BACTERAEMIA AND SEPTICAEMIA

- Are unusual in superficial wound infections but common after anastomotic breakdown.
- Usually transient and follow procedures undertaken through infected tissues (particularly instrumentation in infected bile or urine).
- Bacteraemia is important when prosthetics have been implanted, particularly cardiac valves.

- Septicaemia commonly relates to colonisation and translocation of bacteria in the gastrointestinal tract due to anastomotic breakdown or perforation of gut accompanied by MSOF.
- Aerobic Gram-negative bacilli are mainly responsible but S.*aureus* and fungi may also be involved, particularly after the use of broad-spectrum antibiotics.
- Patients who are immunocompromised, diabetic or have malignant disease are at risk, particularly when anaerobic wound conditions are present with necrotic or foreign material.

- Wound infections are associated with severe local wound pain and crepitus (gas in the tissues which may also be noted on plain radiographs).
- The wound presents a thin, brown, sweet-smelling exudate, from which bacteria can be recognised on Gram’s staining.

Specific wound infections

GAS GANGRENE :

(Infectious gangrene of muscles, Clostridial myositis, Clostridial myonecrosis) :

- Highly fatal, rapidly speading infection caused by clostridial organisms which results in myonecrosis. Earlier it was called as **malignant oedema.**
- Gas gangrene wound infections are associated with severe local wound pain and crepitus (Gas in the tissues)

- The wound produces a thin, brown, sickly sweet smelling exudate, in which Gram positive – bacteria present.
- Early systemic complications with circulatory collapse and MSOF follow if prompt action is not taken.
- The organisms responsible for gas gangrene divided into two groups.

1. **SACCHAROLYTIC -** Those break down starch (60%)

   Ex: Cl. perfringens ,Cl. Welchii, Cl. Aerogenous capsulate.

2. **PROTEOLYTIC** - Those break down protein (40%)

   Ex: Cl. Sponogenes, Cl.Septicum, Cl.oedematiens.

   Cl.Histolyticum

- These are gram positive anaerobe organisms bearing central spore, produce exotoxins which are responsible for muscle necrosis and gas production.

Toxins produced by clostridium perfringens:

- Alpha
- Beta
- Epsilon
- Iota
- Phi toxin - Myocardial depressant
- Kappa toxin – Destruction of connective tissue and blood vessels

EXOTOXINS:

- **Lecithinase** – Most important toxin which is haemolytic, membranolytic and necrotic causing extensive myositis.
- **Haemolysin** – Causes extensive haemolysis.
- **Hyaluronidase** – It helps in rapid spread of gas gangrene by dissolving the intracellular adhesive substances.
- **Proteinase** – Breaking down the proteins in the infected tissue.

EFFECTS OF TOXINS:

- Extensive necrosis of muscles with production of gas (hydrogen sulphide; nitrogen; carbon dioxide) which stains the muscle as brown or black.
- Usually necrosis of muscle involves from origin to insertion.
- Often disease may extend into thoracic and abdominal muscles.
- When it affects the liver, causes necrosis with frothy blood – **foaming liver**, is characteristic.

Source of infection :

- Manured soil or cultivated soil, large intestine.
- High velocity gun shot wounds with sucking entry wound, leaving clothing and environmental soil in the wound in addition to devascularised tissue.

Risk group :

- Patients who are immunocompromised, diabetic or having malignant disease are at greater risk, particularly if they have wounds containing necrotic or foreign material in anaerobic conditions.
- Patients who underwent amputations for ischaemic gangrene of lower limbs, frequently contaminated with their own fecal matter contain gas gangrene organisms.

Pathogenesis :

- Gas gangrene develops in wounds containing foreign bodies associated with laceration and devitalised muscle mass contaminated with manured soil, fecal matter containing sporse of cl.Perfringens.
- Having entered the wound, under favourable conditions like low oxygen tension (Anoxia) dead and devitilised tissue, sporse germinate, multiply, and produce powerful exotoxins.

- Once powerful toxins start acting, various pathological events leads to inflammation, oedema, muscle necrosis and gangrene of the muscles take place.
- In untreated cases, necrotic process continues, septicaemia, renal failure, peripheral circulatory failure and death occur. Foarmy liver is a condition wherein gas accumulated in the liver, as a part of septicaemia.

Pathological changes

Injury ——————

Road traffic accident
soil, foreign body, clothing, clostridial organisms

↓

Spreading anaerobic cellulitis

↓

Multiplication of organanisms

↓

Release of toxins

↓

Breakdown of muscle collagen, change in the muscle's color lost contractility (Ischaemia – dull red, necrosis – - green, gangrene black color

↓

Production of gas by the bacteria – H_2S, *Ammonia*, Nitrogen

↓

Extensive edema with collection of gas in tissue planes

↓

Damage to the blood vessels

↓

Myonecrosis

↓

Gangrene of the limb muscles with spreading infection

↓

Septicaemia and death

CLINICAL FEATURE

- *Incubation period 1 to 2 days*

General features : *Signs of Toxaemia- high grade fever, tachycardia, pallor, hypotension, vomiting, oliguria*

Local features : *Severe pain and gross oedema of the wound with thin, brown, sickly sweet smelling exudate.*

- *If the wound was sutured, sutured line is under tension.*
- *The skin is Khaki brown colour due to haemolysis.*

CREPITUS can be felt due to presence of gas produced by bacteria in tissue planes.

- Colour changes in the muscles due to ischaemia.
- Jaundice may be present if liver is involved.
- Oliguria signifies renal failure

CLINICAL TYPES

- **Fulminant type** causes rapid progress and often death due to toxaemia, renal failure or liver failure leads to 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 or, flexors.
- Single muscle type affecting one single muscle.
- **Subcutaneous type** of gas gangrene involves only subcutaneous tissue (i.e., superficial involvement)

Palpable crepitus – Conditions:

1. Anaerobic infections.
2. Streptococcal infection.
3. Surgical emphysema due to oesophageal, tracheal rupture.
4. Gas gangrene.

INVESTIGATIONS

- X-ray shows gas in muscle plane or under the skin.
- Blood examination for total count, differential count, liver function tests, blood urea, serum creatinine, PO_2, PCO_2 & serum electrolytes.
- Microscopic examination of pus or exudate from the wound reveals gram-positive, spore-bearing organisms.

Prophylaxis :

Highly fetal gas gangrene can be prevented :

1. ***DEBRIDEMENT :***

- ***Clean the wound thoroughly with running water and hydrogen paroxide and with normal saline, apply antibiotic solution, dress the wound loosely..***
- ***Remove all dead, necrotic tissue, bone pieces and foreign material.***
- ***Drain the pus***
- ***NEVER SUTURED THE WOUND WHEN IN DOUBT***
- ***For postoperative wounds, remove the sutures, wide open the wound , drain the pus, excise the dead and necrotic tissue.***

2. PROPHYLACITIC ANTIBIOTICS :

- **Clostridia group of organisms are sensitive penicillian. Injection crystalline penicillian 10-20 lakhs units to be given every 4-6th hourly for a period of 7-10 days.**

3. JUDICIOUS AND MINIMAL USE OF TOURNIQUET :

- **If possible avoid tourniquet in limb surgeries to avoid tissue ischemia.**

4. GENTILE BUT EFFECTIVE APPLICATION OF PLASTER OF CASTE

To avoid compression of blood vessels

Treatment of Established Gas Gangrene :

- Emergency surgery which includes excision of all dead muscles and necrotic tissues by using generous incisions. Debridement is done until healthy tissue bleeds.
- *In severe fulmanent cases life saving procedures - amputation of limb has to be done and stump should never be closed.*
- Injection crystalline Penicillin 10-20 lakhs units, 4-6 hourly to be given for period of 7-10 days. Along with injection clindamycin 600 mgs per day. Injection Metronidazone 500 mgs. 8^{th} hourly and third generation cephalosporins.

- *Fresh Blood transfusions if patient is hypotensive or anaemic.*
- Polyvalent anti gas gangrene serum, 25000 units given *I.V. after test dose, to be repeated after 6 hours.*
- Hyperbaric oxygen - *Provide aerobic environment is useful.*
- Do not hesitate to amputate the limb in if it saves the life.

- Control dehydration by I.V. fluids so as to maintain minimum urine output of 0.5ml/kg/hour
- Ventilator support if abdominal or thoracic muscles are involved.
- *Ward and operation theatre used for the patient should be fumigated for 24-48 hours properly to prevent spread of infection to other patients.*

Summary :

- Proper prophlaxis and wound care.
- *Passive immunisation.*
- Control infection.
- Conduct operation.
- Administer hyperbaric oxygen.
- *Treat dehydration.*
- Give blood transfusion.

TETANUS

- Tetnus – An acute disease induced by the exotoxin of clostridium tetani and clinically characterized by muscular rigidity which persists throughout illness associated with painful paroxysmal spasms of the voluntary muscles, especially the masseters (Trismus or lockjaw), the facial muscles (Rises sardonicus), muscles of back of neck of trunk (Opisthotones) and those of the lower limbs and abdomen.

Aetiopathogenesis :

- Tetanus is caused by clostridium tetani Gram+anaerobic
- terminal spore forming bacillus (Drumstick appearance).
- Incubation period vary from few days to months or years.

CLOSTRIDIUM

C. perfringens: Gas gangrene; food poisoning

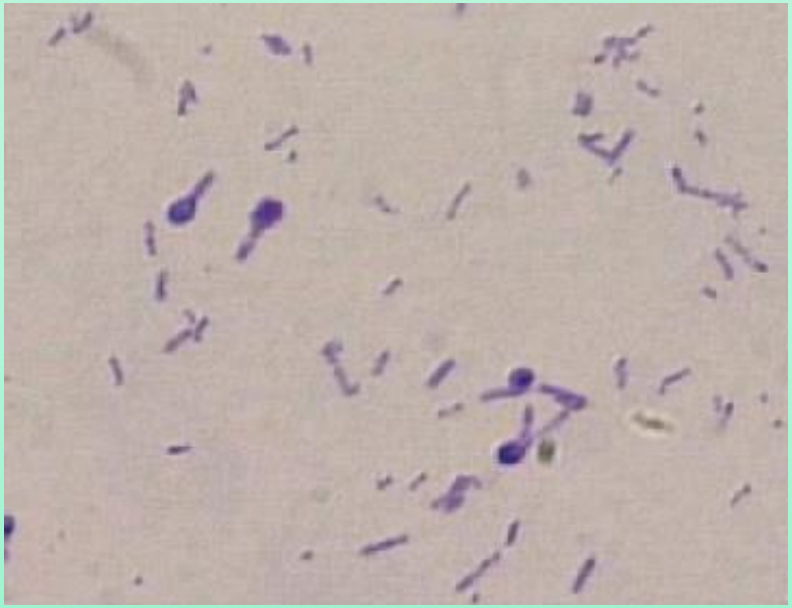
C. Tetani: Tetanus

C. Botulinum: Botulism

C. Difficile: Pseudomembranous colitis

PHYSIOLOGY AND STRUCTURE

- Anaerobic.
- Large gram-positive rods.
- The spores are usually wider than the rods, and are located terminally or subterminally.
- Most clostridia are motile by peritrichous flagella.



Possible routes of infection are :

- Umbilical cord in neonates, seen in communities which practice cowdung application on the umbilical stump.
- Wound - As a complication of road traffic accidents anaerobic conditions produced by the others aerobic organisms introduced at the time of injury.
- Minor injuries with rusted nails, piercing of the ear lobes, tattooing, injections etc.

- Endogenous infection after septic abortion or surgical operation on G.I tract.
- Tetanus due to infection acquired in the operation theatre.
- Thus tetanus is a wound infection, no wound, no tetanus.
- The disease produced wholly by exotoxins. The organism itself does not damage tissue.

The toxins produced by organism are :

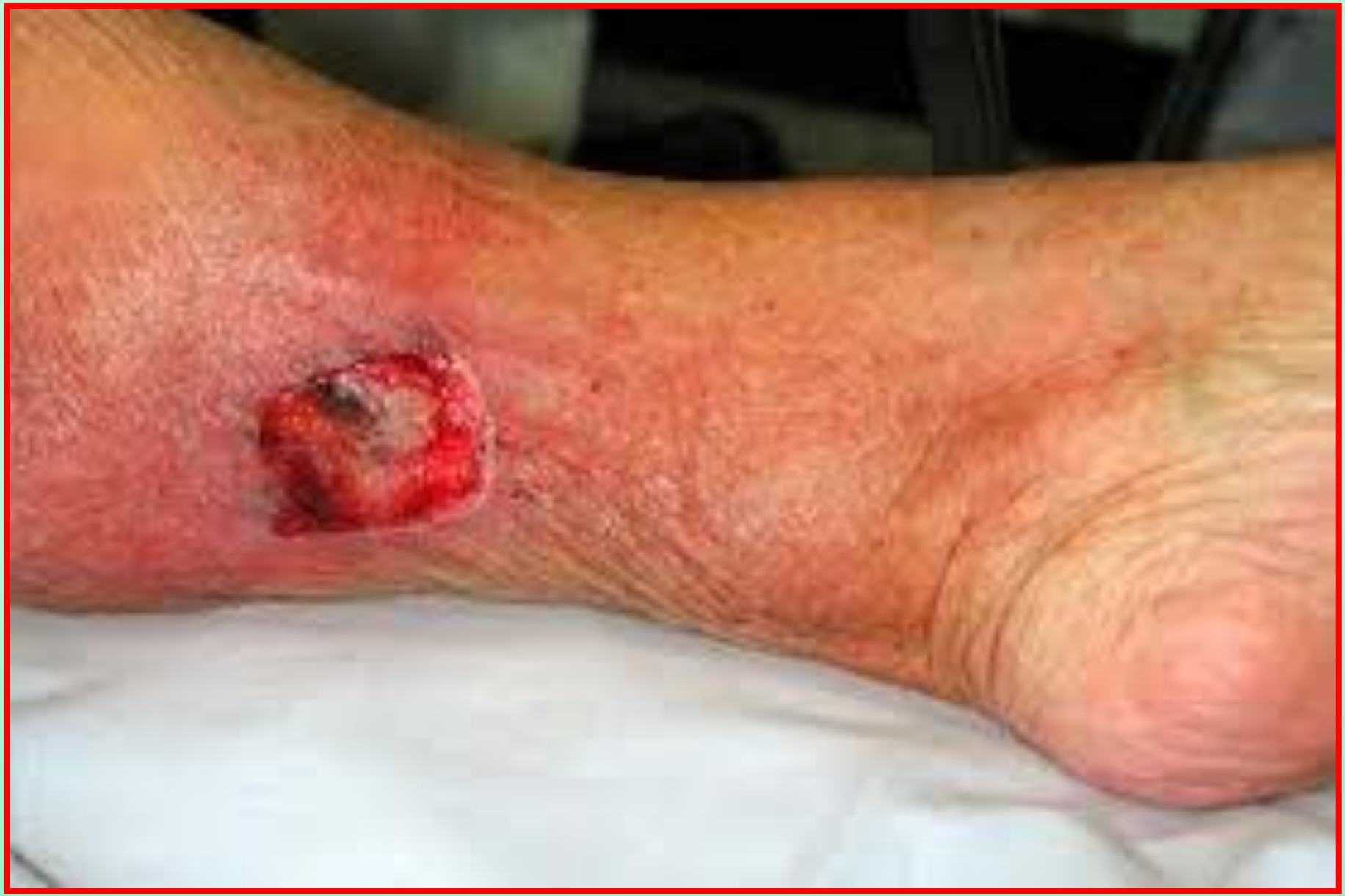
- Tetanospasmin (Neurotoxin).
- Tetanolysin (Haemolysin).
- Tatanospasmin has affinity to wards nervous tissues.
- It reaches central nervous system along the axons of motor nerve trunk.
- The toxin gets fixed to the anterior horn cells.
- The toxin thus fixed acts in two ways.

1. It inhibits the release of cholinesterase which causes accumulation of acetylcholine at the motor end plate, which is responsible for tonic rigidity of the limb, trunk, abdominal and neck muscles.
2. It causes extreme hyper excitability of motor neurone in the anterior.

- Horn cells of spinal cord.

- As a result explosive, wide spread reflex spasm of muscles in response to minor stimulation, the toxin thus fixed to the tissues can not be neutralized by antitoxin.
- Only free toxin can be neutralised.
- ***Factors responsible for development of tetanus :***

1. No immunisation.
2. Foreign body.
3. Injury.
4. Improper sterilisation.
5. Devitalised tissues.
6. Anaerobic conditions.



Clinical feature :

- The first symptoms are difficulty in swallowing trismus (or) lock jaw stiffness of Jaw) succeed by pain in the neck, back & Abdomen.
- Then tonic muscle spasm relatively sparing the limb.
- The sardonic smile (Risus sordon's of tetanus) at this stage of the disease.
- There after reflex convulsion can occur affecting all the muscles, causing great pain opisthotonus, and some times muscle rupture (Psoas, rectus abdominus, pectoral muscles).

- These spasms are spontaneous (or) may be induced by trivial Stimuli of nose,movement (Dhanurvatha).
- If severe, they stop respiration and patient becomes cyanotic.
- A cyanotic tetanic convulsion threatens life tonic muscular spasm remains between the reflex attacks thus distinguishing tetanus from strychnine poisoning.
- Period of on set is time between the first symptom and the first reflex spasm.
- If this period is less than 48 hours death is likely.
- The prognosis improves as the period increase.

Symptoms & Signs	Differential diagnosis
Trismus or lock jaw	Alveolar abscess or T.M joints Involvement
Dysphagia	Tonsillitis
Neck rigidity	Meningitis
Rigidity of back muscles	Orthopaedic disorder
Risus sardonicus	Anxiety neurosis
Generalised convulsions	Epilepsy
Mild temperature and tachycarida	Sympathetic hyperactivity

Patient is conscious thought out even before death.

Types of tetanus :

Tetanus neonatorum :

- It occurs due to contamination of umbilical cord in children born to non-immunized mothers.
- It manifests usually around 6-8 days of birth and is called as eight day disease. It carries almost 100% mortality.

Local tetanus:

- In this, contraction of muscles occurs in the neighborhood of the wound.

Cephalic tetanus :

- Usually occurs after wound of head and face.
- Cranial nerves like facial nerve and oculomotor nerve can get paralysed. It carried poor prognosis.

Bulbar tetanus:

- It is a condition where in muscles of deglutition and respiration are involved. It is fatal

Latent tetanus:

- It develops after few months to year following a wound which might have been forgotten.

Treatment :

Prophylactic:

- Active immunity is confessed by injection of adsorbed toxoid.
- 1 ml administered IM and repeated after intervals of six weeks and six months followed by booster dose every 5 years.
- If such an active immunity is present an extra dose is given at the time of injury.

- *Tetanous neonatorum and puerperal tetanus can be prevented by immunising the mothers with two tetanus toxoid injections of 0.5ml IM given in the third trimester of pregnancy infants and children immunised with tetanus toxoid, diphtheria and pertussis vaccine (DPT, three doses at 6,10and 14 weeks of age.*
- *A booster dose is given at 18 months and school going time (5years) and once in 5 years 1ml of tetanous todoid is given to achieve a active immunity.*

Puerperal tetanus :

- It occurs as a complication of abortion or puerperal sepsis.

Postoperative tetanus :

- Occurs due to improper sterilisation of instruments and carries 100% mortality.

Otitis tetanus :

- It is due to chronic suppurative otitis media.
- In these case, the wound is a tear in the tympanic membrane.
- It can occur in any age group but commonly in children and young adults.

Trematic tetnus :

Due to contaminated injury.

Idiopathic :

- Microscopic trauma
- *Absorption of toxin from GIT.*
- Inhalation of tetnus spores

Management of wound:

- Wash the wound thoroughly with plenty of water.
- Clean the wound with hydrozen peroxide.
- Apply antibiotic ointment dress the wound loosely.

Types of wounds:

1. Superficial & clean wounds
2. Superficial or deep contaminated and perforating, deep seated wounds.

Assessment of Vaccination status of patient:

A. Has complete course of vaccination and has a booster dose within 5 years.

B. Has complete course of vaccination but booster dose has taken more than 5 years back.

C. Has complete course of vaccination but booster dose has taken more than 10 years back

D. Has complete course of TT vaccination booster dose more than 10 years back or has not completed the course of vaccination or immune status not known.

VACCINATION SCHEDULE

Immune status	Clean wounds	Contaminated wounds
Group-A	No active immunisation	No active immunisation
Group-B	One booster dose of Inj. TT . 0.5ml	One booster dose of Inj. TT . 0.5ml
Group-C	One booster dose of Inj. TT . 0.5ml	One booster dose of Inj. TT . 0.5ml + Passive immunisation with ATS
Group-D	Complete course of vaccination i.e., 3 doses of Inj.TT with a gap of one month + Passive immunisation with Inj. ATS	Complete course of vaccination i.e., 3 doses of Inj.TT with a gap of one month + Passive immunisation with Inj. ATS

Passive immunity :

- Every patient with potentially dangerous wounds who has no active immunity must be given A.T.S. (IM) 1500 international units of A.T.S (Anti Tetanus Serum) given with due precautions.
- There after he must have active immunisation with toxoid.
- Many people develop immunity to horse serum. And subsequent use of the same in next accident may not help.
- In such a case large doses of ATS 50,000 units should be given, repeated every other day.

- Human anti-tetanus globulin (ATG): It is an autologus autotoxin and its protective value is hundred times that of ATS.
- 250 units of ATG IM will give adequate passive immunity.
- Use of penicillin & metronidazole in preventing tetanus is doubtful but given to prevent wound infection by other organisms.
- The spores are not effected by antibiotics it is capable of surviving until antibiotic disappears.

Treatment of Established Tetanus :

1. General management.
2. Specific management.

General management :

- Admission and isolation in a quiet room, to avoid minor stimuli which precipitate spasm.
- Wound care which includes drainage of pus, excision of necrotic tissue, removal of foreign body and proper dressing
- Inj. Tetanus toxiod 0.5ml to be given IM.

- Antitetanus serum (ATS) 50,000 units intramuscular (I.M) an 50,000 Units intravenous (I.V). Test dose should be given as ATS is associated with anaphylactic shock in 35% patients.
- Dilute small amounts of ATS with normal saline 10 times inject 0.2ml.
- Subcuteneously after1/2 an hour inject 0.8ml, if no reaction inject the total dose.

- Instead of ATS human anti tetanus globulin is better and safe. It does not cause anaphyulaxis. It is given in the dose of 3000 to 4000 units I.V. no test dose is required.
- Inj. Crystalline penicillin 10 lakh units every 6 hours is the drug of choice against clostridium tetani. It may have to be given for a period of 7-10-days.
- Metronidazole :- 500mg IV 8^{th} hourly for 10 days.
- It has been shown to be more effective than penicillin.
- After recovery full active immunisation with tetanus toxiod is a must.

Mild cases :

- There is only tonic rigidity with out spasm or dysphasia.
- These patients are managed by giving heavy sedation by using combination of drugs so as to prevent spasm or convulsions.
- Benzodiazepines and morphine act centrally to minimize the effects of tetanospasmin.
- Chlorpromazine being receptor blocker, can decrease sympathetic activity, other blockers such as phenoxybenzamine, phentolamine also have been used.

- These drugs are administered in such a way that every two hours the patient receives some sedative.
- The dosage of drugs are adjusted once in 2-3 days so as to get maximum effect of sedation or relaxation.
- Injection diazepam 10mg, tracheostomy set, resuscitation set like aryngoscope and endotracheal tube should be kept ready by the side of the patient.

Drug	Dosage	Time
Chlorpromazine	50-100 mg	8 AM - 2 PM - 8 PM - 2 AM
Phenobarbitone	30-60 mg	10 AM - 4 PM - 10 PM - 4 PM
Diazepam	10-20 mg	12 Noon - 6 PM - 12 MN - 6 AM

Seriously ill cases :

- They have dysphagia and reflex spasm.
- A nasogastric tube is passed for feeding purposes and to administer the drugs Tracheostomy, if breathing difficulty arises 0.

Dangerously ill cases :

- This group includes patients with major cyanotic convulsions.
- In addition to continuing sedatives, these patients are paralysed with muscles relaxants and positive pressure ventilation is given till they recover.
- One cannot predict exactly how many days a patient requires ventilatory support during this period adequate nutrition, care of the urinary bladder, care of the bowel, frequent change of position to avoid bed sore, have to be taken care.

Cause of death in tetanus :

- Aspiration resulting aspiration pneumonia.
- Laryngeal spasm and respiratory muscles spasm results in respiratory arrest and death.
- Autonomic disturbances resulting in cardiac arrythmias
- Prolonged ventilations bed sores and septicemia and death.