BLACKLEG

ETIOLOGY

the disease occur caused by *Clostridium chauvoei*, a Gram-positive, spore- forming, rod-shaped bacterium. The spores are highly resistant to environmental changes and disinfectants and persist in soil for many years.

**Epidemiology**

 Cattle 6 months to2 years of age that are rapidly growing and on a high plane of nutrition. Seasonal occurrence in warm wet months. There are often multiple cases in at-risk animals. Sheep of all ages - occurs as outbreaks predisposed by wounds from shearing, docking, castration, dystocia

**Source of infection**

Blackleg is a soil-borne infection but the portal by which the organism enters the body is still in dispute. It is presumed that the portal of entry is through the alimentary mucosa after contaminated feed or associated with ingestion of erupting teeth. The bacteria may be found in the spleen, liver, and alimentary tract of normal animals, and contamination of the soil and pasture may occur from infected feces or decomposition of carcasses of animals dying of the disease.

PATHOGENESIS

In true blackleg the stimulus that results in growth of the latent bacterial spores is unknown. There is usually no history of trauma, although trauma while passing through an alley is reported as the likely inciting factor in one outbreak. Toxin formed by the organism produces a severe necrotizing myositis locally in skeletal muscles, and a systemic toxemia that is usually fatal. In cattle and sheep atypical outbreaks of sudden death occur in which the lethal lesion is a clostridial

cardiac myositis

**CLINICAL FINDINGS**

Cattle

If the animal is observed before death there is

1- severe lameness, usually with swelling of the upper part of the affected leg.

2-very depressed and have complete anorexia and ruminal stasis

3- high temperature (41°C ) and pulse rate (100-120/min) .

4-In the early stages the swelling is hot and painful to the touch but soon becomes cold and painless

5- edema and emphysema can be felt.

6-The skin is discolored and soon becomes dry and cracked.

7-Although the lesions are usually confined to the upper part of one limb, occasional lesions are present in other locations such as the base of the tongue, the heart muscle, the diaphragm muscles, and the udder.

8-Lesions are sometimes present in more than one of these locations in one animal.

9-The condition develops rapidly and the animal dies quietly 12-36 hours after the appearance of signs.

10- Many animals die without signs having been observed.

**Sheep**

**When blackleg lesions occur in the limb musculature in sheep,**

there is a stiff gait and the sheep is disinclined to move because of severe lameness in one limb or, more. Subcutaneous edema

is not common and gaseous crepitation cannot be felt before death.

Discoloration of the skin may be evident but skin necrosis and gangrene do not occur.

In all instances there is high fever, anorexia, and depression, and death occurs very quickly.

Lesions of the head may be accompanied by severe local swelling due to edema and there may be bleeding from the nose.

**Horses**

The clinical syndrome in horses is not well defined. Pectoral edema, stiff gait, and in coordination are recorded

**N ECROPSY FINDINGS**

Cattle found dead of blackleg are often in a characteristic position;

1-lying on the side with the affected hindlimb stuck out

stiffly.

2-Bloating and putrefaction occur quickly and bloodstained froth exudes from the nostrils and anus.

3-Clotting of the blood occurs rapidly

4- Incision of the affected muscle mass reveals dark red to black, swollen tissue with a rancid odor and thin,

5-sanguineous fluid containing bubbles of gas.

6-Freshly cut surfaces are often dry and may have a metallic sheen.

**Diagnosis**

1-case history

2-clinical signs

3-P.M lesion

4-isolation of bacteria

**Differential diagnosis**

NTIAL DIAGNOSIS

**• Malignant edema.** In typical cases of blackleg in cattle a definite diagnosis can be made on the clinical signs and the necropsy findings .

Definitive identification of *C. chauvoei* is by fluorescent antibody staining.

• Anthrax

• Lightning strike

• Bacillary hemoglobinuria

• Other causes of sudden unexpected death.

TREATMENT

Treatment of affected animals with penicillin and surgical debridement of the lesion

Large doses (40 000 IU/kg BW) should be administered, commencing with crystalline penicillin intravenously and followed by longer-acting preparations.

CONTROL

Cattle

vaccination of all cattle between3 and 6 months with two vaccinations given 4 weeks apart followed by an annual booster vaccination.

Sheep

With sheep in enzootic areas should be vaccinated twice, the last vaccination of ewes is given about 1 month before lambing.