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Black leg in ruminants

Introduction

Black quarter, black leg, quarter ill are all synonyms for the same acute infectious disease in cattle. Occurrence of the disease is worldwide, although it tends to be localized, even to certain farms or to certain pastures. Blackleg is primarily a disease of pastured cattle; sheep may also be affected. It preferentially affects animals under two years of age [1].

Blackleg is caused by *Clostridium chauvoei*, a highly pathogenic, histotoxic, anaerobic, endospore forming Gram-positive bacterium. The bacterium causes myonecrosis with high mortality and significant losses in livestock production [2].

Etiology

Clostridium chauvoei is a Gram-positive, rod shaped, spore forming and toxin-producing anaerobe bacteria [3]. It is one of the most pathogenic Clostridium species and can be found in the soil, faeces, and the digestive tract of many animals [5,6].

Although *C. chauvoei* is mainly considered to be specific to ruminants, rare fatal cases of fulminant human gas gangrene and neutropenic enterocolitis caused by *C. chauvoei* have been reported and it is assumed that prevalence of *C. chauvoei* causing disease in humans may be higher than currently diagnosed [4].

The pathogenicity of *C. chauvoei* is linked to several toxins and virulence factors. Among these, several hemolysins result in cytolysis and haemolysis, causing the characteristic lesions of blackleg [6,7].

Pathogenesis

The detailed pathogenesis of blackleg is still somewhat uncertain, but many of the critical points in the following proposed sequence of events have been confirmed in the natural disease and in experimental infections in cattle [11].

The spores are ingested from soil, enter the gastrointestinal tract and, by hematogenous route, reach the muscle where the spores remain latent in cells of the mononuclear phagocytic system. The spores may remain latent in the muscle for years [8,9].

Transient trauma or ischemia of the muscle favours the germination of the spores and secretion of cytolytic toxins that cause necrosis of myofibers and vascular endothelia, resulting in oedema and haemorrhage. The toxins are absorbed into the animal's bloodstream which makes the animal acutely sick and causes rapid death. Clostridial proliferation yields gas which appears as bubbles between the muscle's bundles [10].
Since *C. chauvoei*, the causative agent, is already present in the animal before the onset of disease, blackleg is referred to as an "endogenous" infection.
It is important to distinguish this endogenous pathogenesis from the so-called exogenous mechanism of gas gangrene, a disease that arises when spores or vegetative forms of one or more clostridial species, including *C. chauvoei*, enter subcutaneous and/or muscular tissues via skin or mucosal wounds [11].

Clinical signs and post-mortem findings

Animals that succumb to blackleg are usually in good nutritional condition. In the acute form, the body temperature of the animal increases to 41–42°C; the animal shows signs of depression, including a refusal to eat and lethargy; and subcutaneous gas oedema forms, especially in the extremities, with swelling and crepitus in the affected muscles [12,13].

During a large outbreak of blackleg on a Siberian farm following symptoms were reported. Gross swelling was present on the surfaces of the body, although no crepitus was palpable. In different animals, the oedema was localized to the limbs *(Figure 1A)*, chest, abdomen *(Figure 1B)*, and back *(Figure 1C)*. Paracentesis of the oedema revealed fluid accumulation and haemorrhage. During abdominal auscultation, there were signs of increased gas formation in the intestine. Several animals showed signs of papillomatosis on the muzzle and vulva, as well as birth trauma. The papilloma and birth trauma were accompanied by the subsequent formation of oedema and compaction of the surrounding subcutaneous tissue and muscles with the formation of dense nodes *(Figure 1D)*. Abortions and stillbirths were reported in pregnant cows [16].

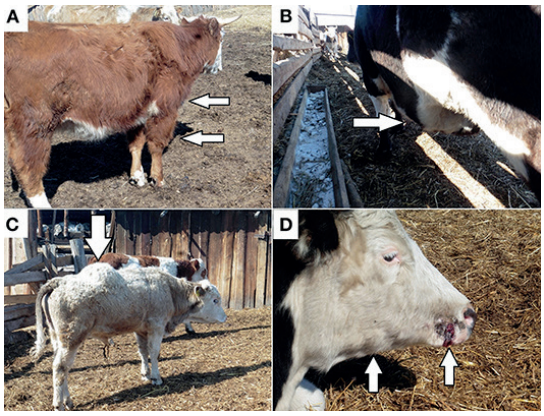


Figure 1: *Distinct clinical signs of blackleg: (A) oedema in the scapular shoulder joint of the anterior right limb; (B) oedema in the abdominal cavity; (C) oedema on the back; (D) papilloma trauma on the cow muzzle with subsequent oedema and the formation of nodes in the subcutaneous tissue in the area of the right branch of the jaw* [16]

In the hyper-acute form, the clinical signs are usually not observed due to the sudden death of the affected animal [14]. When superficial muscles are affected, the overlying skin is stretched by underlying inflammation and is dark. Emphysema of the subcutaneous tissue causes palpable crepitus. Classical necro-haemorrhagic and emphysematous myositis is present in the skeletal muscles, mainly in the hindlimbs, giving those muscles a black appearance, from which the name of the disease is derived. The large muscles of the rear quarters are most frequently affected; other muscular groups are less frequently involved [2,15,28].

Immediately after death, you can observe foamy, bloody fluid from the mouth, nose and eyes *(Figure 2A)*. Findings on necropsy at the sites of oedema can be haemorrhagic-necrotic myositis with gas formation and serous haemorrhagic infiltration of the loose subcutaneous tissue adjacent to muscles (*Figure 2B)*, haemorrhagic lymphadenitis of the pulmonary lymph nodes, cardiac haemorrhage *(Figure 2C)*, ruminal tympany with abdominal anaemia and thoracic hyperaemia, haemorrhage in the serous membrane of a scar *(Figure 2D)* [16].

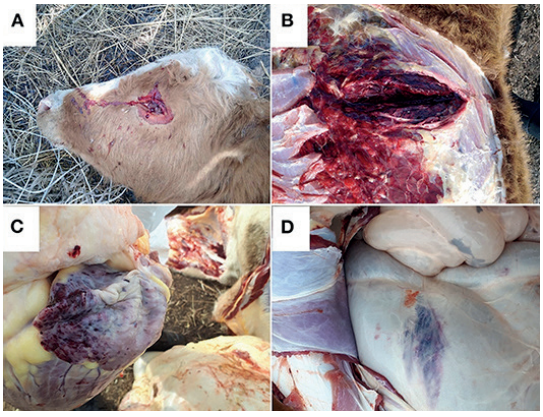


Figure 2: Necropsy findings in blackleg

Diagnosis

In the live animal a preliminary diagnosis of blackleg can be made based on clinical signs and the presence of typical muscle emphysema. Postmortem decomposition often complicates an accurate diagnosis. Traditionally, blackleg is confirmed by micro-biological culture and isolation of the causative microorganism. However, this is not always successful because of the difficulties in obtaining, submitting, and processing the samples in the laboratory [16,17]. *C. chauvoei* is sensitive to oxygen and it tends to be overgrown easily by other microorganisms in the samples. This may complicate obtaining the final diagnosis as it requires detection of *C. chauvoei* in affected tissues. Final diagnosis can be achieved by culture, PCR and/or immunodetection methods, including FAT and IHC.1 Decisions on which tests to use are often based on availability at local diagnostic laboratories [18,19,20,21].

Differential diagnosis

Blackleg should be differentiated from **malignant oedema** also referred to as gas gangrene or stable blackleg, a highly lethal exogenous infection caused by *C. septicum*. Sometimes several clostridial species, including *C. septicum, C. chauvoei, C. perfringens, C. sordellii* (now referred to as *Paeniclostridium sordellii*), and *C. novyi* are involved. Gas gangrene is a clostridial cellulitis and sometimes also myositis, associated with wound contamination [22,24,25].

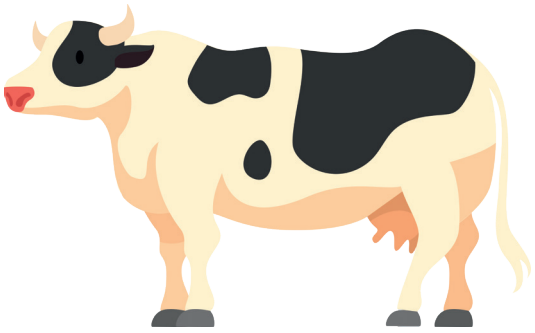
Anthrax caused by *Bacillus anthracis*, is a haemorrhagic per acute disease and the clinical sign is sudden death. Dark tarry discharge from body orifices, absence of rigor mortis, enlarged spleen, degeneration of the liver and kidneys are characteristic postmortem findings in anthrax [23].

Heart water, caused by the rickettsial agent *Ehrlichia ruminantium*, with sudden death and the presence of hydro pericardium, and **snake bites** with sudden death, lack of clinical signs and occasional per pharyngeal and brisket oedema, are also two possible differential diagnosis [23,26].

Treatment and prevention

Antimicrobials (drug of choice procaine penicillin) around affected tissues, aggressive surgical debridement to allow aeration along with supportive treatment can be of value. Treatment of affected animals with penicillin is logical if the animal is not moribund but results are generally poor, and prognosis is guarded because of the extensive nature of the lesion. A large dose should be administered, commencing with crystallin penicillin intravenous and followed by longer acting preparations, some of which should be given in to the affected tissue if it is accessible [23].

Due to the fact that treatment is usually not possible, the best option would be prevention by vaccination. Conventional blackleg vaccines are bacterins, prepared from formalin-treated cultures of *C. chauvoei*, that are generally available in polyvalent



formulations together with other clostridial components. The evidence for the efficacy of these vaccines is mostly anecdotal or based on measurement of antibody titres in vaccinated animals. However, the literature on clinical trials of these vaccines in cattle is surprisingly scant [27].

Nevertheless, the limited information available shows that these vaccines are nearly 100% effective in preventing blackleg after natural exposure, and 50–100% effective against experimental challenge with *C. chauvoei* [27].

Conclusion

Blackleg is an acute and often fatal infection occurring in mostly young, pastured cattle that continues to remain endemic worldwide despite large vaccination programs. The clinical course of disease is rapid and often results in sudden death. The pathogenesis seems logic and acceptable but there is no definitive supporting evidence available. Differentiation between black leg as an endogenous and gas gangrene as an exogenous infection remains difficult.

Declaration of conflicting interests
The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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