

3

Tetanus and Gas Gangrene

- Tetanus
- Gas gangrene
- Types of gas infections
- What is new? / Recent advances

TETANUS

Introduction

A nonimmunised, eighteen-year-old girl was admitted with moderate tetanus following a nail prick in her foot. In the hospital she developed convulsions, laryngeal oedema and cardiac arrest from which she was resuscitated and shifted to intensive care unit under **anaesthesiologist's care**. Tracheostomy, ventilation and paralysing agents were used. Unlike many others, she was lucky. She walked home after two months of stay in intensive care unit after a lot of suffering and spending a large amount of money. The case history has been written here to impress upon the students, the following:

1. How important is immunisation to prevent tetanus?
2. How serious is this disorder?
3. Is it possible to save these patients who are critically ill?

Aetiopathogenesis

- Tetanus is a serious disorder with very high morbidity and mortality even with treatment. The disease is caused by *Clostridium tetani*, an anaerobic spore-forming bacillus with terminal spore which has a drumstick-like appearance.

PEARLS OF WISDOM

Narcotic addicts who inject themselves beneath the skin at many sites are vulnerable—‘Skin Poppers’.

Possible routes of infection

- **Umbilical cord, in neonates**, seen in communities which practise cowdung application on the umbilical stump.
- Wound, as a complication of road traffic accidents where other aerobic organisms reduce oxygen tension in the wound, thereby facilitating growth of anaerobic *Clostridium tetani* (Key Box 3.1).
- **Minor injuries with rusted nails**, piercing of the ear lobe, tattooing, injections, etc.
- Endogenous infection after **septic abortion or surgical operations** on gastrointestinal tract.
- Tetanus due to infection acquired in the operation theatre. Thus, tetanus is a wound infection. “**No wound, no tetanus**” is true. Having entered the wound, the organism

KEY BOX 3.1

WOUNDS WHICH ARE MORE PRONE FOR TETANUS

- T**ime—wound more than 6 hours old
- E**xcessive contamination by soil, faeces, rust
- T**issue devitalised or denervated
- A**nimal or human bites
- N**o less than 1 cm (more than 1 cm)
- U**lcer or wound—deeper
- S**tellate wounds—burst type
- R**emember as ‘**TETANUS**’



multiply and produce powerful exotoxins which produce the disease. Thus, the organisms by themselves, do not produce the disease. The toxins produced by the organisms are tetanospasmin (neurotoxin) and tetanolysin (haemolysin).

- **Tetanospasmin has affinity towards nervous tissues.** It reaches the central nervous system along the axons of motor nerve trunks. The toxin gets fixed to motor cells of the anterior horn cells. The toxin, which is fixed to the motor end plate, acts in the following 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 acts at the **spinal level** and causes reflex contraction of muscles due to minor stimuli.
- **The toxin which is fixed to the nervous tissue cannot be neutralised.** However, the circulating toxin can be neutralised. Incubation period may vary from a few days to months or years. Hence, it is not important. The interval between first symptom (dysphagia and stiffness of jaw) to a reflex spasm is called the **period of onset**. If this is less than 48 hours, the prognosis is poor and if more than 48 hours, prognosis is better.

Favourable conditions for development of tetanus

- No immunisation
- Foreign body
- Injury
- Improper sterilisation
- Devitalised tissues
- Anaerobic conditions.

Special types of tetanus

1. **Tetanus neonatorum:** It occurs due to contamination of umbilical cord in children born to nonimmunised mothers. It manifests usually around 6–8 days of birth and is called as **eighth day disease**. It carries almost 100% mortality.
2. **Local tetanus:** In this, contraction of muscles occurs in the neighbourhood of the wound.
3. **Cephalic tetanus:** Usually occurs after wound of head and face. Cranial nerves like facial nerve and oculomotor nerve can get **paralysed**. It carries poor prognosis.
4. **Bulbar tetanus:** It is a condition wherein muscles of deglutition and respiration are involved. It is fatal.

5. Latent tetanus: It develops after a few months to years following a wound which might have been forgotten.

6. Puerperal tetanus: It occurs as a complication of abortion or puerperal sepsis.

7. Postoperative tetanus: Occurs due to improper sterilisation of instruments and carries 100% mortality. In a modern operation theatre this type of tetanus should not occur.

8. Otitis tetanus: It is due to chronic suppurative otitis media. In these cases, the wound is a tear in the tympanic membrane. It can occur in any age group, but commonly occurs in children and young adults.

Clinical features (Table 3.1)

- **Autonomic dysfunction:** Increased basal sympathetic tone manifesting as tachycardia and bladder, bowel dysfunction, labile hypertension, pyrexia, pallor, sweating and cyanosis of the digits can occur.
- Episodes of bradycardia, low central venous pressure and even cardiac arrests have been reported due to parasympathetic dysfunction.
- They can develop complications such as pneumonia, urinary tract infection, etc.

Treatment of established tetanus

- I. General management
- II. Specific management.

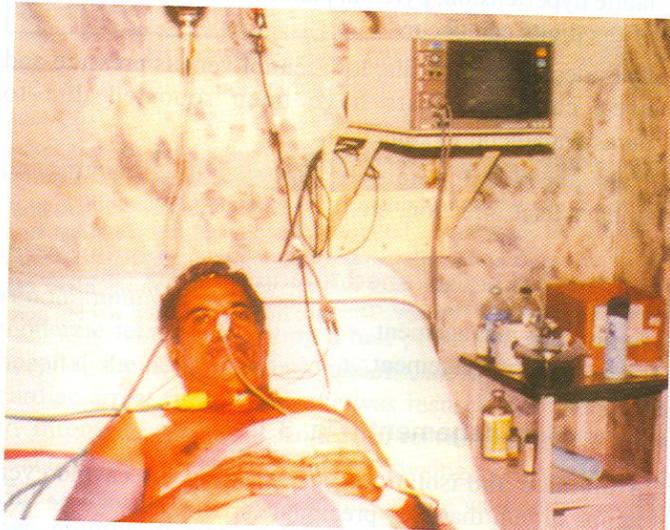
I. General management (Fig. 3.1)

- **Admission and isolation**¹ in a quiet room, to avoid even minor stimuli that may precipitate spasm.
- **Wound care** which includes drainage of pus, excision of necrotic tissue, removal of foreign body and proper dressing. Exudate or pus can demonstrate gram-positive rods.
- **Inj. tetanus toxoid** 0.5 ml to be given IM.
- **Antitetanus serum** (ATS) 50,000 units intramuscular (IM) and 50,000 units intravenous (IV). This should be given only after giving a test dose which consists of diluting a small dose of serum with ten times saline and inject a small amount in the subcutaneous tissue. It has become less popular due to availability of human antitetanus globulin.

¹Isolation for tetanus has been misunderstood by surgeons. It is a fact that in majority of the hospitals, tetanus patients are isolated in a remote corner of the hospital, well away from the reach of a skilled person. Many cases die due to convulsions and laryngeal spasm before they are intubated and resuscitated. The critically ill patients are admitted in an intensive care unit under the supervision of anaesthesiologist's care in our institution (Fig. 3.1). Tetanus is not communicable from person to person.

Table 3.1 Clinical features with differential diagnosis

Symptoms and signs	Differential diagnosis
<ul style="list-style-type: none"> Trismus or lock jaw, occurs due to severe contraction of the masseter muscle, resulting in inability to open the mouth. It is the most common symptom of tetanus. Dysphagia occurs due to spasm of pharyngeal muscles. Neck rigidity Rigidity of back muscles Risus sardonicus due to spasm of facial and jaw muscles. Generalised convulsions wherein every muscle is thrown into contraction, with severe clenching of teeth, arched back and extended limbs is described as opisthotonus (bow-like body; hence the name dhanurvatha). Mild temperature and tachycardia 	<ul style="list-style-type: none"> Alveolar abscess or temporomandibular joint involvement Tonsillitis Meningitis Orthopaedic disorder Anxiety neurosis Epilepsy Sympathetic hyperactivity

**Fig. 3.1:** Tetanus patient recovering in an intensive care unit

- Instead of ATS, human antitetanus globulin** is better and safe. It does not cause anaphylaxis. It is given in the dose of 3000 to 4000 units IV. 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** 500 mg IV 8th hourly for 10 days. It has been shown to be more effective than penicillin.

- After recovery, full immunisation with tetanus toxoid is a must.

II. Specific management

A. Mild cases

- There is only tonic rigidity without spasm or dysphagia. These patients are managed by heavy sedation using a combination of drugs so as to avoid spasm or convulsions. An example of the method of treatment followed in our hospital is given in Table 3.2.
- Benzodiazepines** and **morphine** act centrally to minimise the effects of tetanospasmin.
- Chlorpromazine** being an α -receptor blocker, can decrease sympathetic activity. Other α -blockers such as phenoxybenzamine, phentolamine have also been used.
- These drugs are repeated in such a way that the patient receives some sedative every two hours. The dosage of the drugs is adjusted once in 2 or 3 days so as to get the maximum effect of sedation or muscle relaxation.
- Injection diazepam 10 mg, tracheostomy set, resuscitation set which includes laryngoscope and endotracheal tube should be kept ready by the side of the patient.

B. Seriously ill cases

- They have dysphagia and reflex spasms.

Table 3.2 Method of treatment

Drug	Dosage	Time
Chlorpromazine	50–100 mg	8 am, 2 pm, 8 pm and 2 am
Phenobarbitone	30–60 mg	10 am, 4 pm, 10 pm and 4 am
Diazepam	10–20 mg	12 noon, 6 pm, 12 midnight and 6 am



- A nasogastric tube is introduced for feeding purposes and to administer the drugs.
- Tracheostomy, if breathing difficulty arises.

C. Dangerously ill cases

- This group includes patients with major cyanotic convulsions. In addition to continuing sedatives, these patients are paralysed with muscle relaxants (neuro-muscular blocking agents) and mechanically ventilated till they recover. One cannot predict the duration of the need for ventilatory support. During this period supportive therapy such as adequate nutrition, care of the urinary bladder and bowel, frequent change of position to avoid bedsores, have to be given.

Prophylaxis

1. *Tetanus neonatorum* can be prevented by immunisation of the mother with two tetanus toxoid injections, **half ml IM** given in the second trimester of pregnancy.
2. Infants and children are immunised with tetanus toxoid, diphtheria and pertussis vaccine (DPT) three doses at 6, 10, 14 weeks of age. This is called triple antigen. A booster dose is given at 18 months and school going time (5 years), and once in five years 0.5 ml of tetanus toxoid is given to achieve active immunity.
3. Immunised individual who receives a provocative injury is administered a booster dose if he has not been given in the previous 5 years.
4. Tetanus can be **prevented by giving tetanus antitoxin** in the following situations:
 - Wounds of head and face, penetrating wounds
 - Wounds with contused and devitalised tissues
 - War wound and road traffic accidents
 In such patients, a dose of 250 units of human antitetanus globulin will give adequate protection.

Causes of death

1. **Aspiration** of pharyngeal contents into the lungs resulting in aspiration pneumonia.

2. **Laryngeal spasm and respiratory arrest** resulting in cardiac arrest.
3. **Autonomic disturbances** resulting in cardiac arrhythmias
4. In some patients, **pacemaker insertion** may help if there is refractory bradycardia.

GAS GANGRENE

- It is a highly fatal, rapidly spreading infection caused by clostridial organisms which results in myonecrosis.

Other names for gas gangrene

- Clostridial myositis, clostridial myonecrosis, infective gangrene of the muscles.

Aetiology

- The disease is caused by *Clostridium perfringens* (*Clostridium welchii*)—the commonest organism (60%). Other organisms are *Clostridium septicum*, *Clostridium oedematiens*, *Clostridium histolyticum*.
- These are gram-positive, anaerobic spore-bearing bacilli.

Source of infection

- Manured soil or cultivated soil, normal intestines.

Risk group

In patients who have had lower limb amputations performed for ischaemic gangrene, infection can occur from patient's own bowel organisms. High velocity gun shot wounds with perforation of hollow viscera are also associated with risk of developing gas gangrene (military wound).

Pathogenesis (Table 3.3)

- Gas gangrene develops in wounds where there is heavy contamination with soil or foreign body, or which is associated with laceration and devitalised muscle mass. This type of situation is common today following road traffic accidents. Endogenous infection from patient's faecal matter may be responsible for gas gangrene, in certain cases of contamination of a surgical wound such as

Table 3.3 Predisposing factors for the development of gas gangrene

Factors	Mechanism
Foreign body such as soil, clothing, bullets, glass pieces.	Soil supplies calcium and silicic acid which causes tissue necrosis.
Anoxia due to crushing of the arteries.	Necrosis of the tissues results in proliferation of the organism.
Dead and devitalised tissues.	Anaerobic organisms multiply
Blood clots.	Supplies calcium
Extravasated haemoglobin and myoglobin	Cease to carry oxygen.

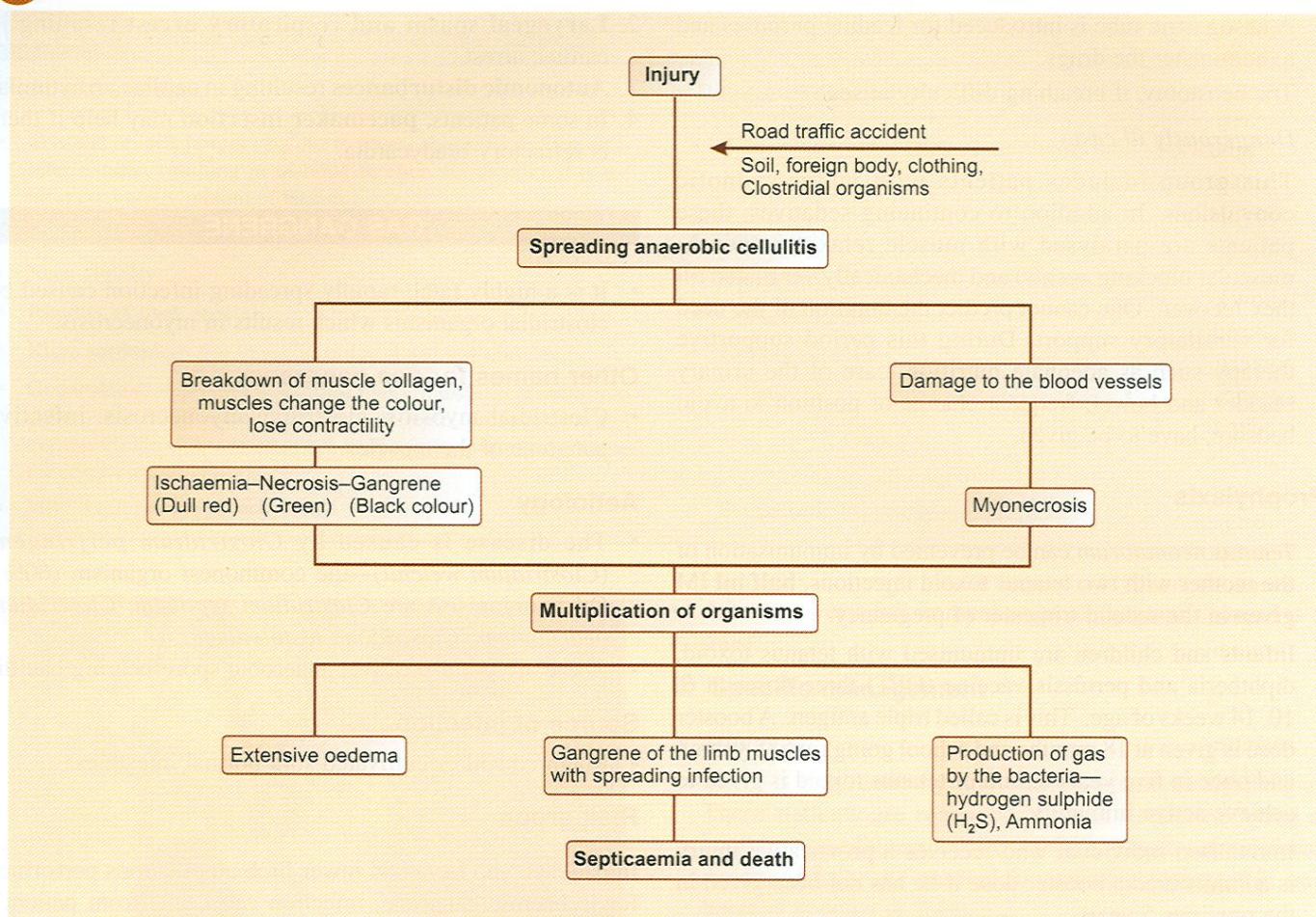


Fig. 3.2: Pathological changes

Table 3.4 Toxins and their effects

Toxins	Effects
1. Lecithinase (alphatoxin)	<ul style="list-style-type: none"> Dermonecrosis Haemolysis Profound toxæmia
2. Beta toxin	<ul style="list-style-type: none"> Necrosis of the tissues
3. Proteinase	<ul style="list-style-type: none"> Breakdown of collagen fibres
4. Hyaluronidase	<ul style="list-style-type: none"> Break the cement substance of the muscle cells—hyaluronic acid

Table 3.5 Clinical features

Local features	General features
Severe pain and gross oedema of the wound	Anxious and alert
Sutured wound is under tension	Toxic and ill
Thin brownish fluid escapes which has sickly sweet odour	Rapid increase in the pulse rate
Palpable crepitus	Hypotension due to suppression of adrenals
Colour changes in the muscles	Vomiting
Skin becomes khaki-coloured due to haemolysis	Low grade fever

**KEY BOX 3.2****PALPABLE CREPITUS—CONDITIONS**

- Anaerobic infections
- Streptococcal infections
- Surgical emphysema due to oesophageal, tracheal rupture
- Gas gangrene



below knee amputation done for some other cause. Having entered the wound, *clostridia* multiply and produce powerful toxins.

- All these factors contribute to create a low oxygen tension. Under these favourable conditions clostridial organisms multiply and produce toxins which cause further tissue damage. The toxins produced by the organisms and their effects are given in Table 3.4.
- Once powerful toxins start acting, various pathological events such as inflammation, oedema, muscle necrosis and gangrene of the muscles set in. These events are summarised below.

Clinical features (Table 3.5)

- In untreated cases, necrotic process continues, septicaemia, renal failure, peripheral circulatory failure and death occur. Foamy liver is the condition wherein gas is produced in the liver, as a part of septicaemia.

Prophylaxis

Being highly fatal, gas gangrene is better prevented by observing following principles while managing the wound:

- 1. Debridement:** All dead, necrotic tissue, bone pieces and foreign material are removed. Pus is evacuated. Wound is thoroughly irrigated with antiseptic agents.
- 2. Prophylactic antibiotics:** Penicillin is the drug of choice. Injection crystalline penicillin 10–20 lakh units, 4–6th hourly is given for a period of seven days.
- 3. Judicious and minimal use of tourniquet:** If possible, avoid tourniquet while managing such a wound in the leg.
- 4. Gentle but effective application of plaster cast** with or without treatment of associated fractures to avoid compression on the blood vessel.

PEARLS OF WISDOM

When in doubt, do not suture the wound.

Diagnosis

- In addition to clinical suspicion, one of the easy methods of confirming the diagnosis is to examine the pus under microscope after staining with Giemsa stain.
- These organisms are gram-positive and spore-bearing.

Treatment of established gas gangrene

- **Emergency surgery** which includes excision of all dead muscles and necrotic tissues by using generous, long incisions.
- **Penicillin** to be continued.
- **Blood transfusions** before, during and after surgery.
- **Polyvalent anti-gas gangrene serum.**
- **Hyperbaric oxygen** will reduce the amount of toxin produced by the organisms (controversial).
- **Do not hesitate to amputate** if it saves the life, because this is the only measure in late cases.

Summary (Key Box 3.3)**KEY BOX 3.3****SUMMARY OF GAS GANGRENE**

- Correct hypotension
- Control infection
- Treat dehydration
- Conduct operation
- Administer hyperbaric oxygen
- Give blood transfusion
- Passive immunisation
- To save life, amputate.

**CLINICAL NOTES**

A forty-year-old gentleman (Fig. 3.3) presented to the hospital with massive gas gangrene involving upper limb, chest wall, abdominal wall and back. It started after an injury to the elbow. The patient received initial treatment in a local hospital. Due to lack of proper facilities, he was neither subjected to any surgical procedure nor was he given any resuscitation. When he came to the hospital, he was in septic shock.

It was too late when we saw the patient. He was having severe hypotension and renal failure. Resuscitation and debridement was done. However, within six hours of admission to the hospital, he expired.

KEY BOX 3.4**INFECTIONS BY CLOSTRIDIUM WELCHII**

- Gas gangrene of the limb
- Gas gangrene of the abdominal wall
- Gangrenous appendicitis, cholangitis
- Necrotising enteritis, food poisoning
- Infection of the uterus following septic abortion.





30

Manipal Manual of Surgery



Fig. 3.3: Gas gangrene

EVIDENCE BASED APPROACHES

TYPES OF GAS INFECTIONS

- Clostridial cellulitis:** In this condition, healthy muscle is not involved. It involves necrotic tissue and produces features of cellulitis such as tense, swollen parts with palpable crepitus. However, it is a mild infection, which can be managed by antibiotics without surgery (Key Box 3.4).
- Local type:** It refers to infection confined to a single muscle.
- Group type:** It refers to infection confined to one group of muscles in the compartment. Such cases benefit from a compartmental excision.
- Massive type:** Gas gangrene involving the entire limb, needs to be treated by amputation.

CLINICAL NOTES



A 48-year-old lady underwent vaginal hysterectomy 10 days back and was brought to our hospital, as a case of 'tetanus'. On examination, she had neck rigidity and difficulty in opening mouth. Abdominal rigidity was mild. She was looking pale. Pallor was attributed to anaemia caused by 'dysfunctional uterine bleeding' (DUB), for which she was operated. Diagnosis of postoperative tetanus was made and treatment started. Next day, she was not responding to commands. Laboratory reports which were sent previous day, showed a total WBC count of 44,000 cells/mm³ clinching the diagnosis. It was a case of 'leukaemia'. Neck rigidity was due to leukaemic infiltrates in meninges. Now you know the cause of uterine bleeding!!!

WHAT IS NEW IN THIS CHAPTER? / RECENT ADVANCES



Magnesium titrated to clinical endpoints has been tried with success as a first line therapy in the routine management of tetanus.

Various studies concluded that magnesium therapy protocol recommended for tetanus differs from that of eclampsia in the following aspects:

The rate of infusion of magnesium

The rate of infusion of magnesium (after the loading dose of 5 g bolus over 20 minutes) should be titrated not only to the control of spasms but also to muscle rigidity. Rigidity should be reduced to a level acceptable to the patient permitting swallowing of saliva, mouth care and limb physiotherapy. **The hourly dose required may be as high as 4–5 g/h, which is far greater than that used in eclampsia.** Total abolition of rigidity is not required and may lead to muscle hypotonia.

The patellar reflex cannot be used as a valid indicator of serum magnesium concentration in all patients, as the reflex is sometimes masked by rigidity and tends to be lost early in elderly patients. The patients should, therefore, be monitored for depression of ventilation.