration and maintaining optimum urine output (30 ml/hour) (0.5 ml/kg/hour) and monitoring serum electrolytes, calcium, coagulation profiles and renal profile.

SURGICAL MANAGEMENT

Surgical decompression and debridement

- Each muscle should be carefully inspected and if infected, it should be resected to the point of origin.
- All dissection should be done without tourniquet to allow continuous oxygen to living tissue.
- Copious irrigation should be performed with sterile normal saline solutions and/or 3% liquid hydrogen peroxide
- If the wounds were treated elsewhere and closed, it is safest to reopen them, clean them, and leave them open with negative-pressure wound dressing therapy (if available) or just a sterile dressing.
- Fasciotomy, excision of dead tissue, and if necessary amputation of affected part

- All wounds should be left open at least for 48 hours.
- Repeated if necessary on daily basis.