Infectious diseases of equine
Bacterial and mycotic diseases
Rhodococcus equi infection
Pneumonia in foals

It is an infectious disease of foals characterized by pneumonia, abscessation of lungs, suppurative lymphadenitis, septic arthritis, osteomyelitis and diarrhea

Etiology

The disease is caused by *Rhodococcus (Coryne bacterium) equi*.

Epizootiology

Geographical distribution

It is a disease of worldwide distribution.

Susceptibility

The disease affects foals, adult horses and pigs. The age of onset of clinical signs varies between two weeks and six months.

Sources of infection

Infected animals and soil are the sources of infection.

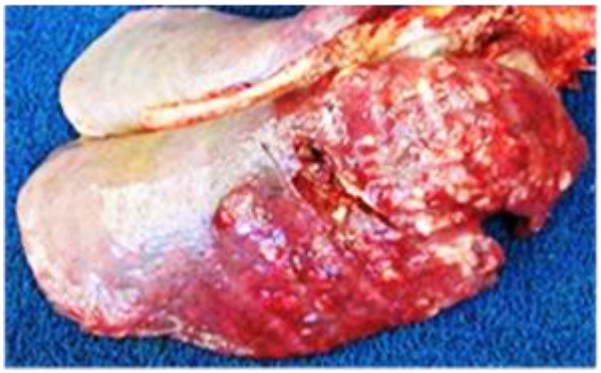
Transmission

Infection occurs by inhalation of the organism in dust.

Pathogenesis

Foals with immunodeficiency are particularly susceptible to infection. Virulent strains are facultative intracellular parasites of macrophages and neutrophils. The combined action of humoral and cellular immune systems is important in preventing development of the disease after inhalation of bacteria. The inability of the pulmonary macrophages to destroy the organism leads to persistent infection in the lung and a chronic bronchopneumonia develop with extensive abscessation of the lungs and suppurative lymphadenitis.

Intestinal infection is common in foals with *Rhodococcus equi* pneumonia.



Clinical signs

1. Fever, depression, inappetance and tachypnea.
2. Severe respiratory distress and cyanosis may be present.
3. Auscultation of the chest reveals crackles and wheezes.
4. Foals with *R. equi* abscess may not have abnormal lung sounds.
5. Subcutaneous abscesses, osteomylitis and septic arthritis may be present
6. Severe diarrhea may follow or accompany the respiratory signs.
7. Severely affected animals die within 1-2 weeks.

Post mortem lesions

1. Pyogranulomatous pneumonia
2. Lymphadenitis of the bronchial lymph nodes.
3. Ulcerative enterocolitis and abscessation of the mesenteric lymph nodes.

Treatment

1. Erythromycin-Rifampin combination is effective.
   Erythromycin: 25 mg /kg body weight, orally every 6 hours.
   Rifampin: 10 mg/ kg body weight, every 24 hours

   Therapy should be continued until the foal is clinically normal.
   - Diarrhea may develop in treated foals and treatment should be temporarily discontinued.
   - During hot weather some foals become hyperthermic and should be treated with antipyretics
2. Non-steroid anti-inflammatory drugs.
3. Bronchodialators and mucolytics.

Control

1. Insuring adequate transfer of colostral immunoglobulins.
2. Reducing the fecal contamination of pastures and dusty or sandy areas.
3. Regular physical examination of foals.
4. Administration of a hyper immune serum obtained from mares vaccinated with autogenous vaccine.
5. The use of autogenous vaccine.

Actinobacillosis of foals, Sleepy foal disease
(Shigellosis)

It is an acute fatal septicemic disease of newborn foals.

Etiology

The disease is caused by *Actinobacillus equuli*; a Gram-negative

coccobacillus in the genus *Actinobacillus*.

Epizootiology

Geographical distribution

The disease is of worldwide distribution

Susceptibility

Infections are seen in both foals and adult horses. Newborn foals are highly susceptible to septicemia, which may also occur in breeding mares

Sources of infection

Fetal fluid is the main source of infection. The organism can be isolated from cervix, pharynx and feces of mares with affected foals.

Transmission

Infection occurs through:

1. In utero infection
2. Ingestion of fetal fluids.
3. inhalation
4. Via the umbilical cord at parturition

Pathogenesis

The organism causes an acute septicemia which in many cases causes death before the development of specific lesions. Foals, which survive for more than 24 hours, develop suppurative lesions in the renal cortex, joints and intestines.

Clinical signs

1. Fever, prostration and diarrhea may be followed by meningitis, pneumonia, purulent nephritis, or septic polyarthritis (sleepy foal disease or joint-ill).
2. Foals appear sleepy or comatose.
3. Death within 24 hours is usual.
4. Foals that survive the acute phase develop arthritis, swollen joints and lameness and may die during the 2^{nd} and 7^{th} day. Mortality rate approaches 100%

Post mortem lesions

1. Enteritis and septicemic changes.

2. Foals dying after a longer interval show tenosynovitis and arthritis
3. Characteristic pinpoint abscesses in the renal cortices and adrenals.

Treatment: Infected horses may be treated with chloramphenicol, gentamicin, or third-generation cephalosporins. High doses of gentamycin and fluid therapy are indicated.

Control

1. Application of hygienic measures during foaling.
2. Cleaning and disinfection of foaling boxes.
3. Proper disinfection of the umbilicus.
4. Adequate intake of good quality colostrum.
5. Prophylactic use of antibiotics in enzootic breeding premises.

Strangles
(Equine distemper)

It is a contagious acute purulent infection of the upper respiratory tract and draining lymph nodes of horses.

Etiology

The disease is caused by *Streptococcus equi subspecies equi*; a Gram-positive coccobacillus occurs in chains.

Epizootiology

Geographical distribution

The disease is of worldwide distribution and enzootic in Egypt. Outbreaks are reported in breeding farms when the infection is introduced by new arrivals

Susceptibility

Horses, donkeys and mules are susceptible. The disease can affect horses of any age. Morbidity rate is usually greater in younger horses.

Sources of infection

These include:

1. Nasal and abscess discharges from infected animals which contaminate pastures, feed and water troughs.
2. Carriers may shed the organisms for over three years.

- The organism survives in the environment for at least two months.
- Recovered horse may A source of infection for at least 6 weeks after its clinical signs of strangles have resolved.

Transmission

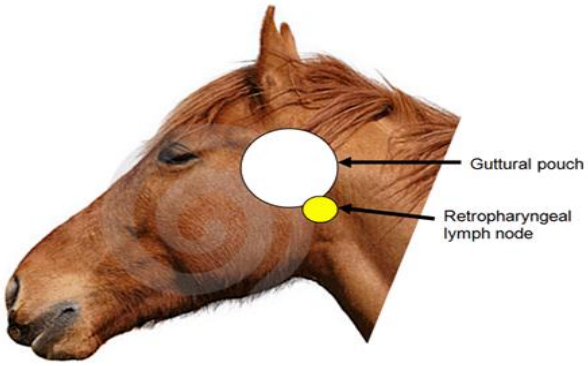
1. Infection occurs by direct transmission from infected animal to susceptible one through direct contact.

2. Indirect transmission occurs through contaminated water sources, feed or feeding utensils and twitches. Ingestion and inhalation can transmit the infection

Pathogenesis

Following infection, the bacteria lodge in the pharyngeal and tonsillar lymphoid tissues where they multiply. Migration of neutrophils into the lymph nodes causes swelling and abscessation. Most abscesses rupture externally and the infection resolves. Metastatic infection of the heart valves, lungs, liver, brain, eye, joints and kidneys causes chronic illness and death. Metastasis may occur due to bacteremia or extension of infection along chains of lymph nodes. Death may occur due to aspiration of infected material.

Incubation period is 1-3 weeks



Clinical signs

1. Fever, anorexia and depression.
2. Serous nasal discharge, which becomes copious and purulent.
3. Severe pharyngitis and laryngitis with soft moist cough.
4. The submandibular lymph nodes enlarge, become hot and painful.
5. Swelling of the retropharyngeal lymph nodes may cause dyspnea and dysphagia.
6. The affected lymph node ruptures discharging thick cream-yellow pus after about 10 days.

7. Other lymph nodes including the pharyngeal, submaxillary and parotid lymph nodes may be involved.
    - Course of the disease is about 3 weeks.
    - Morbidity rate approaches 100%
    - Mortality rate is 3-10 %

Atypical form "bastard strangles":

1. Suppuration may extend to various parts and organs of the body such as mesenteric and mediastinal lymph nodes, lungs, pleura, diaphragm, liver, kidneys and brain.
2. Infection may spread to local lymphatic vessels causing obstructive edema in lower limbs.
3. Guttural pouch, joints, bones, tendon sheaths, veins, and heart valves may also be involved.
4. Generalized systemic infection develops.

Complications

1. Immune mediated conditions:
    a. Post strangles myocarditis (Antibody-M. protein reaction).
    b. Post strangles anemia due to antigen – antibody complexes attach to the RBCs.
    c. Purpura hemorrhagica
2. Suppurative necrotic branchopneumonia due to aspiration of pus from ruptured abscesses in the upper airway.
3. Empyema of the guttural pouch due to rupture of the retropharyngeal lymph nodes.

Post mortem lesions

These include suppuration in internal organs especially the liver, spleen, lungs, pleura, and peritoneum.

Treatment

1. Antibiotic therapy; procaine penicillin G 22,000 I.U/ kg body weight, I.M every 12 hours or sodium penicillin G, 22,000 IU/ kg body weight, I.V every 6 hours for at least 5 days.
2. The use of non-steroidal anti-inflammatories to reduce swelling and provide pain relief.
3. Application of hot poultices to encourage rupture of abscesses.
4. Cleaning and disinfection of ruptured abscesses and surgical treatment.
5. Once an external lymphadenopathy is detected therapy should be directed toward enhancing maturation and drainage of the abscesses.

- During an outbreak, immediate antibiotic therapy in the early

acute phase may prevent focal abscessation. However, the horses will be highly susceptible to reinfection.

Control

During outbreaks, movement of any horses on or off the farm should be stopped, and new horses should not be introduced. Monitoring the rectal temperature and isolating horses at the first sign of fever is one of the most effective ways to stop the spread of infection.

1. Isolation of infected animals.
2. Cleaning and disinfection of fomites including pails, grooming brushes and blankets using phenolic compounds.
3. Disinfection of water troughs.
4. Burning of bedding.
5. Emergency prophylactic treatment.
6. Newly introduced animals should be quarantined for three weeks.
7. Vaccination (bacterin or M.protein extract)
    - M. protein vaccine, I.M, three injections at two weeks intervals with further administration every 6 months for animals at risk.
    - Vaccination of dams 4-6 weeks before parturition and foals at 2-3 months age.
    - The use of intranasal vaccine (Avirulent live strain) is also useful.

Equine purpura hemorrhagica

It is a noncontagious, type III immune-mediated acute fatal allergic condition characterized by sudden onset of cutaneous vasculitis edema and petechiation of mucus membranes.

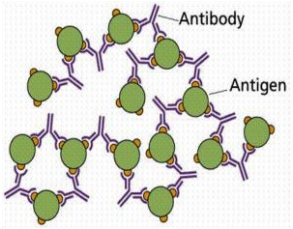
Etiology

The disease is caused by an immune reaction to *streptococcal protein* (immune mediated disease due to type III hypersensitivity).

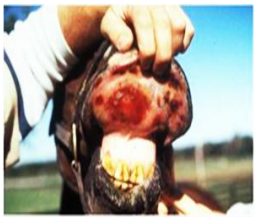
- Vasculitis occurs due to deposition of complexes of antigen and immunoglobulins in the wall of capillaries and small blood vessels.
- The presence of complexes of IgA and streptococcal M protein in sera is an evidence of immune reaction to streptococcal protein.

Pathogenesis

Vasculitis of capillary walls is accompanied by extravasations of plasma and blood into tissues.







Clinical signs

1. Subcutaneous edematous swellings occur on the face and muzzle. They are cold and painless and pit on pressure.
2. Swelling in the head may cause pressure on the pharynx with subsequent dyspnea and dysphagia.
3. Edema of the limbs usually occurs.
4. Submucous hemorrhages occur in the nasal cavities, mouth, and petechiation of the visible mucous membranes.
5. Edema of the gut wall may cause colic.

The course of the disease is usually 1-2 weeks and many animals die from blood loss and asphyxia.

Post mortem lesions

1. Echymotic and petechial hemorrhages are present throughout the body.
2. Subcutaneous swellings.
3. Lungs are edematous and congested.

Treatment

1. Corticosteroides such as dexamethasone 0.05-0.2 mg/ kg body weight, I/v or I/M every 24 hours.
2. Non-steroidal anti-inflammatory drugs such as phenylbutazone, 2.2 mg/kg body weight, orally or I/V every 12 hours.
3. Affected horses should be treated with Potassium or sodium penicillin at an initial dosage of 22,000 to 44,000 IU/kg IV. Intramuscular injections should be avoided because inflamed muscle tissue may be further aggravated.
4. Nasogastric feeding tube may be required.
5. Tracheotomy may be necessary in some cases.

Glanders (Farcy)

It is a contagious highly fatal disease of equine characterized by development of nodules and ulcers that occur most commonly in the upper respiratory tract, lungs and skin.

Etiology

The disease is caused by *Burkholderia mallei,* previously known as

Pseudomonas mallei, Malleomyces mallei, Actionbacillus mallei; a Gram-negative rod shaped organism.

Epizootiology

Geographical distribution

The disease occurs in some areas of Eastern Europe, Asia and North Africa. It has been eradicated from most countries and now it has a sporadic occurrence even in infected areas.

Susceptibility

1. Horses, mules and donkeys are susceptible.
2. Carnivores may be infected by eating infected meat.
3. The disease has been reported in sheep, goats and camels.
4. The disease is transmissible to man.

Sources of infection

Diseased animals, carriers, common watering troughs contaminated by nasal discharges, harness and grooming tools contaminated by exudates from skin ulcers are the main sources of infection.

Transmission

Infection occurs through:

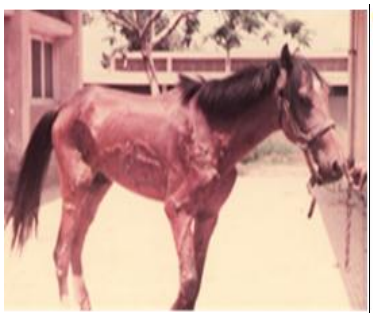
1. Ingestion of contaminated food and water.
2. Inhalation.
3. Contamination of skin abrasions by direct contact or from harness or grooming tools.

- The disease is more likely when animals are in a stressed state.

Pathogenesis

Infection occurs mostly by ingestion. Invasion occurs mostly through the intestinal wall where they enter the lymph vessels and pass through the mesenteric lymphatic glands and finally reach the blood stream. As a result, septicemia (acute form) or bacteremia (chronic form) is set up. Localization occurs in the lungs, nasal mucosa and skin where the characteristic nodules develop associated with lymphangitis and lymphadenitis. Bronchopneumonia usually develops and death occurs due to anoxic anoxia.

Incubation period is two weeks to three months


Clinical signs

Horses tend to develop the chronic from while mules and donkeys the acute form.

Acute disease

This form is manifested by:

a. High fever, cough and nasal discharge.
b. Rapid spread of ulcers on the nasal mucosa.
c. Rapid development of nodules on the skin of lower limbs or abdomen.
d. Death occurs in few days due to septicemia.

Chronic disease

There are three common forms:

a. Pulmonary form.
b. Nasal form.
c. Skin form.

a. Pulmonary form (chronic bronchopneumonia)

This form is characterized by:

1. Cough and labored respiration
2. Frequent epistaxis.
3. Rapid fatigue and exhaustion.

- Later on characteristic lesions appear on the nasal mucous membranes and skin.

b. Nasal form

1. In the early stages there is a serous nasal discharge which may be unilateral. Later, it becomes purulent and blood stained.
2. The submaxillary lymph nodes become enlarged.
3. Lesions appear on the lower parts of turbinates and the cartilaginous nasal septum. Lesions appear as nodules (1cm in diameter) which ulcerate producing ulcers with irregular raised edges. On healing, ulcers are replaced by a characteristic stellate scar.

c. Skin form, cutaneous glanders or farcy.

1. The predilection site for cutaneous lesions is the medial aspect of the hock, but they can occur on any part of the body.
2. Lymph vessels become thick and fibrosed.
3. Subcutaneous nodules 1-2 cm in diameter develop along the course of lymph vessels.
4. Nodules soon ulcerate forming ulcers with raised irregular borders (crater like) which discharge pus of the color and consistency of dark honey. Ulcers have no tendency to heal
5. Lymph nodes draining the area become involved and may discharge to the exterior.

Postmortem lesions

1. Nodules, ulcers or scars on nasal mucosa.
2. Lymphangitis and purulent lymphadenitis.
3. Nodules in the lungs.
4. Nodules and ulcers on the skin.

Control

1. Application of strict quarantine of affected premises.
2. Restriction of horse's movement in affected areas.
3. Clinical cases should be destroyed.
4. Proper disposal of contaminated material and carcasses
5. Proper disinfection of premises and food and water troughs.
6. Contact animals should be subjected to Mallein test at intervals of three months until all reactors are removed.

Epizootic Lymphangitis

Pseudoglanders, Histoplasmosis

It is a chronic contagious disease of equine characterized by purulent inflammation of the superficial lymphatic vessels and lymph nodes.

Etiology

The disease is caused by *Histoplasma farciminosum* (*Zymonema, Cryptococcus, Blastomyces farciminosum)*

Epizootiology

Geographical distribution

The disease occurs in Asia, Africa and Mediterranean area.

Susceptibility

Horses and mules are highly susceptible and donkeys are more resistant.

Sources of infection

These include, infected animals discharging the organisms in pus, infected bedding, soil, grooming utensils and horse blankets. The organism has been isolated from the alimentary tract of flies.

Transmission

Fungal spores are carried form infected animals or from bedding, grooming utensils and gain entry through abrasions especially on the lower limbs

Pathogenesis

The fungus gains entry through wounds, then invades subcutaneous tissues producing a local granuloma and ulcers at the portal of entry. The organism spreads along the lymph vessels and causes lymphangitis. Nodules develop along the course of lymph vessels, and then rupture discharging thick creamy pus.

Incubation period takes few weeks

Clinical signs

1. An ulcer develops at the portal of entry. The lesions usually develop on limbs particularly about the hocks but may also be present on the back, sides, neck, vulva, and scrotum.
2. Lymphatics become corded and nodules develop along its course.
3. Nodules rupture, discharging thick creamy pus.
4. Local lymph nodes enlarge and may rupture.
5. Thickening of the skin in the affected area and general swelling of the whole limb are common.
6. Occasionally lesions appear on the nasal mucosa but do not involve the nasal septum. These lesions appear due to licking of the lesions of the limbs.
7. Ocular involvement is manifested by keratitis and conjunctivitis.
8. Primary pneumonia may develop.
    - Mortality rate is 10- 15%
    - The course of the disease lasts for 3-12 months.
    - Spontaneous recovery may occur with development of solid immunity.
    - Many animals are destroyed due to the chronic nature of the disease and inability to work.

Postmortem lesions

1. Lesions are present on the skin, subcutaneous tissues, lymph vessels and lymph nodes.
2. Granulomatous lesions may present in lungs, liver, and spleen.

Treatment

In enzootic areas, the less severe cases may be treated.

1. Surgical excision of lesions.
2. The use of parenteral iodides.
3. Antifungal drugs as amphotericin B.

- In most cases it is better to destroy the diseased animal due to the chronic nature of the disease and exhaustion.

Control

Free areas

Outbreaks in free areas are best controlled by destruction of affected animals.

Enzootic areas

1. Severe cases should be destroyed.
2. Application of strict quarantine and treatment of less severe cases.
3. Proper disinfection of stables.
4. Destruction of infected bedding, harness and utensils.
5. Prophylactic treatment of wounds and abrasions.

6. The use of vaccines.

Sporotrichosis

It is a contagious disease of horses, cattle, cats and humans characterized by the development of cutaneous nodules and ulcers on the limbs. It may be accompanied by lymphangitis.

Etiology

Sporotrichum schenckii (sporotrichum equi) is a dimorphic fungus.

Epizootiology

Geographical distribution

The disease occurs in Europe, India, and USA and reported in Egypt.

Susceptibility

Horses, donkeys, mules, cattle and cats are susceptible.

Sources of infection

Discharges (pus) of infected animals and soil are the main sources of infection.

Transmission

Infection occurs by contamination of cutaneous wounds by contact with discharges from infected animals or from contaminated surroundings**.**

Pathogenesis

The fungus gains entry through wounds in the skin and spreads via lymphatics. Nodules are formed which ulcerate discharging pus.

Clinical signs and lesions

1. Development of multiple small cutaneous nodules on the lower parts of the legs usually about the fetlock.
2. The nodules ulcerate discharging a small amount of pus.
3. Lymphangitis may occur.

Treatment

1. Systemic treatment with iodides.
2. Local application of tincture iodine.
3. The use of amphotericin B and griseofulvin.

Control

1. Prophylactic treatment of wounds and abrasions
2. Isolation and treatment of infected cases
3. Proper disinfection of bedding and harness

Ulcerative lymphangitis

It is a contagious disease of horses and cattle characterized by lymphangitis of the lower limbs.

Etiology

The disease is caused by sero type II (equine-bovine strain); a nitrate positive strain.

C. pseudotuberculosis is the causative agent for human lymphadenitis, ulcerative lymphangitis in cattle, equine lymphangitis in horses, and caseous lymphadenitis in sheep and goats.

Epizootiology

Geographical distribution

It is a disease of worldwide distribution.

Susceptibility

Horses and cattle are susceptible.

Sources of infection

Infected animals and soil are the main sources of infection.

Transmission

Infection occurs through abrasions on the lower limbs either directly or indirectly. Flies may transmit the infection.

Pathogenesis

Infection occurs through cutaneous wounds followed by lymphangitis and the development of abscesses along their course.

Clinical signs and lesions

The common seat of the disease is the fetlock area.

1. The affected part becomes swollen and painful.
2. Nodules develop in the subcutaneous tissue particularly around the fetlock but can spread to all parts of the body.
3. Nodules enlarge to five cm in diameter and rupture discharging creamy green pus resulting in formation of ulcers with ragged edges and a necrotic base.
4. Lymphangitis.
5. Lesions heal in 1-2 weeks but new lesions may develop and cause persistence of the disease for up to 12 months.

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Treatment

1. Local treatment of ulcers.
2. Parentral injection of penicillin or oxytetracycline.

Control

1. Prophylactic treatment of wounds and abrasions
2. Treatment of affected animals.
3. Proper disinfection of stables
4. The use of autogenous bacterin.

Tetanus

It is an acute highly fatal infectious disease of all warm blooded

animals and man. It is characterized by hyperesthesia, tetany, convulsions and death.

Etiology

The disease is caused by toxins of *Cl. tetani*. The organism is a Gram- positive rod forms spores, which can persist in soil for many years.

Epizootiology

Geographical distribution

It is a disease of worldwide distribution

Susceptibility

Horses, mules and donkeys are most susceptible. Sheep, goats, cattle, pigs, dogs, and cats are also affected.

Sources of infection

Soil contaminated by feces of animals especially horses and contaminated surgical instruments are the main sources of infection.

Transmission

Infection occurs through:

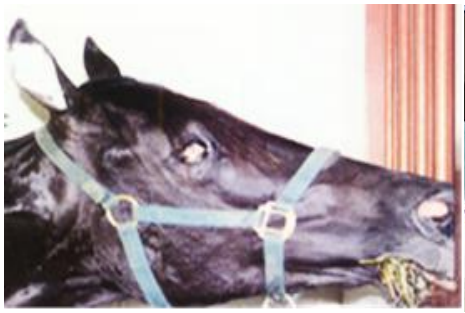
1. Deep puncture wounds of hooves are common in equines.
2. Genital tract injuries at the time of parturition in cattle
3. Castration, sheering and docking in sheep.
4. Teeth eruption, umbilical infection, and dehorning.
5. Injuries in mouth due to fibrous feed or in the gastrointestinal tract may cause what is called idiopathic tetanus.

Pathogenesis

Tetanus spores remain localized in the wound until lowering of the local tissue oxygen tension occurs. The organisms proliferate and produce tetanolysin and tetanospasmin. The tetanolysin promotes local tissue necrosis. The tetanospasmin diffuses to the systemic circulation, bounds to motor end-plates and travels up peripheral nerve trunks and inhibits inhibitory neurotransmitters resulting in hypertonia and spasms. (It blocks the release of inhibitory neurotransmitters) .The toxin causes spasmodic tonic contractions of the voluntary muscles by nerve irritation. Central potentiation of normal sensory stimuli occurs and innocuous stimuli cause exaggerated responses. Death occurs by asphyxiation due to fixation of the muscles of respiration.

Incubation period is 3-10 days and up to 3 weeks, this depends on:

1. Number of spores.
2. Age of the animal.
3. Nature and site of the wound.



Clinical signs

1. Increase in muscle stiffness accompanied by muscle tremors.
2. Third eyelid prolapses.
3. Tetanic spasms of the jaw muscles, erection of ears, dilatation of the nostrils and erection of the tail.
4. Mastication is prevented by tetany of the masseter muscles and saliva may drool from the mouth.
5. Constipation and urine retention.
6. Increased response to external stimuli (hyperesthesia).
7. In later stages, temperature may rise when muscular tone and activity are increased. Temperature may also increase due drenching pneumonia or paralysis of heat regulating centers.
8. Recumbence, prostration, and death.
    - In cattle bloat is an early sign.
    - The duration of the fatal illness in horses and cattle is usually 5-10 days.
    - In mild cases recovery occurs slowly over a period of weeks or months.
    - Signs of bad prognosis are recumbence, prostration, rise in body temperature and lock jaw.
    - Sheep usually die on about the third or fourth day due to tympany.

Treatment

1. Elimination of the organism
    a. The wound should be cleaned and irrigated with hydrogen peroxide with local application of penicillin, this should be applied only after administration of the antitoxins

b. Parentral injection of penicillin in large doses

2. Neutralization of the toxins: Antitoxin should be administered early. High doses are required 90.000 – 300.000 I.U. for three injections every 12 hours.
3. Relaxation of muscle tetany using tranquilizers like chlorpromazine one mg/ kg body weight, I.M., or 0.4-.08 mg / kg body weight, I.V. twice daily.
4. Nursing.
   a. The use of stomach tube or intravenous feeding.
   b. Slinging of the animal.
   c. The use of enema and catheterization.
   d. Animal should be kept in quiet dark place with well bedded quarters.

Control

1. Proper disinfection of instruments at castration, docking and shearing.
2. Proper disinfection of the wound.
3. The prophylactic use of antitoxins in cases of penetrating wounds or deep lacerations and surgical interference. (1500 I.U).
4. Active immunization using toxoid (alum precipitated purified toxoid). Fools should be vaccinated at 4 months of age (2 doses, 3–4 weeks apart). Horses are revaccinated yearly with a single booster injection. Pregnant mare should receive a booster injection, 4-6 weeks before foaling.